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SYSTEM OF MEDICINE.

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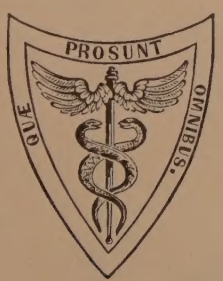
IN THREE VOLUMES.

VOL. II.

DISEASES OF THE RESPIRATORY AND CIRCULATORY SYSTEMS.



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A SYSTEM OF MEDICINE.

LOCAL DISEASES (*continued*).

DISEASES OF THE RESPIRATORY SYSTEM.

A. Diseases of the Larynx.

§ I. Primary Diseases of the Larynx.
Acute Laryngitis.
Chronic Laryngitis.
Morbid Growths.
Neuroses.
Diseases of the Motor System.
Paralytic Affections.
Spasmodic Affections, Laryngismus Stridulus.
Diseases of the Sensory System.

§ II. Secondary Diseases of the Larynx.
In Acute Affections; the Exanthemata.
In Chronic Affections.
Laryngeal Phthisis.
Syphilitic Disease of Larynx.

Appendix, on the Use of the Laryngoscope.

§ III. Croup.

DISEASES OF THE LARYNX.

By Morell Mackenzie, M.D.

In order to facilitate the treatment of this subject, Diseases of the Larynx have been divided into Primary and Secondary. The first includes all those conditions in which the larynx is the part first affected, and where the disease is generally, though not necessarily, of a purely local character. The second embraces those conditions where the laryngeal affection is a complication of a previously developed (acute or chronic) morbid state of the system. The primary affections are the inflammations, the morbid growths, and neuroses; the secondary are the occasional phenomena met with in the acute exanthemata, in phthisis and in syphilis. The classification is based on convenience. The limits allotted to this article forbid my occupying space by defending the arrangement, or by anticipating and explaining away any possible charge of apparent inconsistency in carrying it out.

SECTION I.

PRIMARY DISEASES OF THE LARYNX.

Acute Laryngitis.

Definition. — Inflammation of the lining membrane of the larynx, in which the vessels of the submucous areolar tis-

sue may or may not participate, characterized by dysphonia, or aphonia, dyspnœa, and stridulous breathing, cough, slight pain in the larynx—generally referred to the *pomum Adami*, and increased on pressure externally—and dysphagia. There is generally high constitutional fever.

Synonyms.—*Latin*—Cynanche Laryngea, Angina Laryngea, Angina Epiglottidea; *French*—Laryngite, Catarrhe Laryngien; *German*—Katarrlische Kehlkopfenzündung; *English*—Inflammation of the Larynx. Laryngitis is by some subdivided into Mucous Laryngitis (the *Laryngite muqueuse* of the French), and Submucous, or Œdematous Laryngitis (*Laryngite œdemateuse*).

Causes.—(*a*) *Predisposing.*—That relaxing habits predispose to the disease is rendered probable by the fact that residents in towns are more liable to it than those living in the country (Niemeyer); and of the former, those engaged in indoor occupations are much more susceptible than those much exposed to the weather. At the Hospital for Diseases of the Throat, laryngitis is much more often met with among tailors, shoemakers, porters, and people thus engaged, than among coachmen, cab-drivers, policemen, and others who are constantly exposed to the most inclement weather. Previous inflammation, and of course repeated previous attacks, render the part particularly prone to be affected. Males are more liable to it than females, and adults than children; but it proves far more fatal to the young; more than four-fifths of the mortality occurring before the tenth year.

(*b*) *Exciting Causes.*—Cold draughts of air, whether inspired or bearing on the neck externally, exposure of the body in general to cold, and especially allowing the feet to remain wet and cold for any length of time, are circumstances which in some people may give rise to the disease. Violent functional efforts (in giving the word of command, preaching, singing, &c.), and straining the parts in coughing, are not uncommon causes of it. Dusty air and irritating vapors ought, perhaps, to be considered as the *traumatic* causes; they are both probably sometimes concerned in the production of the disease, without even the patient being aware of their operation. The catarrhal form of the disease is often propagated from the nares, and œdematous inflammation sometimes from the pharynx. Extension of the disease occasionally takes place from below, the bronchial tubes being first affected; but the opposite sequence more often takes place, the laryngeal disease passing off with the occurrence of bronchitis.

Symptoms.—The approach of the disease is generally insidious, and a slight catarrh may suddenly become a most serious affection.

(*a*) *Subjective Symptoms.*—The patient complains at first of a slight dryness or soreness of the throat, or he may have nothing more than a feeling of roughness, or a tickling sensation with disposition to cough, or there may be a sense of constriction about the throat, and slight difficulty of swallowing; but the period at which this symptom supervenes, as well as its degree, depends on the part of the larynx first and most affected; in other words, it occurs at an early period, and is greatest when the epiglottis or ary-epiglottic folds are much affected, and later, and to a less degree, when the more internal parts of the larynx are attacked.

In severe cases all the true laryngeal symptoms become greatly aggravated. There is often a sensation as if a foreign body were lodged at the part, the breathing becomes extremely embarrassed, and the patient feels great anxiety about getting his breath. In fatal cases, the restless agony of impending suffocation generally gives way at last to a comatose state.

(*b*) *Objective Symptoms.*—(1) *Vocal.*—There is generally dysphonia in the early, aphonia in the later, stages. The cough is at first clear and shrill, then harsh and croupy, finally aphonic. It is generally frequent, and often paroxysmal. Its exact character and variations, however, depend on the particular part of the larynx which is affected.

(2) *Respiratory.*—The inspiration is at first a little prolonged and wheezing, afterwards very much lengthened and accompanied with stridor. In the later stages there is a kind of groan in expiration. In addition to these sounds mucous râles can generally be heard on auscultation over the larynx. As the calibre of the larynx becomes contracted from œdematous infiltration and spasmodic approximation of the vocal cords, the patient expends all his energies on the respiratory process. Sitting up in bed he desperately clutched the bed-clothes, and in his violent efforts to get breath, the shoulders are seen to rise and the whole chest to heave.

(3) *Laryngoscopic Signs.*—In the early stages, and in mild cases, the mucous membrane is merely seen to be of a bright red color; the hyperæmia is, as a rule, diffused, though sometimes there may be distinct injection of the vessels. In severe cases œdema soon appears, the parts affected being seen to be red, swollen, and semi-transparent. When the epiglottis is acutely inflamed, it frequently presents the appearance of a raised ridge in the median line, with two

large tumors on each side; the valve is in fact folded upon itself, and only its upper surface is visible. This condition occludes the view of the larynx. When the ary-epiglottic folds are attacked, their shape becomes very irregular; the ventricular bands are sometimes seen to be in a highly turgid state, and in this case they eclipse the vocal cords. If the latter are visible, they are of a bright red color, slightly swollen, especially posteriorly; their sharp free edge is rounded, their mobility is impaired, and on inspiration their normal action is occasionally seen to be reversed, the glottis tending to become closed instead of open.

[Fig. 1.

Œdema of Glottis. *a*. Tongue. *b*. Mouth of Larynx. *c*. Trachea. From a specimen in the cabinet of Dr. Gross.]

(4) *Miscellaneous Symptoms.*—The laryngeal secretion is generally very scanty, tenacious, and difficult to expectorate; in favorable cases, where the disease is passing off, it may become thick, purulent, and abundant. In the early stages, though not generally till a few hours after the local symptoms have manifested themselves, there are signs of inflammatory fever; the tongue is white and furred, the tip and edges being generally red. The pulse is frequent and hard, the skin hot, and the face flushed. At a later stage the constitutional conditions resemble that of hectic, the skin under the immense respiratory efforts being bathed in perspiration, and the pulse small, feeble, frequent, and irregular. The countenance is of an ashy pallor, the lips purple, and the eyeballs protrude from the dark halo which surrounds them.

Course and Termination.—The acute stage seldom lasts more than three or four days, and I have seen a case terminate fatally in twenty-four hours. Death has been known to occur in seven hours.[1] It is rare for the symptoms to remain serious after the fifth day, unless a kind of chronic œdema sets in. The disease may terminate in any of the following ways:—(1) Spontaneous resolution may occur. (2) Resolution may be brought about by therapeutics. (3) The acute symptoms may pass away, and chronic congestion remain. (4) Death may take place very suddenly, from the combined effects of œdematous swelling and spasm of the glottis, less suddenly from the former cause acting alone, or slowly, and often preceded by delirium from the effects of exhaustion and imperfectly aërated blood. (5) Threatened suffocation may be averted by the operation of tracheotomy.

Diagnosis.—In very young children it is impossible to distinguish between acute laryngitis and croup; but, where the laryngoscope can be used, the presence or absence of false membrane can of course be ascertained at once. Even with this instrument, however, the essential nature of the morbid process cannot always at any early period be ascertained, as the apparently simple inflammation may be an early stage of the plastic form of disease.[2]

Laryngismus stridulus differs by its very sudden accession, by its generally occurring during sleep, by its passing off and leaving the child in an apparently normal condition as regards the laryngeal symptoms and respiration, and by the absence of constitutional fever. Spasm of the glottis in adults is easily differentiated by the general symptoms, and still more so by the employment of the laryngoscope.

Pathology.—The disease is essentially a simple inflammation of the mucous membrane, and submucous areolar tissue of the larynx; the danger of the disease being in proportion to the extent that the last-named structure participates in the morbid process. When the deeper tissues are affected, the products of inflammation accumulate beneath the lining membrane and cause the tumefaction which in this situation is attended with such imminent risk. When the inflammatory process is superficial, its effects are of less importance. The character of the secretion becomes altered, being at first clear and gummy in character, and afterwards containing an increased quantity of pus corpuscles. There is partial destruction and imperfect formation of the normal epithelial structure, but the process scarcely ever leads to ulceration. The danger is not due to the œdematous swelling alone, but also to the spasm of the glottis which

[1] Dr. Wood, Pract. Med. vol i. p. 780.
[2] See article "Croup."

the infiltration causes—partly by reflex action, partly by direct irritation of the adductor muscles of the vocal cords.

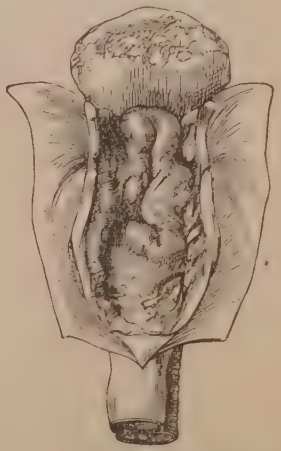
[Fig. 2.

Œdema of Glottis.]

Morbid Anatomy.—The superficial appearances, a few hours after death, resemble those described under the head of Laryngoscopic Signs. In children, the mucous membrane is generally slightly softened, and of a bright red color. In adults, on the other hand, the redness seldom remains after death, as in those cases which prove fatal the activity of the morbid process is in the submucous tissue. The product of the inflammatory process is generally of a serous character, but it may be sero-purulent, or may even be of the nature of what is called "healthy pus." In the latter case, the condition is that of diffused abscess: circumscribed abscess—as far as I am aware—never occurs as a sequel of acute inflammation of the larynx. The effusion, however, is much more frequently of the serous character. It generally collects in those parts where the areolar tissue is most lax: thus the epiglottis and the ary-epiglottic folds are the parts which are both the most frequently distended, and which become the most swollen; next to them the ventricular bands (false vocal cords) are most commonly affected; the vocal cords may be a little tumefied, but they are rarely swollen to any extent. The muscles are often saturated with the serous fluid. If the patient survives the acute stage and dies from other causes, the parts previously swollen and œdematous, present a peculiarly sodden and shrunken appearance.

Prognosis.—In giving an opinion as to the danger, the age of the patient is the most important consideration. In early life, that is, before the development of the larynx has taken place at puberty, the disease is always attended with great danger. As regards adults also, a very serious opinion must always be given. The danger depends on the amount of œdema present; and though tracheotomy remains as a last resource, there is always a risk of the disease extending down the windpipe, so that both bronchitis and pneumonia often supervene.

[Fig. 3.



Acute Œdema Glottidis; exposed from behind.]

Therapeutics.—If the case come under observation at a very early period, it is really quite impossible to tell whether the disease is a simple catarrh of the larynx, or is likely to turn out a violent inflammatory affection. Under these circumstances a system of rational expectancy must be adopted; a warm, moist and uniform temperature enforced, and gentle diaphoretic and mild purgative medicine administered.

The same kind of treatment cannot be carried out in the case of children as where the patients are *adults*. The following will be found useful for the latter class of patients:—In the early stage, and in slight cases, an inhalation of hot steam, or steam impregnated with the volatile principles of benzoin, or hop, or conium, may be used.

The following forms will be found serviceable:—

1. ℞. Tinct. benzoin. comp. fl. dr. j ad fl. dr. ij:

to be added to a pint of water at 150° F., and inhaled (from a quart jug with a narrow neck, or from a special inhaling apparatus) for ten minutes every three or four hours.

2. ℞. Ol. lupuli ♏ xv.
Mag. carb. lev. gr. x.
Aquæ ad fl. oz. iij. M.

A teaspoonful in a pint of water at 150° F., and used as No. 1—for five to eight minutes, three times a day. The addition of a scruple of camphor to three ounces of any of the foregoing will be found useful, if a rather more stimulating effect is de-

sired. The juice of conium in the following form is often beneficial :—

3. ℞. Succi conii fl. dr. ij.
Sodæ carb. gr. xx.

Mix and add to a pint of water at 150° F., and use as No. 1. Or—

℞. Coniæ gr. ½ to gr. ½.
Sp. vini rect. fl. dr. j.

Mix and add to a pint of water at 150° F., and use as above. Where there is much pain, or tendency to spasm, chloroform (ten to thirty drops) may be added once or twice at intervals of five minutes during the inhalation. These remedies can be used alone or in combination. If crescent inflammation of the pharynx accompanies the laryngeal hyperæmia, the local action of guaiacum administered in the form of lozenges will often prove most beneficial.

If, however, the disease makes head under this treatment, and the parts are acutely inflamed, *without being œdematous*, an attempt may be made to restrain the crescent inflammation by the application of a strong solution of nitrate of silver (60 gr. ad 1 fl. oz.), or perchloride of iron (120 gr. ad 1 fl. oz.); or chloride of zinc (30 gr. ad 1 fl. oz.); or chloride of aluminium (gr. 60 ad 1 fl. oz.). Solutions of nitrate of silver, still largely employed by the profession, have not proved more serviceable in my hands than the mineral astringents, whilst they more often cause spasm and nausea. Inhalations of atomized liquids may be tried, and among these tannin (5 gr. ad 1 fl. oz), ferri perchloridi (3 gr. ad 1 fl. oz.) are most likely to do good. If the inflammatory process is not arrested by the action of these remedies, the œdema, which is almost sure to supervene, should be treated by free scarification with the aid of the laryngoscope, and a properly constructed laryngeal lancet. Should this treatment, however, be impossible or ineffectual, and should the dyspnœa be of a threatening character, tracheotomy must not be delayed. In these cases the result of the operation is especially favorable. General blood-letting, leeching, blistering, mercury, and antimony, were the most weighty remedies of the profession twenty years ago; but they cannot be put in the balance against the topical treatment which the laryngoscope renders possible. Non-depressant emetics, however, such as sulphate of zinc (20 gr. to 30 gr.), sulphate of copper (5 gr. to 10 gr.), in plenty of warm water, are sometimes useful where there is much œdema; and leeching and blistering may be conveniently resorted to by country practitioners who have not the opportunity of applying remedies with the aid of laryngoscopy.

Treatment of Children.—As it is impossible to distinguish between infantile laryngitis and croup, the treatment must in effect be the same for each disease.[1] In addition, however, to the treatment recommended by Mr. Squire, scarification, as described below, may sometimes be performed with the greatest advantage.

VARIETIES.—Acute inflammation has been here described in its most complete and severe form; but it can easily be understood that congestion of the larynx, or *Subacute Laryngitis*, may come on very suddenly, remain for a few days, and pass away without any further development. The hoarseness which often accompanies faucial catarrh is due to this cause, the vocal cords being in this case the part of the larynx most affected. The symptoms either give way, or the disease assumes the character of chronic laryngitis.

Traumatic Laryngitis may, perhaps, be considered as belonging to the province of surgery; but it is desirable briefly to call attention to that form which occurs to children from swallowing boiling liquids. Children of the poorer class are often allowed to drink tea from the spout of the teapot, and when left alone they attempt the same feat at the boiling kettle. Instant inflammation of the pharynx and orifice of the larynx sets in, and in two or three hours, or even sooner, the epiglottis becomes greatly swollen and œdematous.

Scarification, first recommended by Lisfranc,[2] and since by Busk,[3] Tudor, and others, is the most rational treatment. The age of the patient generally renders the use of the laryngeal mirror out of the question; but the fauces should be illuminated as in laryngoscopy. In children, under these circumstances, the swollen and œdematous epiglottis can be seen in an erect posture at the back of the tongue. It may be scarified with a gum lancet, or a curved, sharp-pointed bistoury, which should be quite blunt (or covered with strips of plaster) up to within two or three lines of its extremity. Emetics either before or after scarification are often useful. The pressure which the act of retching exercises on the œdematous tissue, perhaps proves beneficial in consequence of the mucous membrane rupturing, and allowing the aqueous matter to escape. A strong solution of nitrate of silver or of some other mineral astringent may sometimes arrest the crescent inflammation *before œdema has taken place*. The local abstraction of blood is recommended by some, and Dr. Bevan[4] has reported four severe cases successfully treated by application of leeches to the margin of the sternum, an emetic followed by a cathar-

[1] See article "Croup."
[2] Journal Général, Année 1825.
[3] Lancet, August 13, 1859.
[4] Dub. Quart. Journ. of Med., Feb. 1860.

tic, two grains of calomel every half hour, and mercurial inunction. Scarification, however, fairly and fully carried out, ought to supersede all other treatment.

Tracheotomy, from which *à priori* the most satisfactory results might be anticipated, is not a very successful operation in these scalded throats ;[1] but nevertheless recourse must be had to it when other remedies fail, and the dyspnœa threatens death.

Chronic Laryngitis.

Definition.—Chronic inflammation of the lining membrane of the larynx characterized by hoarseness or loss of voice and generally by more or less cough.

Synonyms.—*Latin*—Laryngitis chronica ; *French*—Laryngite chronique ; *German*—Der chronische Katarrh der Kehlkopfschleimhaut. For other synonyms see "Laryngeal Phthisis," which disease was formerly confused with chronic laryngitis.

Causes.—The causes of the disease are the same as those indicated under the head of Acute Laryngitis, to which disease it often proves the sequel. The chronic forms of inflammation, however, more frequently extend from the pharynx, and the effects of continuity of texture are often seen in chronic alcoholism and the abuse of tobacco. It is also more frequently caused by functional excesses.

The great and sudden development of the larynx which takes place at puberty in males, is often attended by a mild form of laryngitis — the so-called "cracked voice" of boys being always associated with marked congestion of the vocal cords. There seems also to be a rare constitutional condition, where there is a tendency to chronic inflammation of many of the mucous canals. Four such cases have come under my notice ; the patients were all men over fifty years of age. I have at present a gentleman under my care suffering from chronic laryngitis, slight thickening of the walls of the lower third of the œsophagus, gastro-intestinal derangement, and chronic cystitis. The influence of age and sex is very marked, adult males being by far the most common sufferers, and children the rarest.

Symptoms.—*Subjective.*—The patient's sensations are not generally very vivid, a tickling feeling being generally all that is complained of ; in some cases, however, a pricking or burning pain is felt. The congestion of the vessels and perhaps the presence of an altered secretion causes in some cases a frequent desire and effort to clear the throat.

Objective.—(1) *Vocal.*—Impairment of function is the most characteristic symptom of the disease. It varies in degree from slight modification in tone, to complete loss of voice. It is characteristic also of this form of hoarseness *in the early stage*, that it is most marked when there has been rest of function for some time. Thus a person with slight chronic congestion may be extremely hoarse on attempting to speak after being silent for some time, but the voice may become almost normal after the function has been exercised for a few minutes. The improvement probably depends on the quickened capillary circulation, and stimulated nerve-force of the part. It has its analogy elsewhere. In dysphonia, dependent on feeble approximation of the vocal cords on the other hand, the voice is strongest when first exercised, and gradually becomes weaker as it continues to be exercised. Sometimes the voice is clear and natural in its ordinary tones, and the discordance is only observed when powerful exertions are made (as in singing, acting, public speaking, &c.). The cough is generally rather frequent, but it may amount to nothing more than a hawking or "hemming" noise, and sometimes it is almost altogether absent. On the other hand, it may be the most troublesome symptom.

(2) *Respiratory.*—The respiration is not materially affected, though moist râles can usually be heard over the larynx.

(3) *Laryngoscopic Signs.*—The congested condition of the lining membrane of the larynx is at once apparent on using the laryngoscope. The hyperæmia may be general or partial. The following is the order of frequency in which the mucous membrane over the different parts is affected :—First, the capitula Santorini ; secondly, the ventricular bands ; thirdly, the epiglottis ; fourthly, the vocal cords, and least frequently the ary-epiglottic folds. The redness generally fades off gradually into the healthy colored membrane, but injection of the minute vessels is sometimes apparent on the epiglottis and vocal cords. On the former the injection is generally arborescent, on the latter the arrangement of the vessels is usually linear, along the attached side of the vocal cord. Sometimes one vocal cord is seen to be bright red, whilst the other is of its usual white color, and the congestion may even be limited to a small portion of a cord. Sometimes the anterior half or third of the cord, sometimes the posterior portion, is affected ; or even a section of the whole length of a cord may be injected, whilst the rest remains of a normal color. In the latter case it is always the outer attached portion of the cord which is congested. Small pellets of mucus are often seen sticking to different parts of the laryngeal membrane ; and in

[1] See Med. Times and Gaz., vol. xix. p. 366; and Brit. Med. Journ., Jan. 14, 1860.

cases of long-standing disease, the larynx has the appearance of being very much dilated and covered with secretion; on the other hand, the membrane may look dry and glistening. It is often noticeable that on attempted phonation, the vocal cords do not thoroughly approximate the congestion of the membrane interfering with the action of the muscles.

Miscellaneous Symptoms.—The varying character of the expectoration may be inferred from what has been already stated, but it may be observed that it is seldom abundant, unless the laryngeal affection is complicated with bronchitis. The constitution does not generally suffer, but there is occasionally some sympathetic irritation.

Course and Termination.—The tendency of the disease when once fully established is to remain stationary, or the symptoms may disappear for a short time, and then recur. The disease in old people is always complicated with chronic bronchitis, and the symptoms of the later affection mask and outweigh in importance the morbid phenomena dependent on the chronic laryngeal disease. The principal danger is from chronic œdema coming on, but this is an exceedingly rare complication. In some cases, especially between the ages of twenty and forty, persistent chronic laryngitis appears to predispose to the development of phthisis, but it is difficult to tell how far the laryngeal hyperæmia is concerned as a cause, and how far as a consequence.

Diagnosis.—An accurate opinion can only be formed by a careful laryngoscopic examination. It is of the first importance to observe whether there be thickening or not; and in the former case to notice carefully whether there be merely inflammatory tumefaction, œdematous infiltration, or tuberculous exudation.

In œdema the swelling is generally of a bright color, and has a characteristic transparent appearance; in phthisis, on the other hand, the thickened parts are generally of a dull color—though the surface may be congested, and the swelling generally presents the appearance of a solid tumor (see "Laryngeal Phthisis"). In all cases of chronic laryngitis of some months' standing, the lungs must be most carefully examined, the history of the patient and that of his family closely investigated, and his general condition inquired into, before a decided opinion as to the nature of the disease is given.

Pathology and Morbid Anatomy.—The disease is essentially a chronic inflammation of the lining membrane of the larynx, in which the vessels of the areolar tissue participate very little. Enlargement and tortuosity of the small vessels is found in cases of long-standing congestion, and occasionally, but very rarely, dilatation of the laryngeal canal takes place.

Prognosis.—The disease never terminates fatally, unless some complication arises; on the other hand, it is often difficult to cure, especially in old people.

Therapeutics.—Local remedies are the most important agents in the treatment. These are commonly called "caustics," but their action seems rather of an astringent character. Any of the following may be used:—Ferri perchlor. (60 gr.), ferri persulph. (60 gr.), ferri sulph. (120 gr.), cupri sulph. (10 gr.), zinci chlorid. (30 gr.), zinci acet. (5 gr.), zinci sulph. (10 gr.), aluminis (30 gr.), alum. chlor. (60 gr.), dissolved in an ounce of water or glycerine. The latter vehicle, through its denser consistence, is better adapted for keeping up a prolonged action on the part. The chloride of zinc solution is the remedy which I most frequently employ; but provided that the application is made accurately and sufficiently often, it really matters very little which solution is used. The application should be made daily for the first seven days, every other day the second week, twice in the third week, and so on—gradually lengthening the interval between the application. This is a general rule, but it must be modified according to circumstances. In cases where there is excessive secretion from the larynx (laryngorrhœa) the local application of turpentine sometimes does good, though these cases are very troublesome to treat. On the other hand, where there is long-standing hyperæmia, with diminished secretion—where the mucous membrane looks dry and shining—the remedy which I have found most successful is carbolic acid (from half a drachm to a drachm of the pure white carbolic acid to an ounce of glycerine). These local remedies can be best applied with the aid of the laryngoscope—the laryngeal mirror being held in the left hand and a camel's-hair brush (fixed to a slender rod of aluminium at an angle of about 95° or 100°, and fastened in a wooden handle) in the right hand. Those who do not employ the laryngoscope should hold the patient's tongue well out, in such a position that the posterior wall of the pharynx can be seen, and should then pass the brush down between the latter and the base of the tongue. In this way the remedy is likely to reach the desired destination: the old method of pressing down the tongue with a spatula and using a flexible sponge probang could only end in failure. Instruments of the syringe character are quite unnecessary for the application of remedies to the larynx, and they give rise to more irrita-

tion than a simple brush. Powdered substances likewise cannot be recommended —they are, as a rule, either inert or injurious. Great benefit is, however, sometimes derived from inhalation—either of steam impregnated with some stimulating volatile principle, or of atomized liquids of an astringent character. For the steam inhalations the following formulæ will be found useful :—

℞. Ol. pini sylvest. fl. dr. ij ad fl. dr. iij.
Mag. carb. lev. gr. lx to gr. xc.
Aquæ ad fl. oz. iij. Mix.

A teaspoonful to be added to a pint of water at 150° F., and inhaled for five minutes twice or three times daily.

℞. Creasote, fl. dr. iij.
Glycerine, fl. dr. iij.
Aquæ ad fl. oz. iij. Mix.

A teaspoonful to a pint of water at 150° F. as above.

R. Ol. juniperi Ang. ♏xx.
Mag. carb. lev. gr. x.
Aquæ ad fl. oz. iij. Mix.

A teaspoonful for each inhalation as above.

To either of these the addition of a scruple of camphor is often serviceable after the mixture has been used for about a week.

For spray inhalations the following ingredients are most to be recommended : the proportions given are always for one ounce of water ; and the quantity to be used each time should be from two fluid drachms to half an ounce of the solution :—

Alum, 10 to 20 grains.
Tannin, 1 to 20 grains.
Perchloride of iron ½ to 2 grains.
Ditto, 2 to 10 grains (in hemorrhage).
Sulphate of zinc, 1 to 6 grains.
Chloride of zinc, 2 to 10 grains.

It is almost unnecessary to observe that the voice should be exercised as little as possible. For singers, actors, clergymen, and others whose occupations require them to use the voice much, rest of the vocal organ is of the utmost importance. When complete silence cannot be enforced, the least possible exertion should be made in speaking—the patient should, in fact, whisper. All direct sources of irritation must of course be removed. Thus, if the uvula is much elongated, it must be amputated before a radical cure can be effected. As the pharynx is almost invariably more or less affected, astringent lozenges will be found very useful. Tannin, rhatany, and kino may often be prescribed in this form with great advantage.[1] Change of climate is often very beneficial. Generally speaking, a warm dry atmosphere suits best, but providing there is no humidity the temperature is not so important. Thus, in parallel cases, I have seen equal benefit follow from a short residence in Algiers and a few weeks spent at Cromer or Margate. The warm relaxing climate of the south coast is generally injurious ; but cold winds, especially those of an easterly character, often give rise to subacute inflammation. The waters of Ober-Salzbrunnen and Ems (source Kränzchen) are especially recommended by Niemeyer, who observes, that "the influence of these waters, so manifestly favorable in many cases, cannot be explained by physiology." Weilbach, Eger, Kissingen, and Marienbad are recommended by other German writers, whilst French physicians praise the waters of the Pyrenees. Where suitable atmospheric conditions cannot be selected, the patient must wear a respirator, when the weather is at all cold and damp, and must protect the neck and body generally by warm and suitable clothing. Medicines and hygienic treatment may be necessary in some cases, and must vary according to circumstances.

Varieties.—(1) Chronic Glandular Laryngitis. (2) Phlebectasis Laryngea.

(1) Chronic glandular laryngitis is a variety of chronic inflammation in which the minute racemose glands are principally affected. The credit of first noticing it is generally given, in this country and in America, to Dr. Horace Green of New York, but according to French writers it was described by Professor Chomel (*Gazette Médicale*, April, 1846) at least six months earlier. It has many *synonyms :* thus we have dysphonia clericorum (mal de gorge des ecclésiastiques, clergyman's sore-throat), angine glanduleuse, laryngite granuleuse (ou granduleuse), follicular laryngitis, follicular disease of the pharyngo-laryngeal membrane, and tubercular sore-throat. As the glands of the larynx are all of the racemose variety (Kölliker), the term follicular laryngitis is obviously incorrect, and glandular laryngitis designates the condition more accurately. The causes of the affection are the same as those which gave rise to simple inflammation. The French, indeed, consider that it is of an "herpetic" nature, but this term is used in such a vague way by French authors, that it really has no definite meaning. The morbid process of the larynx often results from an extension of the disease from the pharynx, in which situation *the follicles* are principally concerned ; it may, however, originate in the larynx and afterwards reach the pharnyx. The disease is not peculiar to the clergy, nor is the chronic laryngitis, from which they often suffer, as a rule, of the glandular character. The disease

[1] These lozenges have been prepared for me by Messrs. Bullock and Reynolds, 3 Hanover Street, who will be happy to give the formulæ to any practitioner.

might with equal truth be called "costermonger's sore-throat." It is often associated with indigestion, but whether there is any causative relation between these conditions is uncertain. The symptoms are the same as those of simple chronic laryngitis, but perhaps milder—weakness of voice, fatigue after speaking, a constant inclination to clear the throat and swallow the saliva, or perform an act of deglutition, being the principal morbid phenomena. With the laryngoscope the enlarged orifices of the glands may sometimes be seen on the epiglottis and the posterior part of the vocal cords as pale specks on the congested membrane, or as small red circles on the pale membrane; the other laryngeal appearances do not differ from simple laryngitis, except that the approximative action of the vocal cords is more often feeble and imperfect. Constitutional debility was thought by Dr. Green to be a characteristic phenomenon, but there is very often no general weakness or evidence that the system at large is at all affected. As regards the pathology of the disease, it may be remarked that it is essentially a disease of the secretory system, the normal secretion of the minute racemose glands, instead of being clear and transparent, becoming thick, white, and opaque. The morbid process is essentially mild, but very chronic, in character. The treatment should be the same as that recommended for simple chronic laryngitis. Solutions of the crystals of nitrate of silver (from two to four scruples of the salt to an ounce of distilled water) were strongly recommended by Green, and since by other writers, but they do not seem to me to act more beneficially than other mineral astringents. The sulphuretted waters of the Pyrenees, especially of Les Eaux Bonnes, are viewed by the French as almost specific in their action; and several patients that I have sent there have derived undoubted benefit from the use of those waters, where the voice remains weak after the glandular disease has been cured. Benzoic acid lozenges often act very beneficially as nervo-muscular stimulants.

(2) Phlebectasis laryngea,[1] or venous congestion of the larynx, is an extremely rare affection. It may depend on general or local causes; that is to say, it may occur "in persons affected with a morbid preponderance of the venous system" (Hasse), or may be due to a local strain. The symptoms are generally slight; some alteration in the voice, an uneasy sensation in the larynx, and perhaps a more or less frequent cough, being the principal morbid phenomena. The laryngoscopic appearances may be thus described:—In mild cases, when the disease is very limited, extremely fine dark vessels may be running along the upper border of the ventricular orifice: in more severe cases there is less regularity in the distribution of the distended veins, which may be observed on the ventricular bands, vocal cords, and arytenoid cartilages. Cases have come under notice in which streaks of blackened mucus adhering to the larynx have been mistaken for varicose veins. This error needs only to be mentioned to be avoided. This condition of the larynx, independently of the inconvenience it occasions, is probably attended with some danger, as it most likely predisposes to passive œdema. The disease should be treated by the application of strong astringents; and of these a saturated solution of tannin in glycerine is the best. Constitutional remedies of a tonic and invigorating character should also be used.

[1] This disease was first described by the author in the Lancet, July 6, 1862.

Morbid Growths.

Definition.—New formations, whether of simple or malignant character, appearing as distinct tumors, projecting from the mucous membrane of the larynx, and more or less separated by a line of demarcation from the tissue from which they grow.

Synonyms.—*Latin*—Polypus Laryngis; *French*—Polypes du Larynx; *German*—Kehlkopfpolypen Neubildungen im Kehlkopfe; *English*—Polypus of the Larynx, Warty Growths, Warts, New Formations, Excrescences, Cancerous Growths, &c.

Natural History.—*Causes.*—Benign growths in the larynx are probably almost always dependent on local hyperæmia, and therefore their primary causes must be sought for under the head of Laryngitis. Chronic inflammation of persistent character but low degree is, probably, the condition most favorable to their development. In young children the disease is often attributed to an attack of croup, and it probably does originate sometimes in this way. The presence of a warty growth in the larynx, however, produces symptoms closely resembling, and very likely to be mistaken for, those of croup. Neither syphilis nor phthisis are predisponents. The evolution of cancerous growths in the larynx (as elsewhere) is dependent on laws of development which are not understood.

Symptoms.—(*a*) *Subjective.*—The early symptoms are very vague, as the chronic laryngitis which precedes the new formation causes the same sensations (see Chronic Laryngitis). Patients occasion-

ally complain of a feeling of something striking in the throat, and when the growth is pedunculated there is sometimes the sensation of a body moving about in the larynx. This, however, is quite the exception. Even in cases of true cancer there is seldom much pain. Difficulty of swallowing is generally present, if the growth attain to any size—especially if it affects parts concerned in deglutition or projects into the food-tract. Where the growth is large, shortness of breath is experienced.

(*b*) *Objective Symptoms.*—(1) *Vocal.*—The voice is generally, but not necessarily, hoarse. Dysphonia is more common than aphonia. Small warts more often destroy the function than the larger and polypoid varieties (Czermak). The voice has sometimes a kind of paroxysmal or intermittent character, being one moment almost normal, and at the next very hoarse, or even quite suppressed.

(2) *Respiratory.*—The breathing is embarrassed if the tumor is large, and there is sometimes stridulous breathing. The extent to which the respiration is affected bears a direct relation to the respective sizes of the growth and the laryngeal canal.

On auscultation, moist sibilant râles may be heard over the larynx, and a valvular murmur has been described as being very characteristic of the presence of a morbid growth (Rühle): it is not, however, to be depended on. The cough varies in character and in frequency in different cases, and sometimes is of a croupy character.

(3) *Laryngoscopic Signs.*—With the laryngoscope, the disease is generally at once revealed. The appearances vary according to the pathological nature of the tumor. *Papillomata*, which are the most common of all benign growths, vary in size from a grain of mustard to a walnut—their most common size being that of a large split-pea. These growths have generally a mammillary, lobulated, or cauliflower configuration. They are generally of a lighter color than the surrounding mucous membrane, and most frequently grow from the vocal cords. They are generally sessile and multiple. *Fibrous tumors* are seldom seen of such small size as those of papillary character, and they are also much less common. They vary in size, from a split-pea to an acorn, and have usually a smooth surface. They are generally single and pedunculated, and in seven of the fourteen cases that have come under my notice, the growth sprang from the vocal cords, and was confined to those parts. *Fibro-cellular tumors* are comparatively rare, and represent only about 5 per cent. of all benign laryngeal growths. They sometimes attain the size of a cherry, but are generally smaller. They are invariably pedunculated, generally sessile, and usually of a pale color. *Cystic tumors* are seen as round egg-like projections, and as they usually give rise to some local irritation, they are themselves red and surrounded by a hyperæmic area. The other kinds of growth are too rare to require a special description. In cancer there is generally irregular thickening of some parts, and destruction of others. The former process usually precedes the latter. Destructive ulceration is most characteristic of epithelial cancer, and thickening of the encephaloid variety. The epiglottis and ventricular bands (false vocal cords) are the most common seats of true cancer. In encephaloid cancer the parts are often so much displaced that there is difficulty in recognizing them. The epiglottis may be pushed completely on one side, and an ary-epiglottic fold or ventricular band may be so much swollen and inflamed as to cover the parts situated below, on the opposite side of the larynx. Growths, especially small ones, on or about the vocal cords, are apt, as Türck first pointed out, to give rise to functional paralysis of one of the vocal cords.

(4) *Miscellaneous Symptoms.*—Occasionally the presence of a morbid growth is proved by the patient coughing up particles which can be examined microscopically. Sometimes the growth rises out of the larynx, and can be seen when the mouth is widely opened.[1] In children it is sometimes possible to introduce the finger into the larynx and feel the growth; but the small size and soft structure of these warty productions make it almost impossible thus to distinguish them. The expectoration is generally increased and altered slightly in small benign growths—very much in the case of larger ones and in cancer. Where the growth is small and benign, constitutional symptoms may be, and generally are, altogether absent; but where the tumor is so large as to interfere seriously with respiration, the system at large is likely to sympathize: in some of these cases there is irritative fever, whilst in others the constitutional symptoms are more those of hectic. In the case of true cancer, the characteristic cachexia is present.

Course and Termination.—In the case of non-malignant growths, the symptoms generally develop themselves slowly, taking several months for their evolution; after attaining a certain degree of severity, they often remain stationary, unless some complication, as œdema or spasm of the glottis, occurs. The latter condition is likely to supervene if the growth is large; and it gives a paroxysmal character to the

[1] Horace Green on Morbid Growths in the Larynx, p. 62; and Rayer, Maladies de la Peau, tome ii. p. 422.

symptoms. The advanced symptoms are those of impending suffocation. Epithelial cancer usually gives rise to more distressing local symptoms; there is more expectoration, and deglutition is often difficult and painful. In encephaloid cancer the symptoms progress more rapidly, the fatal termination usually taking place within a few months of its first appearance. This, however, is not always the case.[1] The patient sinks from the combined effects of gradual suffocation, slow starvation, and the intrinsic nature of the disease.

DIAGNOSIS. — Tumors in the larynx cannot well be mistaken for any other disease, if a laryngoscopic inspection can be made. The possibility of eversion of the ventricle must not, however, be forgotten. The tubercles of syphilis are seen as irregular whitish prominences on the congested mucous membrane — the posterior wall of the larynx being their most common site. The thickening of laryngeal phthisis is not so great as that of true cancer, nor has it the defined character of the benign kinds of growth. It is not quite so easy to distinguish between the benign and the malignant epithelial growths. The former, however, are more strictly defined, and never (unless quite accidentally) ulcerated, whilst in the latter there is generally irregular thickening from interstitial exudation, and frequently ulceration. The microscope cannot be relied upon for differential diagnosis should particles be expectorated, or removed during life with the aid of the laryngoscope. Several cases have come under my notice where the histological features were decidedly those of cancer, whilst the clinical history was of a totally opposite character. The laryngoscopic appearances and constitutional symptoms furnish much more important indications in relation to the differential diagnosis of the various kinds of growth than the microscopic examination.

PATHOLOGY. — The non-malignant kinds of tumor are essentially local productions — the result of a perverted nutritive process in which growth is excessive and development imperfect. Hence the formation of tissues of abnormal size and morbid structure. They are probably always associated in their origin with local hyperæmia. In the case of malignant growths, in addition to the local changes, there are constitutional influences in operation which will be found fully described elsewhere.

MORBID ANATOMY. — The benign growths found in the larynx are Papillomata, Fibromata, Fibro-cellular Tumors, Cystic Tumors, Sarcomata, and Lipomata, and they are here enumerated in their order of frequency. The malignant growths belong either to Epithelial or Encephaloid varieties.

Papillomata are by far the most common of laryngeal growths, three-fourths of all the benign cases being of this nature. The papillary growths, "in their general form and arrangement, have many points of resemblance, but on the enlarged scale, to the papillæ, which in various localities constitute natural projections from free surfaces, more especially from the skin and mucous membranes. Their basis substance is formed of connective tissue, which is continuous with that which normally exists in the part; whilst the free surface is covered by an epithelium, which may vary in thickness and its number of layers according to the seat of the tumor. Blood-vessels and even nerves enter into the interior of the papillæ."[1] These papillary growths vary in size from a pin's-head to a cherry, and may even attain a larger size; but after reaching a certain magnitude their growth sometimes ceases spontaneously. They grow rather quickly, especially in their early stages. To the naked eye they have a rough lobular laminated appearance, and they are generally soft and even friable to the touch. Dr. Andrew Clark has kindly made microscopic examinations of many of the growths of this kind, which I have removed during life with the aid of the laryngoscope. He has described them generally as "consisting of more or less perfect connective tissue, clothed with many layers of epithelium." In some of them "enlarged racemose glands, the terminal vesicles of which were filled with minute nucleated cells and granular matter," were observed. Dr. Andrew Clark thus described one case:—"The growth was found to consist of two sets of particles, one membranous, the other warty or obscurely papilliform. The membranous portions consisted of from twenty to thirty layers of scaly epithelium, surrounded and penetrated by a confervoid growth. The epithelial cells composing the layers were polygonal, flattened, nucleated, and easily affected by weak alkalies and acids. The nucleus of each cell was oval, abruptly defined, rather large in proportion to the containing cell, in most cases surrounded by a clear halo, and in some

[1] See specimen No. 28, Series xxv., in the Museum of St. Bartholomew's Hospital.

[1] Lectures on Surgical Pathology By James Paget, F.R.S. 3d edition, p. 591. For an elaborate description of these growths, the reader is referred to Virchow's Krankheiten Geschwülste, vol. i. p. 334 *et seq.* This eminent pathologist regards Papillomata as a sub-order of his large division of Fibromata.

showing signs of division. The papillary portion consisted of simple outgrowths of nucleated connective tissue, and rudely-formed bloodvessels, clothed with numerous layers of scaly epithelium, similar to those already described. Some of the papillæ exhibited large vacuoles or spaces filled with colloid matter, which in one or two instances had burst through the covering epithelium." Papillomata show a greater disposition to recurrence than other growths.

Fibromata.—The fibrous tumors of the larynx, like those occurring elsewhere, are found to consist of bundles of white fibres diverging and interlacing in various directions. They seldom attain a larger size than a hazel-nut, and when removed show no disposition to recurrence.

Fibro-cellular Tumors of the larynx are comparatively rare. Their growth is rather slow, but they sometimes attain a huge size. "They consist," says Mr. Paget, "of delicate fibro-cellular tissue, in fine undulating and interlacing bundles of filaments. In the interstitial liquid or half-liquid substance, nucleated cells appear imbedded in a clear or dimly granular substance."[1] Unlike the mucous polypi of the nose, they exhibit no disposition to recurrence.

Cystic Growths are still more uncommon in the larynx. I have only met with two cases—both situated on the epiglottis. They are, however, occasionally found near the ventricular orifice. They contain a thick, white, semi-fluid sebaceous material, which on microscopic examination is found to consist of epithelial cells undergoing fatty degeneration, with perhaps a small amount of the proper secretion of the glandulæ. When thoroughly emptied they show no disposition to form again.

The other kinds of benign tumors, such as Lipomata, Vascular Tumors, Hydatids, &c., are too rare to require a special description in an article of this kind.

Cancer.—Under Cancer we must consider the two kinds which are found in the larynx. These are (1) malignant epithelial, and (2) encephaloid. (1) Malignant epithelial. Though considered with reference to other morbid conditions of the larynx, epithelial cancer is not common; as compared with many other situations, the larynx must be regarded as a favorite site. The epiglottis is the part most frequently attacked and next to it the ary-epiglottic folds. It often gives rise to large ragged ulcerations. The disease may be primary, or consecutive to disease in adjacent parts—the pharynx, œsophagus, or thyroid gland. Cancer in the larynx is seldom secondary, in the sense that the term is generally employed by pathologists. (2) Encephaloid cancer is less common than the epithelial variety, but, like it, it is often consecutive. It is characterized by its greater tendency to induce interstitial exudation and consequent thickening, and perhaps by its lesser proneness to ulceration. As already remarked, it often produces considerable displacement.

In addition to the different forms of morbid growth here described, others are said to have occasionally existed in the larynx. Ryland quotes a case of "hydatids" in the ventricle of the larynx, from Andral, which gave rise to the symptoms of a foreign body at that part.[1] The same author speaks of "cartilaginous tumors" in the larynx, and gives several illustrations of supposed tumors of this sort. There is no reason why enchondroma should not be developed in this part, but it is exceedingly rare, and has not been observed by any pathologist of note. Erectile tumors are described as occurring in the larynx, by Rokitansky.

Prognosis.—The prognosis as regards a fatal termination depends on the nature of the growth, and as regards recovery of function, on whether the growth can be removed. Cases of true cancer of course, always prove fatal: whilst the other kinds of tumor ought never to do so.

Therapeutics.—Small growths not giving rise to functional disturbance, and showing no disposition to increase in size, need not be interfered with; but where the neoplasm is large, and where it shows a disposition to grow, it should, if possible, be removed with the aid of the laryngoscope. This removal may be effected by evulsion, for which various instruments are suitable. Most to be recommended are forceps of different lengths and opening in different directions; thus, for a growth on the ventricular band, a pair of short forceps opening in the lateral direction is required, whilst for a growth on or below the vocal cords a much longer forceps, and opening in the antero-posterior direction are indicated. My tube-forceps are very useful, especially if the larynx is small. *Ecraseurs* are not, as a rule, to be recommended, but Stoerck's *écraseur*, which has a rigid metal loop protecting the wire, is, however, often serviceable. No force should be used in the evulsion of growths. Where they cannot be removed without undue effort, crushing of the growth will often cause atrophy. In some cases, the base of the growth may be incised with the laryngeal lancet; or if the tumor be too large for this method, forceps having a cutting edge may be employed with perfect safety and with the best results. Galvano-cautery, on ac-

[1] Op. cit. p. 456.

[1] Ryland, Diseases of the Larynx, p. 226.

count of the pain it generally causes, and the risk of subsequent inflammation, cannot be recommended. Caustic solutions I have not found to be of any service except in those cases where the growths, small in size and symmetrical in situation, are of the nature of condylomata. In these cases the tumors possess but a very feeble organization, and are often dispersed by the application of caustic or astringent solutions.

Should the growth be very large and threaten suffocation, and should severe spasm be induced by attempts at removal through the upper opening of the larynx, the operation of tracheotomy should be performed; the neoplasm may be afterwards removed, either with the aid of the laryngoscope or by division of the thyroid cartilage. In cancer, relief can sometimes be obtained by the inhalation of simple hot steam, or steam impregnated with various sedative principles, as recommended in the treatment of Acute Laryngitis. I have seen a few cases in which removal of a malignant growth, situated so as to seriously impede respiration and deglutition, has been attended with very great temporary relief. One such case is reported in Pathological Transactions, vol. xxi. p. 53.

Neuroses (Nervous Affections of the Larynx).

Under this head are included—(1) Diseases of the Motor System, and (2) Diseases of the Sensory System.

Diseases of the Motor System.

This division embraces—first, Paralysis of the Muscles of the Vocal Cords; and secondly, spasm (or Spasmodic Approximation) of the Muscles of the Vocal Cords.

The varieties of paralysis are so numerous, and their nature so different in different cases, that it is better to consider them separately under the following heads: (1) Paralysis of the Adductors of the Vocal Cords; (2) Paralysis of the Abductors of the Vocal Cords. These may again be divided into (*a*) unilateral and (*b*) bilateral paralysis.

Bilateral Paralysis of the Adductors of the Vocal Cords.

Definition.—A condition in which, owing to the non-approximation of the vocal cords on attempted phonation, there is loss of voice.

Synonyms. — *Latin* — Paralysis Glottidis, Aphonia Paralytica, Aphonia; *French* — Aphonie; *German* — Kehlkopflähmung; *English*—Functional Aphonia, Hysterical Aphonia, Aphonia.

Causes.—Debility and hysteria are the most frequent causes of this condition; it often, however, originates in congestion, and remains after the hyperæmia has passed away. The rare cases of intermittent aphonia dependent on malarious influences, which have been reported,[1] probably belong to this category. I have once seen it caused by extensive cerebral disorganization from a tumor at the base of the brain.

Symptoms.—The condition is seen with the laryngoscope, on directing the patient to attempt to produce some vocal sound, —that is, to try to say "*ah*" or "*e*;" the vocal cords may not move, or may approach each other only very slightly—in all cases remaining distinctly apart. As the vocal cords remain at the side of the larynx, the condition might be called "bilateral paralysis with lateral fixture." The laryngeal mucous membrane is generally pale. The voice, of course, is always suppressed. It is only the voluntary action of the adductors of the vocal cords which is impaired; the involuntary or reflex movements, especially those of a forcible character, are not generally affected. Thus, coughing and sneezing are usually accompanied with *sound*, showing that the cords approximate. In laughing, however, where the expirations are much less forcible, especially in feeble people, there is often no sound. In other words, these patients do not laugh, but only smile, the term "laughter," strictly considered, being an audible manifestation. The constitutional condition is such as has been already indicated under the head of the Causes of the local phenomena.

Pathology. — The pathology of the disease probably consists in the "nerve-force" being feebly or imperfectly evolved, or not directed in the proper channel: there is no lesion here. The muscles, which are paralyzed, are the crico-arytænoidei laterales on both sides, and the arytænoideus proprius.

Prognosis. — The prognosis is very favorable in almost all cases.

Treatment.—The treatment consists in the use of local remedies which tend to excite approximation of the vocal cords. Stimulant solutions were formerly recommended, but faradization is a far more effective remedy. One pole should be applied over the thyroid cartilage externally,

[1] Valleix, Bulletin de Thérapie, 1843.

the other one to the vocal cords. My "laryngeal electrode"[1] will be found very useful. With it, I have cured aphonia of eight, and even ten years' standing. The instrument has been also successfully used by other laryngoscopists, both in this country and on the Continent. The patient's health may generally be benefited by constitutional (tonic) remedies.

Unilateral Paralysis of the Adductors of the Vocal Cords.

Definition.—A condition in which, owing to one of the vocal cords not being drawn to the median line on attempted phonation, there is loss of voice.

Causes.—The condition may be caused by local injuries, may occur in chronic toxæmia (lead and arsenic), or may be due to cerebral disease, or pressure on the pneumogastric or its recurrent branch. It is difficult to say whether its occurrence as a sequel of diphtheria is due to the first or second cause.

Symptoms.—The condition is seen with the laryngoscope. On attempted phonation one vocal cord remains at the side of the larynx, whilst the other is drawn to the median line. The condition may be described as "unilateral paralysis with lateral fixture." The mucous membrane covering the affected cord is generally congested. There is aphonia or dyspnœa, and usually an absence of constitutional symptoms. When the paralysis is complete, or even much marked, the acts of coughing, sneezing, and laughing are always altered in character, and often unaccompanied by sound: indeed, a modification of the natural cough or sneeze is sometimes one of the earliest symptoms of the condition. When the unilateral paralysis is accompanied with loss of power of the same side of the tongue and palate, it indicates serious cerebral disease near the nucleus of the spinal accessory nerves.[2]

Pathology.—As regards the pathological anatomy, I may observe, that in the only case of this disease—a case of seven years' standing, which I have examined after death, there was considerable atrophy of the crico-arytænoideus lateralis on the affected side. The arytænoideus proprius did not seem to suffer. The disease is probably often due to inflammatory exudation, either of a simple or dyscrasic character, into the substance of the muscle. When the pneumogastric nerve or its recurrent branch are pressed upon, the abductor muscle is always so much more affected than its antagonist, that the function of the adductor seems to be little affected.

Prognosis.—The condition not being in itself dangerous, and being generally due to local causes, need not, as a rule, give rise to serious apprehensions. If there is evidence, such as paralysis of other parts, to show that the disease is due to cerebral causes, the prognosis is, however, serious. It is always very difficult to cure.

Treatment.—This should be the same as that recommended for bilateral paralysis of the adductors; it is not, however, generally so successful. When resisting the action of my ordinary electrode, the "No. 3 laryngeal electrode," by means of which one pole can be passed into the larynx and the other into the hyoid fossa, so that the current passes through the crico-arytænoideus lateralis, may be employed. Constitutional remedies may be used with advantage in cases of chronic toxæmia.

Bilateral Paralysis of the Abductors of the Vocal Cords.

Definition.—A condition in which, owing to the vocal cords not being drawn aside (but remaining fixed near the median line) in inspiration, there is great dyspnœa and stridulous breathing, without much alteration in the character of the voice.

Causes.—The causes of this condition are generally cerebral, but morbid influences which affect both pneumogastric or both recurrent nerves may give rise to it. In a case of ex-ophthalmic goitre, I once saw it caused by an enlarged and constricting thyroid gland, which passed round the trachea and pressed on both recurrent nerves; scrofulous deposit in the bronchial and cervical glands, especially in children, is apt to give rise to it. In cancer of the œsophagus, when the deposit affects the anterior wall of that tube, both the recurrents may be involved. It is, however, most commonly caused by central disease of the nervous system. It sometimes depends on simple degeneration of the muscles, without there being any evidence of implication of the nerves. The condition is fortunately very rare.

Symptoms.—On making a laryngoscopic examination, the vocal cords do

[1] Made by Mayer, 59 Great Portland Street, W.

[2] See Dr. Hughlings Jackson's valuable Illustrations of Diseases of the Nervous System. Lond. Hosp. Reports, vol. i. p. 361, and vol. ii. p. 330.

not separate at all on inspiration. There is a slight interval between them, which alters very little, except in forced expiration, as when the vocal cords approximate completely. As the vocal cords always remain near the median line in this form of paralysis, it might be called "bilateral paralysis with central fixture." The vocal cords are generally slightly congested. The voice is usually but little affected, but it may be rather harsh: if the patient does not move at all, the respiration may be little affected; the least exertion, however, brings on dyspnœa and stridulous breathing. The cough is croupy. The condition is in itself apt to produce constitutional symptoms—such as wasting and febrile excitement; and it is often accompanied by paralysis of other parts, or by the cachexia of the disease which indirectly causes it. In children, it produces the symptoms of laryngismus stridulus, and Dr. Ley considered that laryngismus was always of a paralytic nature, and always due to the same cause—namely, pressure on the recurrent nerves. True laryngismus depends on other causes which operate in an opposite way.[1] The paralysis of the abductors of the vocal cords, which produces symptoms of laryngismus, is found in children of a more advanced age than those who are attacked by the ordinary form of laryngismus—that is, by spasmodic laryngismus; of course, however, the paralytic form may also occur to the youngest infants. It differs also, inasmuch as the symptoms do not completely pass away; exacerbations may occur, but there is always a certain amount of constant stridor and dyspnœa.

Pathology and Morbid Anatomy.—The pathology of the disease has, to a certain extent, been discussed in considering its etiology. The disease consists essentially in a loss of power of the arytænoidei postici, the powerful abductors of the vocal cords, and is dependent on the interception or non-generation of the nerve current which, through the medium of the pneumogastric and it branches, supplies those muscles in the normal state. In the case of a patient under the care of Dr. Hughlings Jackson in the London Hospital, where I had diagnosed bilateral paralysis of the abductors during life, these muscles, when examined by Mr. Rivington after death, were found to be greatly atrophied. There is probably, generally, also atrophy of the nerve structure.

Prognosis.—The prognosis is very serious both on account of the immediate danger of suffocation implied by the condition, and from its being sometimes an indication of some very serious disease elsewhere, either in the brain or along the trunks or branches of both pneumogastric nerves. The condition is in itself highly dangerous; for though the simple action of the adductors (the abductors being paralyzed) is not generally sufficient to close the glottis completely, the addition of a little inflammatory swelling or œdema would soon bring about that state.

Treatment.—The operation of tracheotomy should be performed without delay, to save the patient from dying of suffocation. The operation would be likely to exercise a favorable effect on the cerebral disease, for the indirect influence of the exceedingly narrowed glottis (through the respiratory system) on the cerebral circulation must be highly injurious. I cannot recommend any medical treatment either of a local or general character.

Unilateral Paralysis of the Abductor of one Vocal Cord.

Definition.—A condition in which, owing to one vocal cord not being drawn aside (but remaining near the median line) on inspiration, there is some dyspnœa and stridulous breathing, without much alteration in the character of the voice.

Causes.—The causes which lead to paralysis of one abductor are the same as those which produce the bilateral form of paralysis, but the condition now under consideration is more often due to peripheral causes; that is to say, to pressure on one pneumogastric or one recurrent nerve. Aneurisms of the arch of the aorta by pressure on the left recurrent nerve not unfrequently produce this kind of paralysis of the left vocal cord;[1] and in the year 1866 a case of aneurism of the right carotid occurred in my practice, in which the right vocal cord was paralyzed. Cancerous tumors occasionally involve the pneumogastric or its branches, and the strumous glands along the trachea may do so likewise. In malignant stricture of the œsophagus, when the disease affects the anterior wall of that tube, one of the recurrent nerves is occasionally affected.

Symptoms.—The condition can be observed with the aid of the laryngoscope; on directing the patient to inspire, the affected vocal cord is not drawn to the

[1] See Spasm of the Vocal Cords, p. 448.

[1] Med. Times and Gaz., January, 1864, and Pathol. Transactions, vols. xvii., xix., and xxi.

side as in the normal state; its inner edge, however, is not quite in the median line. The vocal cords are generally congested. The condition may be described as "unilateral paralysis with central fixture." There is stridulous breathing and dyspnœa on the slightest exertion, but the last two symptoms, as might be expected, are not quite so severe as where both cords are affected. The constitutional symptoms vary with the different conditions which give rise to this form of paralysis, but this kind of glottic obstruction generally after a time causes symptoms of irritative fever.

PATHOLOGICAL ANATOMY.—The immediate nature of the disease and condition of the nerves and muscles is the same as that which is found in bilateral paralysis with central fixture, but here the disease only affects one side. In several cases which I have brought before the Pathological Society of London,[1] the muscle of the affected side has been seen to be completely wasted—only a few of its inner and lower fibres remaining, whilst its fellow on the opposite side was healthy and well nourished. In some of these cases the left recurrent nerve has been so completely incorporated in a cancerous or an aneurismal tumor, that its course (after entering into the tumor) could not be traced.

PROGNOSIS.—The condition is generally indicative of very serious disease elsewhere, and the most unfavorable opinion should be given as to the prospects of the case.

TREATMENT.—There is generally little to be done towards the cure of the disease: tracheotomy should be performed if the symptoms of suffocation are at all urgent.

VARIETIES.—In addition to the more palpable forms of paralysis which on the one hand produce aphonia, and on the other lead to suffocation, there are certain states in which loss of power is manifested, by the inability to produce certain notes in singing. Here the crico-thyroid or thyro-arytenoid muscles—muscles, the action of which, though generally supposed to be antagonistic, is probably, in point of fact, co-ordinate—are generally at fault. The limits of this article render a detailed handling of this difficult subject impossible; but for further details the reader is referred to my pamphlet on the subject.[2] The prognosis, as regards cure, must depend on the age of the patient, the duration of the condition, and the natural character of the voice (whether it be tenor or bass). The treatment must sometimes be stimulant (electricity, astringent solutions, &c.), at other times sedative.

[1] Pathological Transactions, vols. xvii., xix., and xxi.

[2] Hoarseness and Loss of Voice. Churchill, 1868.

Spasm (or Spasmodic Approximation) of the Muscles of the Vocal Cords.

DEFINITION.—A condition in which there is sudden temporary complete or incomplete approximation of the vocal cords, characterized in the former case by arrest of the respiratory movements and apnœa, in the latter by stridulous inspiration and dyspnœa.

SYNONYMS.—*Latin*—Laryngismus stridulus, Laryngitis stridula, Spasmus glottidis, Cynanche stridula, Cynanche trachealis, spasmodica, Asthma Koppii, Asthma Millari, Asthma intermittens infantum, Asthma thymicum; *French*—Laryngite striduleuse, Faux croup, Pseudo-croup nerveux, Spasme de la Glotte; *German*—Kehlkopfkrampf, Stimmritzenkrampf (Cerebral Croup, Pseudo-Croup); *English*—Millar's Asthma, Crowing Inspiration, Child-crowing, Spasm of the Glottis, Spasmodic Croup, Spurious Croup, Cerebral Croup, &c. &c.

Causes.—The causes of spasm of the vocal cords are involved in a considerable amount of obscurity, and there is evidence to show that many influences may be concerned in its production; hence it is not surprising that the etiological features concerning it should have undergone various changes and modifications.

The causes may be divided into (1) central, and (2) peripheral—the latter being subdivided into (*a*) direct, and (*b*) reflex.

1. The disease was at one time considered to be always dependent on cerebral disease, or at least on a disordered state of the functions of the brain,[1] and this view, which has been assailed in various ways, seems to be again gaining ground. Numerous cases are on record, where other admitted symptoms of cerebral disease manifested themselves before the occurrence of laryngeal spasm. Limited congestion or interstitial exudation of serous fluid, near the origin of the pneumogastric nerves, is probably the condition of the brain which is concerned in the production of this phenomenon. In many cases, however, the structural alteration of the brain, if present, is of too delicate a nature for detection, and still more frequently a morbid condition of that organ is produced by the sudden apnœa. Hence, even when the brain is the primary seat of the disease, it is impossible

[1] Commentaries on Diseases of Children, by Dr. John Clarke.

to speak with certainty as to the nature of the morbid condition. The cerebral affection is probably often dependent on a dyscrasic state.

A rachitic condition of the bones of the skull has frequently been noticed. Out of ninety-six cases of laryngismus examined by Lederer, there was craniotabes in ninety-two.[1] The experience of Sir William Jenner and Dr. Wiltshire is of a similar character.[2] It has been suggested that the thickness of the cranial bones in rickets allows pressure to be exercised on the brain in the occipital region, when the child lies on its back (Elsässer); but it is more probable that the rachitic dyscrasia is accompanied by morbid changes of a nutritive character in the structure of the brain itself.

Scrofula has also been regarded as an active predisposing cause of the disease (Marsh). Sometimes an attack is brought on by tossing the child in the air, and it still more often comes on in sleep. These facts have been adduced by some as an evidence of the cerebral nature of the disease; but it must be remembered that both in sleep and in sudden movements of the body the function of respiration not less than the cerebral circulation is modified, and that the spasm of the glottis may originate in either process. Disease of the cervical portion of the spinal cord sometimes gives rise to it (Marshall Hall). In cases of disease of the brain or medulla, external pressure applied over these parts has been known to cause laryngismus. Hydrocephalus exists in some cases; and mental emotion—especially terror and rage—occasionally gives rise to the spasm.

2. (*a*) Direct pressure on the recurrent or pneumogastric nerves by enlarged and tuberculous cervical and bronchial glands has since Dr. Ley's time been regarded as a cause of laryngismus, but in these cases the cause is probably (as Dr. Ley conceived) "paralysis of the dilators of the glottis." Enlargement of the thymus gland was at one time, especially in Germany, considered the essential cause of laryngismus,[3] but at present its influence is considered to be of a very exceptional character. In so far as these causes operate by producing paralysis of the abductors of the vocal cords they belong to the last section of neuroses, but they probably often cause spasm of the adductors by obstructing the venous circulation through the neck, and thus giving rise to cerebral irritation.

(*b*) Amongst the reflex causes of spasm we have those acting directly on the larynx and those operating at a distance. Attacks not unfrequently come on whilst the child is sucking, or rather swallowing, and there can be little doubt that the cause here is the passage of liquid into the larynx. Spasm produced by dangling the child in the air is probably caused by the impression of a current of air on the glottis. Amongst the reflex causes of laryngismus which act at a distance, there is the irritation of teething, the presence of indigestible food or helminthoid parasites in the alimentary canal, and the impression of currents of cold air on the integument. It sometimes supervenes on the cure of a protracted diarrhœa or a chronic skin affection, but these causes probably act by setting up cerebral irritation. It has been noticed by Sir William Jenner[1] that the mother's health has an important influence in the production of rickets, and Kopp has made precisely the same observation with regard to laryngismus. Here there is another link towards the chain of association which Sir William Jenner has attempted to establish between these two morbid conditions. The greater liability of the male sex, which occurs in other laryngeal diseases, holds good here. The disease is most frequent between the ages of six months and two years.

Symptoms.—The age of the patient destroys the value of subjective symptoms, but those of an objective character are sufficiently marked. The following is the common history of a first attack. A child put to bed, apparently in its ordinary state of health, wakes up suddenly at about midnight with difficulty of breathing, inspiration being accompanied by a crowing noise similar to that heard in croup. After two or three of these stridulous inspirations, the frightened child bursts out crying and in a few minutes is fast asleep again, as if nothing had occurred. This description does not apply to every case. The child may have been peevish and fretful for a few days before, may have suffered from loss of appetite, and may have been restless at night, or a slight "catch" in the breath may have been previously noticed. The first attack may come on at any other time, but it most frequently occurs during sleep. The next day the child may be quite well, and there may be no further return of the symptoms, but it often happens that another attack comes on about the same hour the following night. The second attack is generally more severe than the first, both in its character and duration. In severe cases, indeed, the paroxysms are of a most urgent kind and of the most frequent occurrence. A severe fit of

[1] Rühle, Kehlkopfkrankheiten, p. 201. Berlin, 1861.

[2] See art. Rickets, vol. i. p. 472.

[3] Kopp, Denkwürd. in der ärzt. Prax. Frank., 1820.

[1] Op. cit.

laryngismus may be thus described: the breathing suddenly becomes greatly embarrassed, each act of inspiration being much prolonged and accompanied by a harsh stridor: suddenly the sound ceases, the glottis is completely closed, and the respiratory movements of the chest are suspended. The flush which first lit up the countenance gives way to pallor and afterwards to lividity. The eyeballs roll, the veins of the neck are turgid, the fingers close on the thumb, which is bent in the palm, and the hands are flexed on the wrist; spasm likewise affects the feet, the great toe is drawn away from the other toes, and the foot is flexed and rotated slightly outwards. These so-called "carpo-pedal" contractions are probably sometimes accompanied with great pain. The disease, indeed, may partake of the character and assume the form of epilepsy. Notwithstanding the severity of the paroxysm just described, the patient may survive it, the apnœa being succeeded by stridulous breathing, and by relaxation of the spasmodic contractions of the feet and hands; but when the symptoms are of the dangerous character just described, the paroxysm is sure to be quickly followed by others—in one of which the child dies. The severity of the attacks varies between the mild paroxysm which has been described as occurring at the commencement of the disease, and one sufficiently intense to cause death. The spasm is characterized by its sudden occurrence and by its complete remission, as a rule by the entire absence of febrile irritation, and by the progressive severity of the spasm, as regards recurrence, duration, and intensity. Some of the associated symptoms of laryngismus may likewise be present, such as hydrocephalus, a rachitic condition, or enlargement of the thymus body.

Diagnosis.—The non-febrile and distinctly intermittent nature of the affection differentiates it from true croup, and its own distinctive characters from all other diseases.

Pathology.—The pathology of the disease has been considerably encroached upon in considering its causes, but there still remains something to be said concerning its nature. There are two points on which it appears to me necessary to insist: these are (1) that in all cases there is an altered state of the nerve-centres; and (2) that the immediate cause of the phenomena of the stridulous inspiration and apnœa is spasm of the adductors of the vocal cords. The facts which point to an alteration in the brain substance (whether recognizable or not) are first, that both sides of the body (both vocal cords) are affected; secondly, that the various causes (such as dentition, indigestion) are not only often in operation without the production of laryngeal spasm, but when they do give rise to that symptom they necessarily act through the brain; thirdly, that frequently other admitted symptoms of cerebral irritation, such as the carpo-pedal contractions, are present. That the disease depends on spasm of the adductors of the vocal cords appears probable for the following reasons:—(1) The other phenomena are those of spasm (carpo-pedal contractions). (2) Complete closure of the glottis never takes place under physiological conditions, and therefore it is improbable that the simple action of the adductors of the vocal cords could cause complete closure of the glottis (the action of the abductors being in abeyance); in support of this view I may observe that in three cases of paralysis of the crico-arytenoideus posticus which have come under my notice, the inner edge of the affected cord was not adducted to the median line. (3) The total remission of so urgent a symptom points to its cause being of a spasmodic nature; there being, as far as I am aware, no instance of complete paralysis of a truly paroxysmal character.

Prognosis.—The prognosis depends on the character of the paroxysm and its supposed cause. The cases mainly dependent on reflex causes (dentition or irritation of the alimentary canal) generally do well, whilst those due to direct pressure, and those mainly caused by cerebral irritation are more frequently fatal. Thymic asthma is especially dangerous, and if there is evidence (such as considerable enlargement of the gland) to show that the spasm is of that character, the most unfavorable opinion must be given. The length of the intervals between the paroxysms is a good prognostic guide—the longer the interval the better the chance of recovery.

Treatment.—The treatment must be twofold: first, to relieve quickly the spasm of the glottis; secondly, to attack the source of the disease. The immediate treatment generally falls to the nurse or mother. The little patient should be raised and placed in a sitting posture, and then he may be slapped on the back, cold water may be dashed in the face, and ammonia or strong acetic acid held to the nose. These measures are often successful by giving rise to violent expiratory actions; but remedies calculated to relieve spasm are equally successful. The warm bath may be used and emetics given directly there is a sign of the stridor—when the paroxysm is on, the child will not drink. A favorite remedy in Germany, and one that is highly successful, is tickling the fauces with the finger or a feather until vomiting is produced. De-

pressing enemata, such as tobacco, have likewise been recommended, but their use is attended with considerable danger. The ordinary rules for the treatment of disease apply here; that is to say, gentle remedies should be used in mild cases and those of a more powerful character in dangerous ones. Putting the lower part of the child's body in a hot bath and dashing cold water in its face is a simple and sometimes successful plan. The inhalation of chloroform is a very valuable remedy, but of course must be used with great care, and cannot safely be employed by non-professional persons. If the child appears to be sinking from the apnœa, the trachea must, of course, be opened, and artificial respiration resorted to. Indeed this should even be adopted by the practitioner, should he arrive shortly after the apparent extinction of life. Some practitioners recommend the use of antispasmodic remedies (whether animal, vegetable, or mineral) between the fits. As regards the *fons et origo mali*, the most suitable treatment will be found detailed in the various articles in these volumes which treat of scrofula, rickets, hydrocephalus, dentition, parasites, &c. Enlargement of the thymus must be treated by the application of leeches (according to the age and strength of the patient), and afterwards by counter-irritation.

VARIETIES. — The ordinary kinds of laryngismus, according to my views, are essentially due to spasm of the adductors of the vocal cords, but that variety which is caused by pressure on the pneumogastric or recurrent nerves is due to paralysis of the abductors. It has been treated of in the last section of neuroses; and differs from ordinary laryngismus, in the ways there indicated. It appears to me that Dr. Ley was right as to the cause of the symptoms of a certain form of laryngismus, but mistaken in regarding a rare variety as a typical example. This view explains the very opposite opinions which have been held concerning the etiology and pathology of the disease.

I have thought it more convenient to treat spasm of the glottis as an infantile affection, but it must be borne in mind that it sometimes occurs to adults. Women are generally the subjects of it; and it is commonly regarded as an hysterical phenomenon. In one case, however, that came under my notice, there were no symptoms whatever of hysteria, the stridulous inspiration being so much worse during sleep, that the patient, a woman in the London Hospital, was obliged to be placed in a separate room, on account of keeping the other patients awake. In this case, though the rest of the mucous membrane was much congested, the vocal cords were perfectly healthy. The case recovered under the local treatment recommended under the head of Chronic Laryngitis.[1]

The treatment should be the same as that advised for children, though inhalations of sedative and anæsthetic vapors may here be employed with advantage. Spasm of the glottis, dependent on the inhalation of poisonous gases and the impaction of foreign bodies in the œsophagus, requires the most prompt treatment; if not immediately relieved, laryngotomy or tracheotomy should be performed without delay. One form of spasm of the vocal cords is that met with in hooping-cough —the essential phenomena of this complaint being a series of short, rapid, and violent expirations, followed by a prolonged stridulous inspiration—the disease which will be found treated in detail elsewhere.[2] The laryngeal cough, sometimes met with in hysterical women whose larynx is seen with the laryngoscope to be perfectly healthy, is also due to a spasmodic tendency of the adductors of the cords, the spasm only occurring in expiration; and the same may be said of the sharp ringing cough which occasionally affects children, and is usually looked upon as of a reflex nature. The nervous laryngeal cough of adults is as difficult to treat as most hysterical complaints. I have found the most satisfactory results follow from the use of warm sedative and anæsthetic inhalations; but the results are often disappointing. Lasègue reported a case successfully treated by belladonna;[3] but in a severe case that came under my care, atropine was given till its full physiological effects were produced, but without relief of the cough. Dr. Harley has reported a case[4] in which valerianate of zinc effected a cure.

DISEASES OF THE SENSORY SYSTEM OF THE LARYNX.

Hyperœsthesia.

Increased sensibility occurring independently of inflammatory disease or structural alteration of the tissues of the larynx, is undoubtedly a rare morbid condition, but it may occur either in an intermittent form or without any periodic character. A case of the former kind is reported by Dr. Gerhardt,[5] and a few of the latter have fallen under my notice. Several cases have also been reported by Dr. Handfield Jones.[6] Neuralgic cases

[1] Med. Times and Gazette, Nov. 15, 1862.
[2] Vol. i.
[3] Archives Générales, May, 1854.
[4] Med. Times and Gazette, vol. ii. p. 116.
[5] Virchow, Archiv xxvii. Heft 1 and 2.
[6] Med. Times and Gazette, May 2, 1863.

should be treated on the ordinary principles which regulate the therapeutic management of such cases. The inhalation of hot sedative vapors and anæsthetics does good in cases of a non-intermittent character; and the internal use of narcotics is also indicated.

Some of the morbid phenomena already referred to under the head of Motor Affections (such as pertussis and nervous laryngeal cough), may be due to increased sensibility of the mucous membrane of the vocal cords—the hyperæsthesia manifesting itself in reflex action.

Anæsthesia.

Although there is great difference between the sensibility of the glottis in different people, anæsthesia rarely occurs as a distinct morbid affection.

Disease affecting the origin or trunks of the pneumogastric nerves or their superior laryngeal branches, would be likely to diminish the sensibility of the larynx in proportion as the function of the nerves was interfered with.[1] Romberg has observed that in cholera there is impaired sensibility of the mucous membrane of the larynx.[2] Some morbid phenomena of a functional character, such as a vocalist's inability to produce certain notes which previously could be easily formed, are probably in some cases (where the larynx appears healthy) due to impaired muscular sensibility.

SECTION II.

Secondary Diseases of the Larynx in Acute Affections.

SMALLPOX.

The laryngeal affection may be a mild papular or pustular eruption of the mucous membrane, or it may be a severe inflammatory disease accompanied by the presence of false membrane. The former, as a rule, causes little or no inconvenience; the latter is often fatal. In the year 1863, through the courtesy of Mr. Marson, the author of the able article on Smallpox (vol. i. p. 127), I was enabled to examine several patients in the Smallpox Hospital, with the laryngoscope. In one patient laboring under severe purpuric smallpox, I found ecchymotic spots on the under surface of the epiglottis, and on the mucous membrane over the arytenoid cartilages. In a convalescent case, there was a distinct pustule on the edge of the epiglottis; in another case, in which the entire body was covered with pustules, the larynx appeared perfectly healthy, and in another similar case there were no pustules, but there was a marked congestion of the mucous membrane; in another case, the upper surface of the epiglottis was covered with pustules. Rühle, who in a bad epidemic of smallpox, in Greifswald, in 1856–57, made no less than fifty-four post-mortems, observes,[1] "Although I have seen here and there pustule-like elevations, I nevertheless consider the essential peculiarity of the laryngeal affection to be of a croupous or diphtheritic inflammation." Dr. Rühle further observes, that, as "out of the fifty-four cases there was not a single case in which the larynx and windpipe were in a normal state, he cannot but attribute a certain proportion of the mortality to the laryngeal affection." Pathological examples of the diphtheritic complications of smallpox are to be found in the museums of St. Thomas's and St. Bartholomew's Hospitals, and in other collections. In two instances, I have known permanent paralysis of the adductor of a vocal cord follow smallpox: in both, the larynx was affected at the time, and it is probable that the affection was of the diphtheritic character. As regards treatment, it may be observed that in the pustular form of the diseases interference is unnecessary, and that in the diphtheritic form it is almost useless: in the latter case, however, the local treatment elsewhere recommended for primary diphtheria can be adopted.

MEASLES.

In this disease the affection of the larynx may be either a simple catarrh, or a severe croupous affection.

The catarrhal form of laryngitis may occur before the eruption appears, a day or two after the rash has come out, or when it is beginning to decline. It is more common than the croupy form of disease; and though occasionally the inflammation runs high, it is seldom of any importance. In some epidemics, catarrhal laryngitis comes on when the eruption has almost disappeared.[2] In these cases, there is generally obstinate hoarseness. In a number of Professor Hebra's patients in the General Hospital at Vienna, in different stages of measles, Dr. Stofella[3] found a highly injected condition of the mucous membrane of the larynx in almost all the cases which he examined laryngoscopically.

[1] Hufeland's Journ. der pract. Heilkunde, Feb. 1853.

[2] Ibid. Feb. 1832.

[1] Op. cit. p. 247.

[2] Bohn, Königsberg Mediz. Jahrbücher, 1858.

[3] Wien Medizin. Wochenschrift, Nos. 18, 19, 20, 1862.

The croupy or diphtheritic form of inflammation, observes Dr. West, "seldom begins until the eruption of measles is on the decline, or the process of desquamation has commenced. Its appearance is most frequent from the third to the sixth day from the appearance of the eruption, but it oftener occurs at a later than an earlier period."[1] The treatment should be similar to that recommended for primary croup, but it must always be borne in mind that the secondary disease is of a less sthenic type.

SCARLATINA.

The affection of the larynx in these cases may be either an acute œdema of the glottis or a croupous inflammation: they are, fortunately, both rare complications. The œdema which sometimes occurs in scarlet fever may be dependent on the debility which exists during the convalescence of severe febrile complaints, or may be due to the renal affection which sometimes follows scarlatina.

The croupy inflammation of the larynx, though not common, is peculiar to some epidemics. Goupp described an epidemic in Wurtemberg, in which, in the greater number of cases, croupy symptoms appeared from the third to the fourth day of the illness; in some cases death took place before the exanthem appeared.[2] It has been observed, that in diseases of the larynx dependent on, or associated with, scarlatina, there is a great tendency to the ulcerative process. A specimen (No. 36, series W), in the Museum of St. Thomas's Hospital, supports this view. The larynx was taken from an adult patient, who died of scarlatina: there is a very thin layer of lymph covering the mucous membrane of the larynx, and the right arytenoid is laid bare by a large ulcer.

The treatment of the plastic form of inflammation should be such as is recommended for diphtheria, viz., the internal use and local application of the persalts of iron, a highly nourishing diet, and the free use of alcoholic stimulants, well diluted. The practitioner must always have tracheotomy in view. In œdema, this operation is also likely to be necessary, but scarification should be first tried.

ERYSIPELAS.

In erysipelas of the head and neck there is always more or less congestion of the mucous membrane of the larynx; and even when the erysipelatous inflammation is seated on the limbs, there is sometimes sympathetic or concomitant inflammation of the larynx. It sometimes, though less frequently, occurs in hospital gangrene.[1] It may result in an acute œdema, which rapidly tends towards a fatal termination. The symptoms of the disease are, difficulty of swallowing, hoarseness or loss of voice, and pain; the latter is increased on pressure. Dr. Semeleder has examined five cases with the laryngoscope; in four of them the erysipelas affected the face, and in these he found inflammatory redness and swelling of the epiglottis and larynx down to the vocal cords, though there was no dyspnœa or dysphonia. The inflammatory symptoms in the larynx disappeared gradually with the desquamation of the skin; and in one case a relapse of the cuticular affection was accompanied by a recurrence of laryngeal inflammation. In the fifth case—erysipelas of the lower extremities—there was no hyperæmia of the larynx. The poison of erysipelas sometimes confines itself to the larynx, the skin being free from inflammation; at other times it passes from the larynx to the external parts. Cases are on record, at least, which tend to support these views.[2]

The treatment should be active, and such as has been recommended in ordinary inflammation and œdema of the larynx.

TYPHUS AND TYPHOID.

In *typhus* there is nothing characteristic about the laryngeal affection; congestion of the mucous membrane, plastic deposit on its surface, gangrenous inflammation, and œdema, being conditions which are all occasionally met with. The ulceration is generally of the most destructive character, and whilst it often involves a large surface, it frequently penetrates deeply and exposes the cartilages. It is generally at the posterior parts of the larynx, that is, *at the under part*, in the prone position of a patient with low fever, that the disease is most frequently found; and it is commonly thought to be caused, at least in part, by hypostatic influences. The cricoid cartilage is frequently seen to be denuded, and of a blackish-gray color, and there is frequently a corresponding discoloration of the opposite wall of the pharynx.

In *typhoid* the same conditions are met with as in typhus; but there seems to be a greater liability to œdema, the ulcerative process more often appears to originate in a typhous deposit, — "laryngo-typhous being, as it were," says Rokitansky, "the completion of abdominal typhous;" and

[1] Diseases of Infancy, p. 236.
[2] Rühle, op. cit. p. 243.

[1] Ryland, Diseases of the Larynx, p. 8.
[2] Ibid. pp. 73 to 77.

it is said that the cartilages often become independently diseased, *i. e.* become diseased without the superjacent tissues being primarily affected. So many conditions of the larynx are met with which tend to lead to destruction of the cartilages, that it seems unnecessary to resort to the theory that these structures become independently diseased. If in cases where the cartilages are affected the patient survives the fever, the pathological changes described at page 39 take place, and the case runs the course of laryngeal phthisis. Dr. Wilks has especially called the attention of the profession in this country to the ulceration of the larynx occurring in typhoid.[1]

TREATMENT.—In these cases, where subjective symptoms are often altogether absent, and those of an objective character are to a great extent masked, the dictates of rational medicine should lead us to be prepared by surgical interference (tracheotomy) to prevent death from laryngeal obstruction, rather than to attempt to control or oppose the disease.

SECONDARY DISEASES OF THE LARYNX IN CHRONIC AFFECTIONS.

LARYNGEAL PHTHISIS.

DEFINITION.—Chronic thickening and ulceration of the larynx, usually occurring consecutively to pulmonary phthisis, but sometimes being present before there is any evidence of lung-disease. There is hoarseness or loss of voice, often dysphagia and dyspnœa, with persistent increase of temperature, and continuous wasting of the body.

SYNONYMS.—*Latin*—Phthisis laryngea, Laryngitis chronica, Tuberculosis laryngis, Laryngophthisis, Helcosis laryngis; *French*—Phthisie laryngée; *German*—Kehlkopftuberculose; English—Laryngeal Phthisis, Throat Consumption.

CAUSES.—The causes are the same as those which give rise to other laryngeal affections (such as exposure to cold, functional excesses, &c.), *plus* a special constitutional condition, either inherited or acquired, through which cell-proliferation takes place in the submucous tissues. In ordinary chronic laryngitis the rapid evolution of imperfect cells takes place at the free surface, but in laryngeal phthisis the interstices of the tissue are the seat of deposit. Although Niemeyer has done good service in so decisively combating the idea of the *tubercular* origin of all forms of phthisis, and in pointing out the catarrhal and inflammatory nature of many cases of that disease, there can be no doubt that a disposition to low interstitial inflammation is often inherited, or, at any rate, congenital. The feeble texture is excited to chronic inflammation and cell-proliferation by very slight exciting causes. By the Vienna school, the cell-proliferation was called an "exudation," and probably, in a large number of cases, the deposit is more of this nature than that of a true *growth;* the weak constitution which gives rise to it was called "a diathesis." Our views on phthisis are now undergoing a great change, but however unimportant a *rôle* tubercle may play, that there exists a diathetic predisposition to low inflammatory action cannot be denied. Laryngeal phthisis is often hereditary, and it frequently attacks several brothers and sisters; as in other laryngeal affections, males show a greater proclivity to it than females.

Numerous cases of laryngeal phthisis have come under my inspection, where the most experienced stethoscopists have been unable to discover a trace of lung-disease; but on the other hand, I must admit that I have only three times met with cases of laryngeal phthisis in the dead subject without finding corresponding pulmonary disease.

SYMPTOMS.—*Subjective.*—There is nothing characteristic about the subjective symptoms: they resemble those met with in chronic laryngitis, except that, owing to the thickening of the tissues, the act of deglutition is more often performed with difficulty. Pain is sometimes experienced in swallowing, but it more often happens that the act is difficult—violent coughing coming on from a little food getting into the larynx; sometimes the drink is violently ejected through the nares. The difficulty of swallowing is most extreme when the epiglottis is much thickened; but it also generally occurs when the ary-epiglottic or inter-arytenoid folds are much swollen.

Objective.—(1) *Vocal.*—Dysphonia, or aphonia, is always present; hoarseness being generally the symptom of the early stages, complete aphonia of the later. The aphonia is, of course, generally dependent on structural changes, but it may occur at the commencement of the disease from functional causes (weakened approximative action of the vocal cords, and feeble action of the expiratory muscles). The cough varies in different stages. Sometimes the disease is ushered in with violent and frequent paroxysms of cough which nothing can alleviate; sometimes it is only an occasional dry tickling cough; it is generally aphonic in the later stages of the disease.

[1] Transact. Pathol. Soc. vol. ix. p. 34, and vol. xi. p. 14.

(2) *Respiratory.*—The respiration is at first little affected, but afterwards it becomes embarrassed, and inspiration is often stridulous; mucous râles can generally be heard over the thyroid cartilage and trachea. In the last stage the dyspnœa is so great that tracheotomy occasionally becomes necessary.

(3) *Laryngoscopic Signs.*—In cases of pulmonary phthisis pallor of the mucous membrane is often noticed, and Dr. Semeleder regards anæmia of the larynx, where there is no other cause for its existence, as of some prognostic value with regard to phthisis. Congestion of the mucous membrane is generally the cause of the hoarseness in the early stages of laryngeal phthisis. At this period there is nothing to distinguish the condition from ordinary chronic laryngitis; when, however, exudation takes place, the appearance is characteristic.

The ary-epiglottic folds look like two large, solid, pale, pyriform tumors, the large ends being against each other in the middle line, and the small ones directed upwards and outwards. The surface is, as remarked, generally pale, but there may be accidental congestion. The inter-arytenoid fold is absorbed in these swellings, which interfere with the action of the arytenoid cartilages, and thus prevent the approximation of the vocal cords. Sometimes the swelling only affects the ary-epiglottic fold of one side, and at first the projection of the cartilages of Wrisberg and Santorini interfere with the distinctly pyriform shape of the tumors, but when developed they are pathognomonic of the disease. The condition described is really only chronic œdema of the ary-epiglottic folds, but when once fully established it is as certain to terminate fatally as a case of acute tuberculosis or encephaloid cancer. Its course is, of course, not so rapid as that of the diseases mentioned, but the end is similar. The epiglottis is not unfrequently thickened; sometimes it is so much enlarged as to prevent an inspection of the parts below. Its shape is often somewhat turban-like, the normal contour and surface marks having completely disappeared. In addition to the thickening, the epiglottis is in fact often rolled backwards on itself, so that the free edges cannot be seen in the laryngeal mirror; in other cases, where they are visible, the cartilage is often exposed from ulceration. Thickening and ulceration of the posterior part of the ventricular bands (false vocal cords) can sometimes be seen, but disease may make considerable progress in this part without coming into the field of vision. Ulceration of the vocal cords is not unfrequent, the most common situation being at the *processus vocalis*, the junction of the cartilaginous and ligamentous portions.

(4) *Miscellaneous Symptoms.*—The laryngeal secretion varies greatly both in quantity and quality, and probably depends more upon the condition of the bronchial tubes and lungs than upon that of the larynx. The constitutional symptoms are those of pulmonary phthisis. The course of the symptoms varies with the site of the disease, the progress being most rapid when the epiglottis is affected, and generally much slower when the ary-epiglottic folds are the parts implicated. The termination is nearly always fatal where thickening has taken place to any considerable extent.

Diagnosis.—Where the characteristic pyriform swellings of the ary-epiglottic folds are present, it is impossible to mistake the disease; but where the thickening is not of such a defined character, the diagnosis is not quite so clear. The only conditions which are likely to give rise to an error are acute œdema, and syphilitic thickening. In acute œdema, the rapid occurrence of the disease and the transparent character of the swelling differentiate it, and in syphilis the thickening is not considerable, whilst the ulcerative process is more active.

Pathology.—It is difficult to investigate the pathology of laryngeal phthisis, because of the close mutual interdependence of the conditions of the larynx and lungs. As the result, however, of careful observation with the laryngoscope in a great number of cases, the ordinary course of events appears to me to be as follows:—1st. There is chronic hyperæmia, generally of a higher degree but more limited extent than is met with in ordinary chronic laryngitis. 2dly. Thickening of the tissues takes place, the kind of thickening varying in different parts; thus, the epiglottis and vocal cords appear to become infiltrated with a semi-solid material, whilst the ary-epiglottic folds become distended by a simple serous exudation;[1] the thickening of the ventricular bands (false vocal cords) is generally of the solid character, but is occasionally serous. 3dly. Small ulcers form; these afterwards coalesce and produce larger ulcers (the secondary tubercular ulcers of the larynx of Rokitansky). The small primary ulcers, which are frequently first seen at the posterior extremity of the ventricular bands and on the under-surface of the epiglottis, when watched with the laryngoscope, often appear to commence in the minute racemose glands. Subse-

[1] In 14 of the 274 cases of œdema of the glottis collected by Sestier (Traité de l'Angine œdémateuse; Paris, 1852), the patients suffered from "chronic laryngitis with pulmonary tubercles."

quently the ulceration spreads to other parts; sometimes, however, the ulcerative process commences in the vocal cords—destruction of epithelium often occurring some time before the dense structure of the cord itself is affected. In other words, when the cords are first attacked, denudation of epithelium precedes deposit in the tissues. The actual loss of substance which takes place in laryngeal phthisis is not generally great, but chronic disease of the cartilages is frequently found when the disease has existed for a few months; and it apperrs to me that Dr. Addison's dictum that "inflammation constitutes the great instrument of destruction in every form of phthisis" is true in this instance. Tubercle appears to play a very secondary part, if any part at all. As regards the relation of laryngeal phthisis to pulmonary phthisis, as already observed, I do not consider that the laryngeal affection is caused by the disease of the lungs. As a rule, the pulmonary disease precedes the affection of the larynx; but still, numerous cases occur in which congestion and thickening of the larynx with hoarseness and cough are found before any disease of the lungs can be detected either by auscultation or microscopic examination of the sputa. On the other hand, in the progress of the disease, evidence of pulmonary disease becomes manifest, and I have only met with three cases in which on post-mortem examination laryngeal phthisis was present without any disease of the lungs. As an almost invariable rule, cavities are found in the lungs, or at least breaking down of lung-tissue.

Morbid Anatomy.—On examining the larynx of a patient who has died from laryngeal phthisis, there is commonly found great thickening of the submucous tissues of the larynx, with ulcers varying in size from a pin's point to a shilling. The small ulcers are most commonly found on the under-surface of the epiglottis; the larger ones at the root of the epiglottis, the posterior extremity of the ventricular bands, and at the *processus vocalis*. Sometimes the ulcerative process is limited to the minute glandulæ, and under these circumstances the mucous membrane presents a worm-eaten appearance. Tubercle is said by Rokitansky[1] to be deposited in the form of gray granulations in the submucous areolar tissue, or to be infiltrated as yellow caseous matter beneath the mucous membrane—the true tubercular deposit being rarely found except over the arytænoideus muscle and the subjacent arytenoid cartilages. Rokitansky does not consider the thickening of the epiglottis to be of the true tubercular character. I have never seen the gray granulations referred to, and the deposit in the tissues has appeared to me to consist of a serous fluid with a few compound granule cells, and with molecular and granular matter. This débris may or may not be tubercular, but even at this period it may be of more than historical interest to remark that though Louis[1] found ulceration of the larynx in one-fourth of his cases of pulmonary phthisis, he did not consider that tubercle was ever deposited in the tissues of the larynx. The proportion of cellular elements varies in different cases and in different parts; in the ary-epiglottic folds they are generally very scarce or altogether absent. Pus is sometimes found diffused through the tissues, but rarely circumscribed, unless it be under the perichondrium of the cricoid cartilage.

Caries of the Cartilages—or, as it is commonly called, *necrosis of the cartilages*—far more often results from laryngeal phthisis than from all other diseases together, and it may be regarded as one of the common sequelæ.

The death of the cartilages is generally believed, and probably with truth, to originate in inflammation of the perichondrium. After death that membrane is not unfrequently found to be separated from the cartilage by a quantity of pus, and ossification of the cartilage generally precedes its death. The cartilage, with the exception of its more or less ossified condition, may present almost a healthy appearance, or it may be of a dark gray or even black color. The presence or absence of discoloration seems to depend on whether there is a communication (through ulceration of the tissues) between the cartilage and the atmosphere. In those cases where there is ulceration, their surface, and sometimes even their entire thickness, is discolored.

Sometimes the cartilages are found to be increased in volume, and still more rarely they are completely atrophied. The latter condition is figured by Rühle.[2] The necrosed condition of the cartilages is generally associated with the presence of serum or pus in the adjacent parts of the larynx; the muscles are soaked in the morbid fluid, and the areolar tissue irregularly distended by it. The etiological relations between the sero-purulent effusions and the necrosis of the cartilages are of a doubtful, and probably of a varying, character; in some cases the former seem to depend on the latter, while in others the opposite relation appears to exist. Sometimes the effusion occurs in the parts ex-

[1] Pathol. Anatomy, Sydenham Soc. Translation, p. 33.

[1] Louis on Phthisis.

[2] Op. cit. Plate I.

ternal to the larynx, especially when the cartilages near the surface externally (such as the anterior parts of the thyroid and cricoid) are affected, and there a laryngeal fistula may be produced. According to my experience, the arytenoid cartilages are the most frequently affected, next to them the cricoid, and then the thyroid cartilage; it is, however, commonly stated that they are affected in the following order of frequency: first the cricoid, secondly the thyroid, and thirdly the arytenoids.

PROGNOSIS.—The prognosis is of the most unfavorable character. Where the epiglottis is much thickened, the progress of the case is generally rapid; on the other hand, when the disease is limited to the ary-epiglottic folds, its course is usually chronic. The result of carefully watching with the laryngoscope, during the last ten years, a great number of cases of laryngeal phthisis, has convinced me that when once thickening to any extent has taken place, that is, when once the disease is fully established, nothing curative can be effected by treatment. Out of several thousand cases, I have only seen two patients recover. Of course, however, suffering may be mitigated, and life prolonged.

THERAPEUTICS.—The plan recommended for chronic laryngitis sometimes gives relief—the application of mineral astringents, by diminishing the irritability of the mucous membrane, often relieving the troublesome cough. Hot and anæsthetic inhalations likewise sometimes comfort the patient; and in cases accompanied by excessive expectoration, I have seen the secretion completely controlled by the inhalation of an atomized solution of tannin (gr. v. ad fl. oz. j.).

It is important to bear in mind that there is a tendency to death in three ways—first, by suffocation, the calibre of the laryngeal canal becoming greatly diminished; secondly, by inanition, the dysphagia being caused by the thickening of the epiglottis and other parts concerned in the act of deglutition; thirdly, by the marasmus, which is a characteristic feature of the constitutional malady; and fourthly, by the combined effect of these influences. The fatal termination may, therefore, be postponed by the performance of tracheotomy, when that operation becomes necessary; by feeding the patient with an œsophageal[1] tube, when normal deglutition cannot be effected; and by the employment of suitable remedies (medicinal and hygienic) against the constitutional debility. It is unnecessary to make any remarks concerning the operation of tracheotomy, as the conditions which render its performance necessary are sufficiently evident. With regard to the use of the œsophageal tube, however, a few observations are called for. The dysphagia, it must be borne in mind, is due more to the act of deglutition being imperfectly performed from non-closure of the larynx by the epiglottis, than by the obstruction in the food tract, caused by the thickened epiglottis. It is from food "going the wrong way," not from the fact of its being prevented passing down the gullet, that the difficulty in swallowing arises. Hence there is generally very little difficulty in introducing the œsophageal tube, especially if it be provided with a duck-billed extremity, and be employed with the aid of the laryngoscope. The fatal termination of phthisis is, of course, much accelerated if the supply of food is to a great extent cut off; and I may observe, that I have prolonged life for many weeks by giving a patient food and stimulants in this way. Alcoholic liquids, which the irritability of the throat would not allow to pass, can be readily introduced into the system by this method. Nutritive enemata can be employed instead of the œsophageal tube, but the results have appeared to me much less satisfactory. Where the patient can swallow a little, but experiences difficulty in doing so from the food occasionally entering the larynx, he should be directed to take nothing but thick liquids. A little arrowroot may be used for giving a proper consistence to the fluids. By thickening the drink (in the way directed) it will be much less likely to pass, beneath the edges of the epiglottis, into the larynx. It is also well to direct the patient to take the drink at a draught—to gulp it down, so to speak—not to sip it. This mode of procedure makes the act of deglutition continuous instead of intermittent, and under these circumstances the passage of food into the larynx is much less likely to occur.

Preventive treatment is the only plan which can be adopted with satisfactory results: congestion of the larynx, therefore, in phthisical persons must be treated with the greatest diligence. The most proper local treatment should be adopted; complete rest of the vocal organ enforced;

[1] This instrument, which has been provided for me by Messrs. Khrone and Sesseman, consists of a gum-elastic catheter, about 12 inches long, which is connected with an ordinary pear-shaped India-rubber bottle (provided with a tap) by a bayonette joint. The tube is first passed just beyond the larynx, then the bottle (previously filled with a nutritive fluid) is attached, and the fluid injected. The feeding can be effected with a common catheter and an ordinary India-rubber injecting bottle, but this plan does not answer so well.

and, above all, suitable atmospheric conditions, if possible, obtained. A warm, dry, and uniform temperature is the grand desideratum.

SYPHILIS.

The laryngeal phenomena of syphilis differ at different epochs of the constitutional disease, and must therefore be considered separately. In *secondary syphilis*, condylomata are the most characterized conditions, but chronic hyperæmia (without the mucous tubercles) and superficial ulceration are often met with. Condylomata, occurring in the larynx, present a similar appearance to those found in the pharynx and elsewhere; that is to say, they are raised patches of the mucous membrane. An elaborate article has been published by Gerhardt and Roth[1] on the subject, and by these observers they are described as being papillary formations, uneven, whitish, smooth or jagged prominences, variously situated in the larynx and of various size and extent. These morbid projections were found most frequently on the vocal cords, on the inter-arytenoid fold, and in those situations which by friction become mechanically irritated. Gerhardt found these condylomata present in 20 per cent. of the patients suffering from secondary syphilis. This proportion, however, has not been found by other observers. In fifty-two cases of well-marked secondary syphilis, which I was kindly permitted to examine at the Lock Hospital in the year 1863, condylomata were only found in two cases, that is to say in less than 4 per cent. Gerhardt's cases, forty-four in number, were in the Venereal Department of a General Hospital, and therefore may well be compared with those at the Lock Hospital. The difference is very remarkable. At the Hospital for Diseases of the Throat we constantly meet with condylomata of the larynx, but the proportionate frequency of laryngeal condylomata in the constitutional complaint of course cannot be ascertained at this institution. The inter-arytenoid commissure and the epiglottis are the parts which I have most frequently observed to be affected. In addition to the condylomata of secondary syphilis, superficial ulcerations of a limited extent are also occasionally met with; there is also sometimes very obstinate congestion of the mucous membrane, but it is impossible to tell whether the latter condition is due to the syphilitic dyscrasia. As regards the treatment, there is little to be said; the condylomata rapidly disappear under local treatment of a stimulating character, and probably often spontaneously. In the cases reported by Gerhardt this condition was removed by a mercurial course; the superficial ulcerations may be cured by the common astringent solutions.

In *tertiary syphilis*, rapid, deep, and extensive ulceration is the characteristic morbid condition of the larynx. The ulcerative process frequently destroys the mucous and submucous tissues to a very considerable extent, and the muscles and perichondrium are sometimes attacked.[1] The ulcerative process is often associated with an œdematous tendency; in the latter case, the laryngeal œdema seems often to occur as an extension of disease from the pharynx. Even when the ulcerative process is arrested, however, the danger does not cease; for the cicatrices often undergo a degree of contraction which greatly interferes with the calibre of the larynx. Numerous cases of this sort have come under my notice, and there are many pathological specimens which illustrate it.[2] The epiglottis is peculiarly prone to be affected by syphilitic ulceration. Whilst ulceration is attacking the epiglottis, great dysphagia is generally experienced; but when the ulcers are healed, swallowing can generally be effected without trouble, even though nearly the whole valve is destroyed. When the walls of the pharynx are also ulcerated, there is danger of the edges of the epiglottis uniting with the pharynx. This condition gives rise to one of the most serious forms of dysphagia.

In these advanced stages syphilitic gummata are sometimes formed, not only in the tongue and pharynx, but in the muscles and submucous tissues of the larynx. These generally soften and ulcerate. The later forms of syphilitic ulcerations should be treated constitutionally with iodide of potassium. Five, ten, or in some cases twenty grains may be given with advantage, in combination with ammonia. By largely diluting the medicine with water, its effect is increased, and it does not irritate the throat in being swallowed. The ulcerated surface should be touched every day with the solid nitrate of silver. For this purpose a piece of aluminium wire, suitably curved, and coated with fused nitrate of silver, should be used.

The ulcerative process, though of the most active character, is almost always very tractable under this treatment; in no stage of the disease does it appear to me to be necessary or desirable to use mercury. The chronic laryngitis some-

[1] Virchow, Archiv, Bd. xxxi. 1861, Hft. 1, § 7.

[1] Specimen No. 38, W Series, St. Thomas's Hospital.

[2] Guy's Hosp. Mus. No. 1655–90, and St. Thomas's Hosp. Mus. No. 22, W Series.

times met with in syphilitic persons (associated as it generally is with chronic bronchitis) resists every kind of treatment.

SECONDARY ŒDEMA.

Œdema may occur as a sequel of Bright's disease, and possibly as the result of cardiac or venous obstruction. Dr. Fauvel has applied the term "*aphonie albuminurique*" to the laryngeal œdema occasionally met with in renal disease, but Dr. George Johnson—an acknowledged authority on diseases of the kidney, and an accomplished laryngoscopist—is of opinion that, "Dr. Fauvel has considerably overestimated the frequency and importance of œdema of the larynx as a result of Bright's disease."[1] Though I have seen a great number of cases of laryngeal œdema, I have never met with it as a complication of renal disease, but that it may occur is shown by the history of a specimen[2] in Guy's Hospital, and by the report of cases under the care of Dr. Rees and Dr. Barlow. Œdema is often the consequence of necrosis of the cartilages, and has been referred to under the disease (Laryngeal Phthisis) in which that morbid process most frequently takes place. It also sometimes occurs, as already shown, in the exanthemata: here it is more probably the result of low inflammatory action than of simple dynamic causes.

The treatment should be the same as that recommended for acute laryngitis.

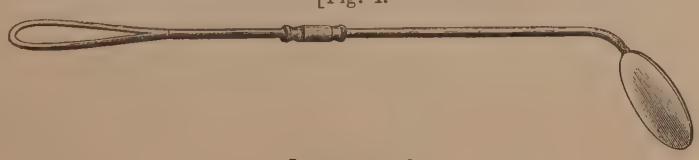
APPENDIX.

ON THE USE OF THE LARYNGOSCOPE.

This instrument, constructed for obtaining a view of the interior of the larynx during life, consists of two parts—(1) a small mirror fixed to a long slender shank, which is introduced to the back of the throat; and (2) an apparatus for throwing a strong light (solar or artificial) on to the small mirror. For this purpose either (*a*) a second (larger) mirror, which reflects the light from a lamp or the solar rays on to the throat-mirror, may be used; or (*b*) the luminous rays from a lamp may be collected and thrown directly on to the smaller mirror, by means of a lens placed in front of the flame. The former method is called "illumination by reflection;" the latter, "direct illumination."

History. — Various independent attempts to examine the larynx have been made at different times by different practitioners. Levret, a distinguished French physician, as far as bibliographical research at present goes, seems to have been the first to invent a laryngeal mirror. This occurred in the year 1743. In the beginning of the present century, Bozzini contrived a Laryngoscope, which to a certain extent complied with the conditions contained in the above definition; but being clumsily constructed, it could not be used effectively. In the year 1825, an unsuccessful attempt to inspect the glottis

[Fig. 4.



Laryngoscope.]

was made by M. Cagniard de Latour; and a few years later, in 1829, Dr. Benjamin Guy Babington exhibited, at the Hunterian Society of London, a Laryngoscope which, excepting that a hand-mirror was used instead of a concave circular reflector attached to the operator's head, closely resembled the modern instrument. In later times,[3] Senn, Bennati, Baumês, Liston, Warden, and Avery made attempts or suggestions towards obtaining a view of the larynx during life; but it was left for M. Garcia to lay the foundation of a method of examination, which, through the genius and perseverance of Professor Czermak, at once reached a high degree of perfection. The employment of the Laryngoscope in practical medicine dates from a paper published by Czermak in 1858.[1]

The Laryngeal Mirror.—The throat-mirror should be of glass backed with a coating of silver (not amalgam, as this is much more readily damaged by heat), mounted in German silver, and fixed at an angle of about 120° to a slender shank or rod about four inches in length of the same material. The shank of the mirror is fixed into a hollow wooden or ivory

[1] The Laryngoscope, 1864.

[2] No. 179, 650. Lancet, Sept. 5, 1863, vol. ii. p. 277, and vol. i. Feb. 27, 1864.

[3] For further historical details see the author's treatise "On the Use of the Laryngoscope," chap. i. 3d edition (Longmans and Co.)

[1] Wien Medizin. Wochenschrift.

handle, about three inches in length and a quarter of an inch in thickness. A laryngeal mirror, the reflecting surface of which is about four-fifths of an inch in diameter, will be found convenient in most cases; where the distance between the uvula and posterior wall of the pharynx, however, is great, the largest size mirror, about one inch in diameter, answers best; in the case of children, a mirror about half an inch in diameter should be used. Circular mirrors cause the least inconvenience, but where the tonsils are very large, oval or ovoid mirrors can be most easily employed.

Illumination by Reflection.—For throwing a strong light on to the laryngeal mirror, and thus into the larynx, it will be found most convenient to employ a circular and slightly concave mirror about three inches and a half in diameter, and having a focal distance of about twelve or fourteen inches. When the solar rays are reflected into the throat, the surface of the mirror should be plane. The mirror should be attached in some way to the operator's head, and may be worn either opposite one of the eyes (Czermak), in front of the nose and mouth (Bruns), or on the forehead (Johnson, Fournié). I follow Czermak's plan. The reflector may be attached to the operator's head either by a spectacle-frame (Semeleder)—and in this case the upper half of the rim of the eye-piece of the spectacle-frame may be conveniently removed—or by a frontal band (Kramer). The mirror should be connected with its support by a ball-and-socket joint. In making an examination after the manner of Czermak, the reflector should be perforated by an oblong hole, the long diameter of which should correspond with the long diameter of the eye.

Any lamp that gives a bright steady light answers the purpose perfectly well. A moderator, paraffin, or argand gas-burner will each be found convenient. My "Rack-movement[1] Lamp" is perhaps the most convenient illuminating apparatus that exists. It is now employed at most of the London hospitals, and is very suitable for the private consulting-room. For strengthening the light a lens may be employed, and various lamps and lanterns have been contrived for the purpose. The "light-concentrator," which forms a part of my rack-movement lamp, will be found useful in Laryngoscopy, for, whilst excluding the lateral rays, it collects all those which can possibly be conveyed to the reflector.

Direct Illumination.—The best mode of using direct light is that employed by most of the French laryngoscopists. The lamp, provided with a lens on the side facing the patient, is placed on a table about a foot wide and three feet long. The observer sits on one side of the table, and facing him on the other side is the patient. The lamp, provided with a strong lens on the side of the patient, and screened towards the practitioner, is placed on the table between them. In operating, the practitioner has one arm round each side of the lamp. The method employed by Stoerk and Walker, in which direct light (strengthened by a glass globe of water acting as a lens) is thrown on to the laryngeal mirror, is less perfect on account of the lateral deflexion which the rays undergo after impinging on the laryngeal mirror. For demonstrating to a class, the oxy-hydrogen light,[1] as employed at the Hospital for Diseases of the Throat, is the most perfect arrangement.

Method of Examination.—The patient should sit upright, facing the observer, with his head inclined very slightly backwards. The observer's eyes should be about one foot distant from the patient's mouth, and a lamp burning with a strong clear light should be placed on a table at the side of the patient, the flame of the lamp being on a level with the patient's eyes. The observer should now put on the spectacle-frame with the reflector attached, and, directing the patient to open his mouth, should endeavor to throw a disk of light on to the fauces, so that the centre of the disk corresponds with the base of the uvula. When the observer has gained dexterity in throwing the light, the patient should be directed to open his mouth widely, and to put out his tongue; and the operator should hold the protruded organ between the finger and thumb of his left hand, previously enveloped in a soft cloth or towel. In thus keeping the tongue out, the greatest gentleness should be used, as the employment of force, by exciting reflex action, only defeats the object in view. Holding the laryngeal mirror, previously warmed over the lamp (to prevent the condensation of the breath on the surface), like a pen in the right hand, the operator should now introduce it to the back of the throat, its face being directed downwards and kept as far as possible from the tongue. The posterior surface of the mirror ought to rest slightly on the base of the uvula, which should be gently pushed rather upwards and backwards towards the posterior nares. The plane of the mirror should form an angle of about 45° with the horizon.

Where the tongue forms an arched prominence at the back of the mouth, the patient should be directed to inspire deeply, or to produce some vocal sound; these acts cause an elevation of the uvula,

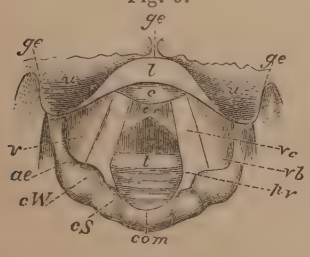
[1] Made by Mayer and Meltzer, 59 Great Portland Street.

[1] For a description of the apparatus, see Medical Times and Gazette, July 24, 1859.

and thus facilitate the introduction of the mirror. It is better to introduce the mirror several times, and keep it *in situ* only a few seconds, than to allow it to remain in the mouth too long, and thereby produce an irritation which prevents further examination at the same sitting. When the epiglottis is large and pendent, the mirror should be introduced lower in the fauces, and more perpendicularly than is usually suitable.

The Laryngeal Image.—In some cases, on introducing the laryngeal mirror, only the epiglottis may be visible, with perhaps just the tips of the capitula Santorini at the posterior part; whilst in others the ary-epiglottic folds, the ventricular bands, the vocal cords, the small cartilages above the glottis, the cricoid cartilage, the rings of the trachea (and, perhaps, even the bifurcation of that tube), can be seen with perfect distinctness. The appearance of parts is shown in the annexed drawings:—

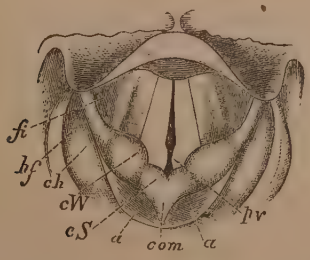
Fig. 5.



Laryngoscopic Drawing, showing the Vocal Cords drawn widely apart, and the position of the various parts above and below the Glottis during quiet inspiration.

ge, glosso-epiglottic folds; *u*, upper surface of epiglottis; *l*, lip of epiglottis; *c*, cushion of epiglottis; *v*, ventricle of larynx; *ae*, ary-epiglottic fold; *cW*, cartilage of Wrisberg; *cS*, capitulum Santorini; *com*, arytenoid commissure; *vc*, vocal cord; *vb*, ventricular band; *pv*, processus vocalis; *cc*, cricoid cartilage; *t*, rings of trachea.

Fig. 6.



Laryngoscopic drawing, showing the approximation of the Vocal Cords, and the position of the various parts, in the act of vocalization.

fi, fossa innominata; *hf*, hyoid fossa; *ch*, cornu of hyoid bone; *cW*, cartilage of Wrisberg; *cS*, capitulum Santorini; *a*, arytenoid cartilages; *com*, arytenoid commissure; *pv*, processus vocalis.

But to properly understand their relation, this book should be held at the same inclination as that which the laryngeal mirror occupies when *in situ* (that is to say, at an angle of 45° with the horizon, the foot of the page being farthest from the observer). The only inversion which takes place in the formation of the image is in the anterior posterior direction; the part which in reality is nearest to the observer, the anterior insertion of the vocal cords, becoming farthest in the image, and the posterior commissure, which in reality is farthest from the observer, becoming nearest in the image. With regard to the lateral and vertical relations of parts, no inversion takes place. That which is on the right side of the larynx (the right vocal cord, for instance), appears on the right side of the mirror, and that which is on the left side of the larynx on the left side of the mirror; in the same way the part which is highest in the larynx (the epiglottis) is highest in the mirror, and the parts lower down (the arytenoid cartilages) are at the lower part of the mirror. It is only when the image is transferred to paper, and becomes a drawing, that its symmetrical character can give rise to mistaken notions concerning inversion.

It is necessary to make a few remarks on the normal color of the different parts. *The epiglottis* is of a dirty pinkish hue on the upper surface; its lip (or free edge, and the immediately adjacent under surface) is of a decidedly yellow color; whilst the cushion (and rest of the under surface, when visible) is invariably bright red. The *ary-epiglottic folds* are about the same color as the mucous membrane of the gums, the cartilages situated in them being of a somewhat deeper tint. *The ventricular bands* are of a bright color, being of about the same shade as the mucous membrane lining the lips. *The vocal cords* should be pearly white, like the conjunctiva of a child. The cricoid cartilages and tracheal rings are of a yellow color, and the mucous membrane between them bright red.

The Introduction of Instruments within the Larynx.—In applying local remedies to, or operating on, the larynx, by the aid of reflected light, as the right hand is required for the instrument used, the laryngeal mirror should be introduced with the left hand. In this case, the patient must hold his own tongue out.

Infra-glottic Laryngoscopy.—In some cases, after tracheotomy has been performed, and where a tube is worn, valuable evidence may be derived by introducing a minute mirror, with its face directed obliquely upwards, through the fenestrated canula. On account of the size of the mirror, it is necessarily made of steel; and as both its size and material cause it to cool very rapidly, a coating of glycerine will be found convenient for neutralizing the effects of the condensation of the water contained in the expired air.

CROUP.

By William Squire, L.R.C.P. Lond.

Definition.—An inflammation of the laryux and trachea in children, commencing in the air-passages and often extending into the bronchi. It induces thickening of the mucous membrane and an altered secretion which may become either membraniform or purulent. There is frequent, sharp, harsh, ringing cough; difficult breathing, with loud, shrill inspiratory sound; altered voice, at first hoarse, afterwards whispering, or extinct; fever, loss of appetite, thirst, and little or no difficulty of swallowing.

Synonyms. — Cynanche Trachealis, Cullen; Suffocatio Stridula, Home; Angina Inflammatoria Infantum, Russell; Acute Asthma of Children, Millar; Cynanche Stridula, Crawford; Angina Trachealis, Johnstone; Angina Polyposa seu Membranacea, Michaelis; Hives, Benjamin Rush; Cynanche Laryngea, Dick; Angina Membranacea, Goelis; Tracheitis Infantum, Albers; Laryngo-tracheitis, Blaud; Laryngite striduleuse, Guersant; Spasmodic Laryngitis, Charles Wilson; [Spasmodic and Pseudo-membranous Laryngitis, G. B. Wood.—H.]

Name.—Croup and Roup (hreopan, Anglo-Saxon, *Clamare*) were the names popularly applied to the disease when it was first investigated by Home; at the commencement of the present century they were equally in use in the neighborhood of Edinburgh. The latter once had a wider range, having been used by Knox as a verb signifying to cry hoarsely, and Burns has "roupet" in the sense of hoarse as from a cold;[1] since then, however, it has disappeared from our literature, and Croup, which before Home's inquiry was as strange to England as to the rest of Europe, has been the world-wide designation of the characteristic group of symptoms attending impediment to the entrance of air into the windpipe. It is somewhat remarkable that even in those countries where the disease is not infrequent, it is rarely distinguished by a proper name; Bräune in Germany, and Hives in America, being the only examples of which I am aware. Strypsiucka in Sweden has more the signification of quinsy or suffocation, and was not popularly applied to this disease when Rosen wrote; our own "strangles," "closing," "chock," or "stuffing," have neither been generally used nor definitely applied.

History.—How large a share Croup has had in the various anginas or cynanches enumerated in the earlier stages of the history of medicine, it is impossible to define; in the subdivision of those terms by Boerhaave, there is evident intention of including it, and there is evidence that it was so included by our English physicians, from Sydenham to Mead. As Home remarks: "Probably it has existed, more or less, in all ages, for the same productive causes must have operated formerly as they do at present."

There are other systematic names under which cases of this disease have also been included, such as the "suffocative catarrh" of Ettmüller, and the "tussis convulsiva puerorum" of Willis; though the first of these names is now restricted to capillary bronchitis, and the second to hooping-cough, yet there is a clear reference to Croup in the pages of Ettmüller. The first evidence of Croup noticed by Baillou was in an epidemic of hooping-cough in Paris, 1576,[1] and the first mention of it by name in this country begins with the distinction between it and hooping-cough drawn by Dr. Patrick Blair,[2] in a letter to Dr. Mead, dated Cowpar of Angus, July 6th, 1713, wherein he says: "The tussis convulsiva, or chink-cough, is also some years epidemical and becomes universal among children; as is a certain distemper with us called the Croops, with this variety, that whereas the chink-cough increases gradually, is of long continuance, seizes in paroxysmes,

[1] John Jamieson, D.D., an Etymological Dictionary of the Scottish Language. Edinburgh. 4to. 1808.

The Mœso-gothic hrop-jan is here given as the root of many words, signifying outcry, as croak, rout, hoop; also of the Teutonic roep-en and the Icelandic hroop. In the northern counties of England roopy and ropy are still used for hoarse, and the latter word is sometimes heard in the southern counties in the same sense.

[1] Baillou, Epid. Ephem. Lib. ii. pp. 197 and 201.

[2] Observations in the Practice of Phys etc. London, 1718.

and the patient is well in the interval; this convulsion of the larinx, as it begins so it continues, so violently that unless the child be relieved in a few hours 'tis carried off within twenty-four, or at most forty-eight hours. When they are seized they have a terrible snorting at the nose and squeaking in the throat, without the least minute of free breathing, and that of a sudden; when perhaps the child was but a little time before healthful and well. The most immediate cure is instant bleeding at the jugular, either by the lancet or leeches; when the most urgent symptoms are gone, then emetics or the like are administered at discretion."

The distinction is not always attended to, as even Huxham,[1] writing "de pertussi puerorum," speaks of an acrid humor sometimes attacking the larynx.

Dr. Russell[2] gives us an account of the disease observed by him in connection with the epidemic of malignant angina then prevalent, from which, however, he is careful to distinguish it. He says:—"I have observed it is most apt to seize children from two years old to eight or ten, but chiefly the younger sort." He details the leading symptoms, and remarks that the whole "fistula pulmonalis" becomes inflamed.

Home's essay[3] is founded on the observation of cases where no epidemic complications prevailed. In a careful and most philosophical inquiry into the causes of the symptoms before him, he determined their dependence on the pathological changes in the larynx and trachea, and regarded the disease as an acute inflammation.

Millar,[4] who practised at the same time in the south of Scotland, and had similar cases of Croup under his observation, remarks upon, but gives undue prominence to, the spasmodic element in the paroxysm of the disease. Further attention is called to Millar's views by the publication in England of a letter from Dr. Rush,[5] of Philadelphia, and a discussion commenced, which has continued to our own day, in which the true nature of Millar's cases is not always remembered.

The latter half of the last century and the beginning of the present are remarkable for the numerous outbreaks of epidemic angina recorded in different countries and places. In the midst of an epidemic at Cremona, Ghizi had described the case of a child dying of laryngeal complication, and attempted to set up a distinction between it and the pharyngeal form of the epidemic. The cases recorded by Starr, in Cornwall, occur in his description of an epidemic of this kind, and it is probable that Russell's were not wholly isolated. Home, careful lest the distinct inflammatory disease which he had constituted should be confounded with an epidemic disorder of so different a nature, drew a distinction at the very commencement of his inquiry between his own observations and those recorded by Russell; yet fresh outbreaks of the epidemic, its liability to spread to the air-passages, and its severity towards children, tended to their confusion.

One effort to avert the fatal mistake that ensued—and it is the only one—is recorded in the treatise of Dr. Johnstone the younger, of Kidderminster.[1] He quotes from Home the "two very different situations of the suffocatio stridula; the former more inflammatory and less dangerous; the latter less inflammatory and highly dangerous: in the former the pulse is generally strong, the face red, drought great, and they agree with evacuations; in the latter the pulse is very quick and soft, great weakness, tongue moist, less drought, great anxiety, and evacuations hasten death." Dr. Johnstone contends that these are not merely two stages of the same disease, but that the latter applies to that complication of the epidemic which has been observed in all its records from the earliest times, and that it was occasioned by the same epidemic cause, and required the same sustaining plan of treatment that his father adopted.

Unfortunately, though argued with learning and experience, these views did not prevail; the name of Croup was applied to the epidemic complication, and the treatment laid down by Home for the one disease was very energetically employed against the other. The divergence of opinion tended to stimulate the collection of facts bearing on the subject. The accounts of epidemic Croup, though generally referring to the disease now known as diphtheria, doubtless comprehend some cases of simple Croup; some of our own accounts of Croup probably include cases of diphtheria. It was to illustrate the tracheal complication of the epidemic that both the great *concours* on Croup were instituted at Paris; yet the prize essays of

[1] Huxham, Obs. de Aëre et Morbis Epidem. Lond. 8vo. 1793. P. 77.

[2] Russell, Dr. Richard. Œconomia Naturæ in Morbis acutis et Chron. Glandularum. 8vo. Lond. 1755. P. 72.

[3] Home, Francis, M.D., &c. An Enquiry into the Nature, Cause, and Cure of Croup. 8vo. Edin. 1765.

[4] Millar, Observations on the Asthma and Hooping-Cough. 8vo. London, 1796.

[5] Rush, on the Spasmodic Asthma of Children. 8vo. London, 1770.

[1] Johnstone, J., M.D. A Treatise on the Malignant Angina, to which are added some Remarks on the Angina Trachealis. 8vo. Worcester, 1779.

MM. Vieusseux and Jurine, of Geneva, and of Albers, of Bremen, are among the most valuable contributions to our knowledge of Croup as an independent disease; and, though the tendency in France has since been to restrict the term Croup to one of the accidents of diphtheria, yet the opposite view has in that country been maintained with great ability by MM. Bricheteau, Desruelles, Emangard, and especially in the valuable original work of M. Blaud, of Beaucaire.[1] A similar controversy, arising under conditions more allied to those of our own country, has been continued in Northern Germany since the time of Wichmann of Hanover, starting from a line of distinction being drawn between its spasmodic or inflammatory nature; the treatise of Goelis of Vienna[2] is, however, sufficiently comprehensive. In America the "Observations on Cynanche Trachealis," published in the first volume of "Medical Inquiries and Observations," by Dr. Benjamin Rush, will ever stand as one of the clearest and most practical accounts of the disease. In our own country we have the careful study of Cheyne,[3] enriched, as it is, by the admirable pathological drawings from the hand of Sir C. Bell: it forms a worthy sequel to the work of Home, conceived and executed in the same spirit, from observations made in the same locality at no great distance of time. We have also the matured experience of the same author, gained in Dublin and its neighborhood, published thirty years later in the "Encyclopædia of Practical Medicine." Dr. Charles Wilson of Edinburgh has published in the *Edinburgh Journal of Medicine* for 1855–56, a philosophical review of the whole subject.

[The opinion that membranous laryngitis or tracheitis, "true croup," is a distinct disease from diphtheria, has been supported in America by Drs. G. B. Wood, A. Flint, J. Lewis Smith, Fordyce Barker, and others. Dr. J. F. Meigs contends against it. Besides those abovenamed, abroad, C. West, Virchow, Niemeyer, Oppolzer, and Letzerich may be cited as favoring the doctrine of the nonidentity of the two disorders.

Croup is a sthenic localized inflammation, whose causation is connected always with some exposure to cold, wet, &c.; it is never epidemic. Diphtheria is a general disease, usually epidemic and asthenic in type; the local inflammation in it is secondary to the constitutional affection. In Croup the false membrane is a solidifying exudation *upon the surface* of the mucous membrane; in diphtheria it involves its *substance* also. Croup is not attended by albuminuria, nor followed by paralysis; both occur not unfrequently with diphtheria. Extension of the pseudo-membranous deposit into the bronchial tubes is rare in diphtheria, not uncommon in Croup; while the commencement of the deposit in the region of the tonsils and pharynx in diphtheria, and in the trachea or larynx in Croup, is a matter of familiar observation.

A table is given in Meigs' and Pepper's treatise on the Disease of Children, which shows that, after diphtheria had, about 1860, become recognized as, at that time, a new disease in Philadelphia, the mortality from it added for several successive years more than 300 to the deaths in each year in that city, while the deaths from Croup continued to number, annually, as before, from 200 to over 400.—H.]

Etiology. — The collection of facts which go to make up our history of Croup is sufficiently extensive; but, besides the uncertainty as to their true bearing which we see in some of them, others are drawn from too limited an area, or considered in too restricted a relation to come before us in their true value. I have therefore availed myself of the kind permission of Dr. Farr, to consult the careful reports prepared under his direction in the office of the Registrar-General for England. These reports extend over a period of twenty-five years; they contain particulars of nearly 95,000 deaths from Croup; and therein the locality of occurrence, the season, the sex, and age at the time of death, are readily investigated. I have also referred to reports for Scotland, extending over seven years and including 6982 deaths registered as Croup.

Croup is specially a disease of childhood, occurring most frequently from the first to the seventh year, and rarely happening, after the tenth. In a hundred deaths from Croup, we may estimate 13 as occurring in the first year of life, 25 in the second, 22 in the third, 16 in the fourth, 11 in the fifth, and 12·3 in the succeeding five years, while the deaths beyond ten years of age may be represented by 0·7, the remaining fraction. The proportion of deaths from Croup to one hundred deaths from all causes, registered at each age, is 0·65 in the first year, 3·25 in the second, 6·5 in the third, 8·0 in the fourth, 7·0 in the fifth, and 3·5 for the five following years together. The proportion for the first and second year of life would be raised to above 1 and 4 respectively, if the deaths at these ages registered under the head of Laryngitis were included; some considerations, hereafter to be given, tend to raise the proportion for the first

[1] Nouvelles Recherches sur la Laryngo-Trachéite. Paris. 8vo. 1823.

[2] De Rite Cognoscenda et Sananda Angina Membranacea. 8vo. Vienna.

[3] Essays on the Diseases of Children. Essay II. Cynanche Trachealis. Edin. 4to. 1801.

and second years and to diminish the already small proportion registered as occurring beyond ten years of age. In Scotland the proportion registered for the first year is 1·5; something over 1 per cent. for the first six months, and for the second six months exceeding 2 per cent.; though the actual number dying from this cause in the first half-year of life is but little below, and in some years has exceeded, the number of deaths in the second. The annual average number of deaths from Croup in England is near upon four thousand; and, though this number is somewhat below the returns for the majority of the last ten years, and in excess of the greater number of any of the preceding ten years, yet the proportion to deaths from other causes has always been very nearly one in the hundred, somewhat above this for the last ten years, a little below it for the preceding ten, again above this proportion for all the preceding years that are registered; the first of these years, 1838, being as high as 1·30. The lowest proportion was 0·869 in the year 1853; the high proportion of 1·335 occurs for the first time in the year 1850; the highest proportion reached is 1·40 in the exceptional year 1858. In Scotland the proportion to other deaths is from 1·5 to 1·8 per cent.; the annual number of deaths is close upon one thousand. Dr. Burke, of Dublin, kindly provided me with the results of the first complete registration for Ireland, which shows that in the year 1864 the whole number of deaths from Croup was 1926, and the proportion to deaths from other causes 2·05.

More boys than girls die of Croup; this fact is obvious over whatever period or district our inquiries extend: the difference is striking, and by frequent notice has been brought more prominently forward than the corresponding fact in the history of some other diseases chiefly fatal in childhood. More boys than girls are born, in a proportion somewhat greater than one in every fifty children, or, to give the result of a very extended examination,[1] there are 511·75 males and 488·25 females in every 1000 births; it appears that of this number 83·71 males and 65·74 females die within the first year, after which the death ratio of the two sexes for the next ten years is nearly equal: still there are a larger number of males than of females living at this period, and the deaths of females from all causes are to those of males as 87 to 100 in the first five years, or as 88 to 100 in the first ten years: now the deaths from Croup are so nearly in this proportion, and of late years have so often shown a difference so much less than this, that a doubt might be entertained as to whether any difference in the liability of the sexes really existed. A comparison between the deaths from all causes of each sex for each year, with the deaths from Croup at each year, sex with sex, shows a difference of excess on the side of the males so constant, that it is rare to meet with an exception, but at the same time so slight that it can only be considered a characteristic of the disease in the aggregate, corresponding with the results of pneumonia and tubercular meningitis, rather than with the more characteristic zymotic diseases, and contrasting with those of diphtheria and hooping-cough, where the excess of deaths is greatly on the side of the females. Some of the results of the preceding inquiry are brought together in the following table:—

Deaths from Croup at each year of age.	1st year.	2d year.	3d year.	4th year.	5th year.	5th to 10th year.	All ages beyond.
To 100 deaths from Croup . .	13·	25·	22·	16·	11·	12·3	0·7
To 100 deaths from all causes	0·65	3·25	6·5	8·3	7·5	3·5	
Males	0·7	3·5	6·7	8·5	7·7	3·7	
Females	0·6	3·0	6·3	8·1	7·3	3·4	

The influence of climate upon Croup is generally admitted. Cases considered trivial in some parts of France are frequently fatal in Northern Germany, and what in our variable climate excites alarm is regarded with reasonable hopefulness on the continents both of Europe and America. A combination of cold and moisture with rapid alternations of temperature, together with some endemic or epidemic influence, has to be admitted. In South America, Buenos Ayres, with its large river, affords frequent instances of Croup; and in some of the large towns of Australia, with their defective sanitary arrangements and large infantile mortality, Croup was not unknown years before the first appearance of diphtheria. The high mortality in Scotland is not greatest in its most northern extremity;

[1] English Life Table, with an Introduction, by W. Farr, M.D., F.R.S. London, 1864. Table III. p. 24.

the mortality from Croup in the northern counties of England is generally over 1 per cent.; but this is equalled and often exceeded in the warm southwestern promontory of Cornwall, with Devonshire and Somerset. The western shores of England, receiving the Atlantic moisture, show a higher mortality from this disease than the eastern. South Wales has the high rate of 1·5 per cent. This is not so much owing to its mountainous interior, as to its large mining population and the defective sanitary state of its large towns. The highest rate for England is in the populous districts of Lancashire and Cheshire; and here there can be no doubt that a dense town population, the children specially living under defective sanitary conditions, causes Croup to be particularly fatal. That it is not merely the combination of cold and moisture, may be shown by the returns from the southwestern corner of Scotland, Wigton and Dumfries, the latter conterminous with our Northumberland and Cumberland, and with a rainfall[1] exceeding that of any part of England. Though the temperature is sometimes very low, it is more equable than that of many parts of our island, and the mortality from Croup, generally below 1 per cent., is sometimes as low as 0·5. The classical Croup districts of Scotland still retain their preeminence; they are not confined to the west coast of Scotland: the eastern coast is deeply indented by the sea, and not only do deep valleys of clay extend from these firths, but their shelving shores leave a great expanse of ooze uncovered at every tide; and during the easterly winds, which here prevail for three months of the year with great bitterness, the characteristic cases of Scotch Croup occur. This part of Scotland forms an isthmus, only thirty miles in width, in the vicinity of which, and of the peninsula formed by the eastern firths, Croup is most fatal, the mortality often exceeding 2 per cent. As high a rate is found for Ireland, where imperfect drainage, unreclaimed bog, and a large expanse of inland water add to the influence of the Atlantic in causing a remarkable humidity of climate.

The influence of season is illustrated by the quarterly reports for London; an average of the ten years from 1844 to 1853 gives the number of deaths from Croup in each quarter as follows: first quarter, 95; second quarter, 81; third quarter, 68; fourth quarter, 92·5; the greatest fatality being in the winter and spring; the greatest variation is found in the second quarter; the third quarter has the lowest number, and shows the least variation; the fourth quarter generally shows a considerable increase on the third, or warm quarter of the year: this was less marked than usual in the year 1852; for while the deaths in the third quarter showed a tendency to increase, being as high as 74, there were only 76 in the fourth quarter, the unprecedented mildness of the season doubtless being the cause of this arrest; the two usually cold months of this quarter, November and December, averaging throughout a temperature of 6° higher than had ever been known during the past eighty years. In the next year severe cold set in before the end of January; in February, the temperature was below the average on most days, there was snow every day; the second week in March was warm, the end of the month cold with snow; the summer was variable, cold, and wet; double the usual quantity of rain fell in July; there was fine weather in August only. Again, from October 21st to November 8th, the temperature rose to 5·3° above the average, at other times it had been below, and November and December were remarkable not only for low temperature, but also for a density of fog and depth of snow hardly ever exceeded in London. The mortality from Croup in each of the four quarters in this year was: first quarter, 93; second, 79; third, 72; fourth, 130; and 145 in the first quarter of the next year, 1854, the weather continuing to be cold. The weekly returns for this period show a correspondence between mortality from Croup and temperature. In the winter quarter of 1852, the weekly numbers for November are, 3, 5, 8, 5; in the corresponding weeks of 1853, they are, 7, 13, 8, 12; the high number occurring on fall of temperature in the second week of that month. In the previous March there was a rise of temperature in the second week: the weekly numbers for this month were, 15, 8, 2, 10; and in the next year, 1854, there are 21 deaths from Croup returned in one week in February. Cold, however, may determine the incidence, but not the prevalence of Croup. In most diseases of the respiratory organs, the greatest fatality is seen in the coldest seasons. The proportional mortality in this class mounts from 11 per cent. in 1852 to 13.5 in 1853, and hooping-cough from 1·8 to 2·3 per cent., while Croup decreased from 0·99 to 0·86 per cent.; the whole number of deaths from Croup being less in 1853 than it had been for several years.

A further illustration of the influence of season and the relation between Croup, the diseases of the respiratory organs, and prevailing epidemics, is afforded by the years 1859, 1860, 1861. The quarterly re-

[1] At Wanlock Head, in Dumfrieshire, there was an estimated rainfall of 80 inches in the year 1861.

turns of the mortality from Croup in London for these three years are as follows:—

	1st Quarter.	2d Quarter.	3d Quarter.	4th Quarter.
1859	132	103	80	81
1860	117	80	105	169
1861	236	190	170	252

In the first of these years the summer and autumn were fine and hot. June had a daily excess of 3° of temperature; in July the mean temperature was 68°, and on the 13th and 18th of that month the thermometer reached 93°; part of October and November was cold, but it was warm at Christmas. The next year, 1860, presents a remarkable contrast to this; a cold period commenced in June: of the three following months, Mr. Glaisher in his report[1] says, "The weather, during the past quarter has been very remarkable for continued low temperature, frequent rain, large amount of cloud, little sunshine, and bad weather generally;" the winter that followed was one of the coldest on record, the thermometer being as low as 6° Fahr. in London on Dec. 6th–7th, and at Nottingham it fell to 8° below zero, or 40° below the freezing-point of water; a rapid thaw set in on Dec. 30th, and though there was severe cold in January, the remainder of the winter was more remarkable for rapid changes of temperature than for continued cold. In 1861 the spring was variable, but the summer and autumn unusually hot and dry: in June the thermometer was 82°, and on August 12th it was 89·5; the years 1770, 1811, and 1831 only had as warm an October; with the exception of a cold week in November, the warm weather continued up till Christmas. In the cold season of 1860, diseases of the respiratory organs advanced from 13·7 to 16·4. Hooping-cough, however, did not increase, and laryngitis observed a considerable decrease. Croup also decreased from 1·29 to 1·05. During the high temperature of 1861 bronchitis decreased, hooping-cough was on the increase, laryngitis and Croup continued the same.

The London quarterly reports give similar evidence. Diseases of the respiratory organs increased from 15· to 20·: bronchitis, from 8· to 10·; pneumonia, from 5· to 6·7; laryngitis and Croup were 0·4 and 0·7 respectively in the warm season of 1861; when the former diseases were declining to their usual standard, the latter made their most rapid increase in London.

Laryngitis is also approximated to Croup in the time of year in which it is most fatal.

The proportional mortality in London from diseases of the respiratory organs is, for each quarter—

Diseases of the Respiratory Organs	25·	15·	10·	20·
Bronchitis	15·	5·	9·	10·
Pneumonia	9·	6·	5·	8·
Laryngitis	0·6	0·5	0·3	0·4
Croup	0·9	1·0	0·6	0·8

There is a strong contrast between Croup and bronchitis as to the time of year at which each is most fatal; the difference is less marked between it and pneumonia, probably from a larger proportion of its victims being among the young; there is a close correspondence between laryngitis and Croup in this respect, and the table shows also the increased proportion in which they have latterly appeared in London.[1] Eighty-five per cent. of the mortality from laryngitis is among children; and in the great increase in the deaths from this cause during these three years, viz. from 260 in 1859 to 386 in 1861, there are 42 of the smaller number, and about 50 of the increased number of these deaths that occurred beyond the tenth year.

One of the influences bearing upon the mortality of Croup is that of associated epidemics. In the year 1853, when deaths from Croup were few, the mortality from diseases of the zymotic class was 20 per cent., or nearly at its lowest. Smallpox was on the decline; measles reduced to 1·1 per cent., having been 2·3 in 1851; and scarlatina, though less than it had been, was still 3·7 per cent. From this time an increase is observable in the number of deaths from Croup. In 1854 the mortality from measles was again over 2 per cent.; scarlatina was over 4 per cent. in this year and the next; and though in 1856 it fell to 3·6, and measles to 1·8, and epidemic diseases generally did not exceed one-fifth of the mortality, yet a new epidemic disease, allied to scarlatina and closely associated with Croup, was developing in England: many of its first victims were registered under this head; and Croup, which was 1·05 per cent. in 1855, rose to 1·34 and 1·27 per cent. in the two following years. The year 1857 was unusually hot;[2] 1858 was an epidemic year;

[1] Remarks on the Weather during the Quarter ending Sept. 30, 1860, by James Glaisher, Esq., F.R.S., in Registrar-General's Report.

[1] The deaths from these causes in London for each quarter average for 1850–53: Croup, 92, 78, 62, 94; Laryngitis, 68, 61, 34, 43. For 1859–61: Croup, 161, 124, 115, 134; Laryngitis, 110, 74, 47, 68. Before 1845 it was not usual to separate infantile laryngitis from Croup. The average quarterly returns of death from laryngitis for the five years 1840–44 were 7, 9, 6, 10; for the subsequent five years, 47, 38, 25, 44; the returns under the head of Croup showing at the same time a diminution.

[2] "The temperature was 2° above the average of the preceding 17 years; the wind, in-

diseases of this class constituted more than one-fourth of the general mortality; diphtheria had not been separated in the registers, and the returns under the head of Croup and scarlatina were increased, the one by a thousand, the other by more than ten thousand, and their proportional numbers to 1·4 and 6·8 respectively. In the next year Croup is 1·3 and scarlatina 4·5 per cent., diphtheria appearing as 2·2 per cent. In the next two years, 1860 and 1861, the mortality from epidemic diseases is reduced to less than one-fifth of the whole, scarlatina and measles are about 2 per cent., and diphtheria 1·25. Croup is again 1 per cent. In 1861 the number of deaths from diphtheria is at its lowest, that from Croup a little higher than in 1860. The increase was chiefly at the end of the year, and was almost confined to London and Lancashire; the deaths in this part of England were more by 2000 in the last quarter of the year than in either of the two previous winter quarters; in Manchester they rose from 1682 to 2123, and other large towns in this district show a similar increase. In London the chief coincidence is that of the increase in the mortality from scarlatina from 467 in the third quarter to 1145 in the last. In the corresponding quarter of 1860 it was 602. Hooping-cough and fever were on the increase in these districts, and from this time the commencement of an epidemic period may be dated.

The year 1862 was cold, wet, and unhealthy; the mortality from Croup is again 1·3 per cent.; from hooping-cough, 2·8; from other diseases of the respiratory organs, 15·6; scarlatina is increased to 3·4 and diphtheria is 1·13; zymotic diseases generally have increased to 21·2; the whole mortality during the year being very great.

It is to some general causes acting unfavorably upon the health of children, rather than to the influence of a particular epidemic, that these variations in numbers are to be attributed. Croup, indeed, seems to hold a place intermediate between diseases of the zymotic class and those of the respiratory organs.

Diseases of the zymotic class generally show a greater mortality among females than males. Croup differs from them as a class in this respect. Hooping-cough exemplifies this point of difference in the greatest degree; Measles, though often followed by Croup, shows no periodical coincidence with it, except in the time of year at which it is most prevalent, cold appearing to increase the fatality of both; Scarlatina is distinguished by being least fatal in the spring; while Smallpox is not modified in its violence by either season or climate. Whenever these diseases have increased there has been an increase, at least, in the fatality of Croup, and the same is noticeable with respect to Diphtheria. This differing from the preceding diseases in its liability to recurrence, and to some extent corresponding with Croup, is distinguished, as to these general characteristics, by showing a greater mortality among females; by being when least epidemic, like scarlet fever, most fatal in the autumn and winter; and when most epidemic, like smallpox, by a progression independent of season and climate.

Croup differs from diseases of the respiratory organs in its periods of greatest mortality, and widely from some of this class as to prevalence at different periods of life; the whole class agrees with Croup in showing a greater mortality of males than of females, and the effects of climate and season are always obvious in both. One disease of this class, infantile laryngitis, has been specially commented upon, to set forth its contrast with the class, and its affinities to Croup, and to show by the intimate correspondence of the two in every particular by which they can be compared, that this form of disease should not be considered, from the absence perhaps of one anatomical character, as in any way different from Croup, nor hereafter be classed apart.

Croup has also some relations to diseases of the constitutional class. In the severer forms of local inflammations, individual susceptibility is concerned; and besides a predisposition, induced either by previous illness or by the causes examined, some constitutional infirmity may predispose, and it is noticeable that this source of disease is more apt to come out in the first decade of life among males, and just after that period in females. Croup affects children of certain families, and certain children of some families, more than others; it is said that those of a florid complexion are often more liable. We see in a family some suffer much under infantile disorders that affect others but slightly, without knowing why. Not only is the tendency to spasm hereditary, but to local congestion, at particular ages. Where there is no diathetic peculiarity, some aberration from health is a usual predisposing cause. There are other causes, such as sudden changes of dress, the impression of cold air after heating exercise, even residence at the sea-side, which may influence the frequency, though not the fatality, of Croup. Children who have suffered an attack are specially liable to a recurrence on exposure to any of these causes, and the recurrent attack is not always the least severe.

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stead of moving at the average rate of 110 miles a day, passed over London at the rate of 81 miles during the 53 weeks. The rainfall was 21·4 inches."—*Registrar-General's Twentieth Annual Report*, p. xliii.

Symptoms.—The symptoms of Croup follow quickly upon the cause which excites them; the first indication is often mere hoarseness in the tone of voice or cry, the child is feverish, and either dull or fretful; is thirsty, and drinks without difficulty; the tongue has a white fur, and is red at the tip and edges; there is some heat and dryness of the skin, and a check to the secretions generally; an occasional short dry cough may be noticed, and a little harshness of breathing. The more characteristic symptoms generally come on at night; during the first sleep the cough is noticed to be sharp and harsh, with that peculiar croupy clang which, when once heard, is always easily recognized; this may be repeated at some intervals without rousing the child from sleep; the heat and dryness of the skin are now more marked, the pulse is frequent and strong, and the breathing loud and difficult, when some repeated clanging cough, with shrill-drawn breath, wakes the child in a fright struggling for breath. He starts up, is flushed and hot, the eyes staring, the conjunctivæ red, a hissing sound accompanies every inspiration, and is very marked and loud after the short dry sounding cough; it is evident that insufficient air enters the chest, although the respiratory efforts are great; the circulation is now also highly excited, the turgescence of the face and neck increases, and the color deepens, the child puts its hand to its throat as if to remove obstruction, speech becomes impossible, and soon, as if in despair, muscular effort relaxes and air begins to enter the chest more freely.

The paroxysm may come on within a few hours of exposure, and sometimes, though rarely, before the usual symptoms of ingress have been noticed; it may begin at any hour, but most frequently at night, and is seldom delayed beyond thirty-six hours from the commencement of the illness; it may last but a few minutes, or be prolonged with varying intensity for more than an hour; its first accession is nearly always followed by a remission, more or less complete, sometimes so perfect that the most careful examination is required to ascertain the presence of the disease, and to prevent a fallacious confidence following too closely upon the first alarm. Its intensity also varies much; but however slight in degree, it occasions an acceleration of pulse, an increased heat and redness of the surface, especially of the face, also an injection of the conjunctival vessels not existing before, and which will probably subside if the paroxysm be not too soon repeated.

The temperature as taken in the axilla has been found at 100°, with the first premonitory symptoms; by the second day, or at night, it will rise to 102°, possibly to 103°, coincident perhaps with the earlier paroxysms, but not with the severer attacks which may follow: indeed, the temperature is generally less on the third day, and will subside by the fifth unless pulmonary complication have arisen. A high temperature at the very onset may point to one of the exanthemata; its persistence to diphtheria.

It is important to consider whether the patient is being seen shortly after such an attack; for if seen before, or at some time after a first slight attack, there will be neither redness of conjunctiva nor coryza, and even though sneezing or some catarrhal symptoms have preceded, there will be no defluxion from the nose; the skin will be dry and harsh rather than hot; the urine will be found to be in small quantity and of a high color, with no marked sediment. The pulse is quick and hard; the respiration is accelerated, but, though disturbed and somewhat oppressed or wheezing during sleep, it is not much altered in frequency; and, unless there be already some implication of the lung, or the presence of some other disease, it will not have a ratio of more than one to three, nor of less than one to four pulsations.

The most valuable indication of the presence of the disease, even in this early period, is drawn from the respiratory sounds and movements. The voice may attract attention, the cough will soon give the trumpet-note of alarm, but, without the impeded respiration and its physical signs, their indication is not conclusive; they may even be absent during some temporary lull in the symptoms, and then is the favorable moment for a careful auscultation. The inspiratory sound is prolonged, and, instead of the ordinary blowing murmur, there is a sibilant tubular sound, high in pitch and of a metallic quality, constituting a prolonged harsh stridor; the expiratory sound is also prolonged, but is low in pitch and harsh, the respiratory murmur is weak, especially in the anterior and upper part of the chest, and is masked by the tracheal siffle; this is very marked over the larger bronchi, but is not always enough to conceal the presence of a certain amount of mucous and sibilant rhonchus in some of the smaller bronchi posteriorly; there is no dulness on percussion, not even over spots where the murmur is altogether absent. The respiratory movement may also be noticed to be deficient in this stage, and when exaggerated during dyspnœa, to be inefficient; the supra-clavicular spaces are depressed during inspiration and though the diaphragm may descend well, the intercostal spaces will not bulge, nor will the walls of the chest be fully expanded. The cough is sure not to be long quiet,

and its short, dry, abrupt character attracts notice; it is not, strictly speaking, a hoarse cough, there is no deficiency in body of sound, and it is high in pitch; a shrill inspiration accompanies each effort; during the paroxysm the cough will be frequent, and it is then the sign most worthy of attention. Our further investigation of the state of the respiratory organs must at this time be limited to inspection of the front of the chest, and percussion at the back; when the attack is over, during sleep, or after vomiting, auscultation can be satisfactorily accomplished.

The condition of the whole extent of the body should now be examined, to remark the absence of spasmodic contraction of the thumbs or toes, the presence or absence of a rash on the skin, of œdema of the extremities or of the face and neck, and the degree of warmth and tone of color of these parts as compared with the body generally; the last point is to be specially noted, so that any variation in the depth or tone of color in these parts and in the face and lips may be readily appreciated. The sides of the neck are to be examined for enlarged glands; those at the outer border of the sterno-mastoid are always palpable, but it is important to note that the glands at the angle of the jaw are not enlarged. An early opportunity must be taken for a full and clear inspection of the inside of the mouth and throat. There may be some redness of the soft palate, sometimes œdema of the uvula; the pharynx will be either of a pallid red or of a brighter pink hue; there will be a remarkable absence of free secretion, and no speck of adherent exudation visible in any part of the pharynx or tonsils: some enlargement of the tonsils has been noticed, and if it be sufficient to press forward the anterior arch of the palate a slight irregularity of outline will be occasioned, but the membrane is continuous, of uniform color, and smooth. Before this inspection is ended, the tongue should be sufficiently depressed to bring the epiglottis into view; the vivid redness and turgescence of its apex contrasts strongly with the surrounding textures, and indicates the condition of the subjacent orifice.

The disease attains its height by the end of the third day at the latest, but the intensity of the attack may hasten the stages of its advance, and death may occur within forty-eight hours of its commencement. The characteristics of this second period are high vascular excitement, and an ever-increasing difficulty of respiration; the cough is now almost incessant, or frequently recurring in shocks of convulsive violence; there is no free secretion; a little viscid phlegm, clear or muco-purulent, may sometimes be expelled, or an opaque mucus be seen in the lower part of the pharynx. Pain is complained of in the front of the larynx, or nearer the top of the sternum; the voice may become whispering or suppressed from the effort to speak being evidently painful; the stridulous inspirations are louder and more continuous, the labored and sonorous breathing being audible at a distance; and now, though the thirst is great, deglutition is not always easy, in some cases, from imperfect closure of the glottis, the liquid provoking great dyspnœa; in others the urgency of the dyspnœa itself not permitting the effort. Even at this stage, if the attacks of dyspnœa have neither been too severe nor too frequent, and air sufficient to maintain life be yet admitted to the lungs, the pulse will steadily maintain its force and frequeney; there will be great heat of surface and profuse perspiration, especially on the face and forehead, which parts will be of a bright red color; the veins of the neck and temples may become distended and dark, and the face and lips at times purple, but if they quickly assume a brighter tint it may not be too late for the disease to take a favorable turn. The first evidence of this is a change in the character of the cough; it becomes lower in tone and less dry, not less in force nor much less in frequency, but becoming moist, and gradually effecting the expulsion of some thick semi-opaque mucus in which not unfrequently small whitish opaque flakes are discernible. At the same time the sibilant inspiration is neither so loud nor so persistent; it is still heard before each cough, and it will be audible during sleep or on first waking; the voice at times regains its natural quality, at others is only hoarse or dissonant in its higher tones; the accessions of dyspnœa are rare and less marked, the febrile excitement subsides, a more equable perspiration is maintained, the urine becomes abundant and frequently affords large deposits of urate of ammonia and sometimes of oxalate of lime; the soft palate, tonsils, and pharynx become paler and less tumid; a loose muco-purulent secretion is often seen in the gullet; the tongue is less red, less furred, and more moist: thirst diminishes, and appetite returns; the harsh tracheal siffle will have been replaced by some mucous râles, the normal respiratory murmur will be everywhere restored during this favorable progress, and, if uninterrupted, three days may suffice to establish convalescence. A persistence of irritability in the air-passages may greatly delay this, and a further extension of disease to the lung endanger it altogether. Auscultation here again becomes the only basis of confidence, as upon the subsidence of the more urgent signs, others, less obvious but not less important, may

be discovered; it may be found that there are parts of the lung to which air is not admitted; that there is an accumulation of mucus in the bronchi, or an amount of bronchitis, with the development of subcrepitant rhonchus, that will seriously impede recovery. An extensive capillary bronchitis, or the existence of pneumonia, will not only be indicated by their special characters, and by the acceleration of the pulse-respiration ratio, but also by the general symptoms; the signs of laryngeal obstruction have diminished, but the respiration is as much embarrassed; there is less effort, but there is no relief; and the disease advances to its close as surely and even more hopelessly than if its advance had been unbroken.

The third stage is that of apnœa and rapidly advancing exhaustion: it may come on in the manner just described, but more frequently is the direct sequence of the more urgent symptoms of the second stage, which, when about to lead to this result, present some additional noteworthy particulars. The tracheal siffle is accompanied by "tremblotement," a laryngo-tracheal mucous râle with a tremulous character heard in both expiration and inspiration, or a click either constant or occasional, may also be heard through the stethoscope;[1] and whatever the character of the râle or siffle, it now becomes audible over the trachea in expiration. The voice is whispering or completely suppressed, the cough stifled, powerless, or altogether absent; it may, however, recur in some paroxysm of dyspnœa and afford temporary relief by the chance expulsion of some membranous shreds. The hand may at times be directed to the mouth or throat as if to remove some obstruction, but the paroxysms become more urgent and without remission, there is restless tossing of the body and limbs on the bed, consciousness is impaired, and voluntary power much diminished; the respiratory efforts may continue for a time to be violent, loud stridor marks both the expiration and inspiration; with the latter act the larynx is seen to be forcibly drawn towards the sternum, the supraclavicular and intercostal spaces sink, and though the abdomen descends, the epigastrium recedes; the head is thrown back, the lower jaw fixed, the mouth partly open, the alæ nasi dilated and depressed; the veins of the neck and temples are distended and dark, the eyes starting, and the face livid; the pulse becomes either too rapid or too weak to be counted, the temperature falls and the perspiration becomes cold and clammy, the neck and even the extremities may be swollen as well as the face, and assume a leaden color, or the whole surface becomes of a marble-like pallor, the features are set, the eyes lose their expression, oscillate in the orbits, become distorted and fixed. Sometimes the whole body is bent backwards, and death has been known to occur at the moment of an inspiratory effort. Suffocation is every moment imminent, but frequently an apparent calm in the more violent symptoms precedes death, a gradually decreasing quantity of air is entering the lungs, the whole chest is flattened and much diminished in fulness and capacity; the countenance, though not livid, no longer retains its florid hue—it becomes shrunken, dusky, or pallid; complete stupor sets in, the limbs become flaccid, the surface cold; the pulse is small, weak, and frequent; the eyes are dull and sunken; the respiration becomes gasping and irregular, the pulse intermitting, and both soon cease. The whole duration of the disease, advancing uninterruptedly to its fatal termination, rarely exceeds five days.

The division into the three stages of ingress, full development, and termination by apnœa, is an arbitrary one; the limits of each cannot be defined, nor is there any natural line of separation; whenever apnœa is commencing, the third stage has arrived, and this may be suddenly fatal, even though the first stage seems unaccomplished; so also in the second stage the signs of increasing obstruction in the trachea may be undeveloped, and yet the third stage have set in and be gradually advancing.

[No allusion is made in the above account to a class of cases familiar to American practitioners, designated by Condie and others as *Spasmodic* Croup, or sudden *Night-Croup*. This is quite a different affection from laryngismus stridulus, with which it has been confounded by some writers. It often occurs without any premonition, in children, in the middle of the night; the child being awakened from sleep by difficulty of breathing, attended by a short, barking cough. This, although alarming, is, under proper treatment, never, or almost never, fatal. All the symptoms, and the manner of their relief (by *relaxation* and *secretion*), point to a combination, in the pathology of this affection, of pre-inflammatory congestion of the laryngeal and tracheal mucous membrane with spasmodic narrowing of the glottis.

Intermediate between this and membranous or "true" Croup, is *Catarrhal* Croup, or *Croupal Catarrh* (alluded to on a subsequent page under DIAGNOSIS). In the latter, the symptoms are those of an ordinary acute bronchial attack, except that the cough has a barking sound, much

[1 A mucous râle sufficiently tremulous to be audible without the stethoscope, is usually a very favorable sign.—H.]

most marked at night; and often, in the night, croupal difficulty of breathing comes on. Under suitable relaxing treatment, these croupal symptoms will seldom recur for more than three nights; but, if neglected, or undue exposure occurs, the disorder may pass into the form of true membranous Croup, with all its dangers.

The common proclivity, in children at least, to anginose symptoms *at night*, is hard to explain. It is, nevertheless, a fact, that the commencement of a hoarse barking cough, with difficulty of breathing in the daytime, and, equally, its continuance through the day after beginning in the night, are much more serious in prognostication than when such symptoms are entirely nocturnal. A day-time barking cough, *with fever*, should always receive prompt and careful attention.—H.]

Diagnosis.—Numerous causes affecting the glottis and larynx, modifying their special functions, and interfering with the entrance of air into the lungs, give rise to croupal symptoms that are to be distinguished from Croup. Spasm of the glottis is readily excited in infancy; irritation of the gums, or of the stomach, or an undue excitability of the nervous system, will suffice for its production without either local congestion or general febrile action. It is often first noticed as the child starts out of sleep with a stridulous or crowing inspiration checked or interrupted by the spasm; the head is thrown back and fixed, the chest motionless and the face livid; in some convulsive action a little more air may enter the chest, when in a few seconds the spasm yields sufficiently for expiration to be effected, perhaps to be succeeded by another crowing inspiration or by a more free entrance of air, and the attack terminates in a fit of crying; the breathing then regains its natural characters, no signs of stridulous inspiration remaining. An attack of this severity is seldom the first to which the patient has been subject, but it is likely to be repeated on the slightest cause of excitement or alarm; the crowing inspiration will not always be heard; there may be only momentary holding of the breath or acts of involuntary deglutition; it sometimes is induced by the act of swallowing or of suckling; or it interrupts a fit of crying, the loud and long expiratory sounds of that act being replaced by a short faint sound, and inspiration becomes long and noisy, instead of being short and free; an expression of alarm is fixed on the face, which becomes red and turgid, and the spasm may either pass rapidly into natural crying, or there may be more serious cause for alarm. The breath may be held for a half a minute, the spine arched backwards and rigid, the thumbs bent inwards on to the palm of the hand, the great-toes separated from the others, and both fingers and toes strongly flexed. Where the convulsive proclivity is marked, and some persistent irritation not in the windpipe keeps up cough which might mislead, this bending in of the thumbs is a valuable aid in the diagnosis. In the slighter cases the attack being accompanied by crying rather than cough, and the tone of the cry being natural, would almost suffice to distinguish these cases of child-crowing, or laryngismus stridulus, from Croup. Dr. Clarke,[1] who describes this condition accurately, remarks that "it has sometimes been called chronic Croup, but it is very different from Croup, and is altogether of a convulsive character."

Some cases of this kind have been described by Franks and Kopp, in Germany, as thymic asthma. The enlarged thymus is only one of the signs of a persistence of the infantile condition, among others a quickness of the pulse and respiration, and even the infantile ratio between these acts of 3 to 1 may be noticed: nor are these attacks limited to the earlier months of infancy, but may be continued into the fifth year; their abrupt commencement and termination, the freedom of respiration in their intervals, and a concomitant derangement of the child's health not of a febrile character, in which slighter symptoms of the same kind occur, the temperature often being considerably below the healthy standard, separate all cases of this kind from Croup.

Foreign bodies entering the glottis[2] produce croupal symptoms which are distinguished from Croup by the absolutely sudden manner in which a child, probably in perfect health at the time, is seized; it may be during a meal, or with a known object in the mouth which has disappeared while about to speak or at play, and not during sleep or at night. There is one kind of foreign body, however, which may find its way into the glottis during sleep, and that is an *ascaris lumbricoides* from the stomach; several instances of this are on record. It might seem possible that matters vomited during sleep should be drawn into the windpipe; this has happened to adults, but rarely if ever to children. The dyspnœa from any of these causes, if not suddenly fatal, is as violent at the first moment as at any subsequent time. There may be remissions from the foreign body descending into the bronchus; in this case the sound of the

[1] Commentaries on some of the most important Diseases of Children, by John Clarke, M.D. Part I. p. 88. London. 8vo. 1815.

[2] A case is recorded in the 1st volume of the Med. Times and Gaz. for 1853, p. 126, of a fruit seed, three-fifths of an inch long and one-fifth in circumference, passing the glottis of a child two years and one month old.

voice or of the cough would be clear, and there would be no tracheal siffle, while the signs of obstructed respiration, more frequently found on the right, must always point out the site of the foreign body: when it is in the larynx or trachea it gives rise to constant dyspnœa, with loud expiratory bruit as well as the stridulous inspiratory sound. The ingress of acute Croup has been attributed to a possible accident of this kind, the alarm occasioned by the sudden onset of dyspnœa having made the mother or nurse oblivious of the premonitory cough; taking the stethoscopic signs and general course of the symptoms together, the diagnosis need not long remain doubtful. When boiling water or chemically irritating fluids have been swallowed, there is no room for doubt, and the state of the mouth, pharynx, and nares, confirms the history; the whole course of the resulting lesion differs only from Croup in its sudden commencement and in its cause.

Injury to the larynx from without is a possibility that suggests a careful examination of the integuments of the neck for any appearance of injury, or of subcutaneous emphysema: the outline of the larynx should be traced; its mobility noted, as also whether there is marked tenseness, swelling, or tenderness, in its immediate neighborhood; these last characters, with a limited extent of redness of the skin and loss of elasticity at the spot, might indicate deep-seated abscess.

There are other chronic affections which in their exacerbations may simulate Croup. Polypus, or growth from the mucous membrane in the interior of the larynx, may occasion croupal dyspnœa, which besides its chronic history is accompanied with stridor, both in inspiration and in expiration. Tumor causing mechanical obstruction of the trachea is rare in children. Spasm of the glottis may be occasioned by tumors, enlarged glands, or abscess, implicating the recurrent nerve; post-pharyngeal abscess may not only cause obstruction, but serious injury to the windpipe; it is generally a sequel to other diseases, and is therefore more likely to be met with in secondary Croup, of which it may not only be a complication but a cause; a digital examination of the posterior wall and sides of the pharynx would give the necessary information of its progress where inspection is unsatisfactory.

Even in acute disorders, and especially in the more urgent and sudden cases of dyspnœa, the pharyngeal surface of the glottis should be examined by touch, whereby an unsuspected cause of obstruction may be at once detected and removed. The interior of the mouth and throat should be inspected in all cases; without this the nature of the diseased action cannot be evident, and by it alone it can often be determined that some disease other than Croup is the cause of the symptoms. The physical examination of the chest must never be neglected; where Croup is present it is the chief means of tracing its progress, and, as pointed out by Dr. Williams, the first sign of the disease, even at the onset, may be detected by the stethoscope, and by the same aid some of the diseases liable to be mistaken for Croup are most easily recognized.

Catarrh in young children, with a proclivity to spasm, may occasion a hard ringing cough, attended by sibilant inspiration in the early part of two or three successive nights; the respiratory surfaces are now the source of the irritation exciting this spasmodic cough. In stronger or older children the same local affection sometimes induces a passing hoarseness or aphonia, and a short harsh cough which is husky rather than dry, and is neither frequent nor spasmodic, unless it be slightly so during the first sleep, and then even sibilant inspiration may be audible; but this again disappears, the breathing is free, there is no acceleration of the pulse, and any heat of skin soon yields to perspiration: the illness has commenced with well-marked catarrhal symptoms; the cough is at times heard with a moist sound, or is attended with secretion, and auscultation furnishes the signs of incipient bronchial catarrh; these are well developed on the second day, and become more extended; they are uninterrupted by the siffle of laryngeal obstruction, which no longer even obscures the normal breath-sounds.

Hooping-cough often has the frequency, more rarely something of the tone, of the cough of Croup on its first commencement, but as it progresses the cough is attended by a shrill inspiration which differs in character from that accompanying the cough of Croup only by the sonorous quality constituting the hoop; the cough is also worse during the first half of the night, and occasions suffocative fits of dyspnœa; these are produced differently from those of Croup, and are more directly the effects of the cough, which consists of short expiratory efforts rapidly succeeding each other, no inspiratory action intervening. Thus a turgescence and lividity of countenance is caused, which rapidly disappears after free inspiration is accomplished; it is at once obvious that sufficient air is received into the chest during the inspiration, as the sonorous quality would seem to indicate, while the perfect relief to the child, the gentle play of the chest walls, and their rounded form offer a sufficient contrast to the dyspnœa of Croup. Moreover, at the period of the illness when the hoop is developed, the cough has already existed some days with precedent

catarrhal symptoms; the more persistent dyspnœa of severe hooping-cough comes on still later, but whether in its earlier symptoms or later effects, it is in the more distant air-tubes that the signs of disease are detected, and not at their commencement. The hoop is, moreover, only an occasional phenomenon, while in Croup the stridor continually increases and becomes unintermitting.

In measles a ringing cough, and dyspnœa resembling that of Croup sufficiently to mislead, sometimes accompany the catarrhal symptoms of invasion; the cough may have exactly the tone of Croup, but the stridulous inspiration is less marked, and both will sooner become catarrhal. The febrile condition is similar, so that unless the presence of measles is expected, or its signs just apparent, there may be no guide as to the nature of the affection but the character of the throat-redness; this is in patches of dusky red instead of being uniformly bright. The croupy condition induced by the ingress of measles subsides when the rash is well out; it is however liable to recurrence, from various and often slight causes, for some time after the original disease has entirely disappeared.

Diphtheria implicating the air-passages produces the effects of Croup, with very similar symptoms; these, though they do not supply the main elements of the diagnosis, afford many points of difference; they are the sequel of a more general or a more prolonged diseased action, and may not appear till after three or four days, or even a week, of illness: their mode of development is most varied; sometimes they have not attracted attention until the dyspnœa is unintermitting; sometimes at their very beginning they will embarrass the respiration and excite the circulation to a degree that completely alters the features which had up to that time marked the disease; at others, in the intervals of dyspnœa, though the respiration is not free, the child will take food, resume its play, and either seem cheerful and excitable, or indifferent to its danger. Though the croupal symptoms may show an exacerbation during the first half of the night, they are as frequently first noticed at other times; they are neither so paroxysmal in their commencement, nor so dependent on the urgency of the cough. An excess of suffocative difficulty may not occur so early as in Croup, but when it has occurred there will not be so complete a remission as after the first paroxysm of Croup; the impeded respiration, with signs of laryngeal and tracheal obstruction in both expiration and inspiration, may be detected before any severe distress has been thereby occasioned. Early notice is often given by a hoarse cough, which has a muffled rather than a ringing sound, and by an unpleasant tone of the voice, which is husky rather than hoarse, or it is nasal in tone, or croaking and deep, but much sooner becomes whispering or permanently extinct.

Where the more general characters of diphtheria are well marked, its epidemic prevalence known, a contagious influence traced, or some days of illness have preceded the attack, there will be little difficulty in rightly estimating the nature and cause of these additional symptoms, and none if the few days of previous illness have been under observation, so that the special products of diphtheria and the asthenic tendency of that disease have been recognized. Where the phenomena of dyspnœa are those first presented to our notice, it will not be possible to arrive at a diagnosis from them, unaided by inquiry as to the history of attack; whatever the character of the dyspnœa, and however recent or even sudden the attack, if severe illness have recently been recovered from, or if there is the history of only a short illness that seemed to be passing off, it may not be diphtheria, but it is probably idiopathic Croup; while if there have been two or more days of illness immediately preceding the first croupy symptoms, if on one day there have been dulness or debility and refusal of food, if one night's extreme restlessness unattended by cough, with excitability or irritability, headache, vomiting, an unusually free action of the bowels, or even a very free secretion of urine, coryza, and congested or glistening conjunctiva, defluxion of glairy fluid from the nostrils, or stoppage of one or both of them, creamy moist tongue and difficult or painful deglutition, the presence of diphtheria may be inferred; there will then be greater enlargement of the lymphatic glands of the neck, and especially of those at the angle of the jaw; these may be so full as to render a free inspection of the mouth difficult, yet the enlarged tonsils, and the unequal congestion of the soft palate, will give further confirmation, even if no patch of diphtheritic deposit come into view.

The fullest and clearest inspection possible of the mouth should be obtained; one spot of diphtheritic deposit brought into view clears up all doubt in this most important matter; it may be below or behind the enlarged tonsil, or almost hidden in the angle of the soft palate and uvula, or completely concealed by the uvula or velum; it may only just be commencing in the follicles of the tonsils, or may have already cleared from their surfaces, leaving only the mark of its attachment, or a faint indication within the substance of the mucous membrane. When extensive deposit in the fauces in-

tades the larynx by continuity, a glance is sufficient to confirm what the general aspect of the patient would suggest, and from the greater enlargement of the lymphatic glands a very limited inspection is often all that can be obtained; there would in such cases probably be considerable fetor of the breath, and secretions escaping from the mouth without effort to expel or restrain them. But the implication of the larynx is as frequently effected by new centres of deposit as by continuity of advance, and there are not wanting cases of diphtheria to show that the diseased action may commence in the air-passages; in these cases the careful record of the mode of ingress—the attack being recent, this is more easily obtained with accuracy—and the consideration of the concurrent symptoms must be mainly depended upon, bearing in mind that it is in these cases where the excitement of the circulation and general heat of surface are most likely to mislead; the pulse, however bounding, quick, or full, is not strong or hard, and the breathing, though disturbed, is not accelerated proportionately with the pulse; indeed, during the development of diphtheria the respiration generally has a ratio of less than one to four pulsations; any precedent vomiting, diarrhœa, or diuresis, with loss of sleep, loss of appetite, or difficulty of swallowing, powerfully aid the diagnosis; while an inspection of the fauces is of the greatest importance, as some of the appearances are the same whether the first product of the disease be deposited there or not.

In diphtheria some part of the pharynx is sure to become the seat of the disease, and to show unequal redness and turgescence at some points before the deposit occurs; or if the first patch have separated from the tonsil, these signs would indicate fresh deposit elsewhere; and while it is rare for the larynx to be the part first attacked, it is very frequently invaded by extension from these new centres of deposit. The aryteno-epiglottidean folds are often the points from which such extension proceeds, and cannot at this time be brought into view; or some point near the posterior nares may be implicated, and be therefore quite out of sight; in these cases a glairy or yellowish mucus in streaks along the back of the pharynx, or collected in some quantity in the gullet, will give indications of the disease: probably other isolated patches may appear either in the nares, on the lips, or even the eyelids; the lymphatic glands are always enlarged, and the deglutition is or has been difficult. In making the discrimination between diphtheria and Croup it is necessary to detect the phenomena which are the earliest to appear, and those which are most easily ascertained, but not to neglect others which may confirm the diagnosis. The occurrence of epistaxis, or of blood in the expectoration, or of well-marked pieces of false membrane in the matters cleared from the throat with specks of blood on one surface, would point to diphtheria. The presence of albumen in the urine is conclusive in the diagnosis, and may sometimes be detected in the pale urine passed at the commencement of the attack, though frequently not appearing till a later period. Paralysis of some of the muscles of vocalization, deglutition or of motion, is equally distinctive of diphtheria. Finally, a prolonged convalescence and a less tendency to recurrence not only confirm the diagnosis, but complete the distinctions between diphtheria and Croup.

Smallpox is sometimes attended by a consecutive affection of the larynx and trachea simulating Croup; about the seventh day of the eruption a little hoarseness, some hard cough, dry at first, but soon with expulsion of tenacious mucus, characterize a secondary Croup, the result of the specific lesion of the larynx and trachea, which is rapidly fatal.

Measles gives rise to secondary Croup, only distinguishable from idiopathic Croup by its more asthenic character, and by its having been preceded by the specific disease. It does not come on till the rash is disappearing and the cough which attended the eruption is subsiding; there is frequently an aphthous condition of the mouth at the same time. We may here infer a specific action on the mucous membrane similar to that exciting pneumonia under the same conditions. Some cases of secondary Croup are subsequent to the pneumonia of measles; these[1] and many of those occurring earlier are really owing to diphtheria attacking a patient debilitated by the primary disease: diphtheria may also complicate measles.

Scarlet fever may possibly excite croupal symptoms by extension of the specific inflammation of the throat to the larynx; more frequently some general turgescence of tonsils, submaxillary lymphatic glands and the parts about the throat excite croupy paroxysms. Croup secondary to scarlet fever is later in its appearance. The majority of these cases of secondary Croup are complications with diphtheria, and in the few which survive recurrence is rare.

Erysipelas is sometimes attended by croupal complication; here the dysphagia is extreme, and the epiglottis is swollen, red, and erect.

Œdema of the glottis may follow continued fever or other prolonged illness; the consequent dyspnœa comes on in the

[1] Archives Générales de Médecine, tome vi. p. 466.

night; and may be rapidly fatal; but if a remission occur there is neither the same degree of sibilant respiration nor the cough of Croup, there is but little dysphagia, and the epiglottis is neither red nor swollen.

Inflammation of the tongue or mouth, either from the effects of mercury or other causes, may extend to the larynx. In all these cases there is the existence of the previous disease, and the affection is of the asthenic type; some of the latter are rare in children of the age at which we expect Croup.

Hysteria may come on in a subject young enough to render it important to exclude the possibility of its being Croup, so closely may it simulate the leading features of the disease. Dr. Cheyne relates a case so deceptive, that he was induced to order bleeding for its relief. This close correspondence is more liable to occur in one who has been the subject of Croup in childhood; but then, though the attacks may have been numerous up to eight or nine years of age, there has been an immunity for the past three or four years. Cough with occasionally a croupy sound is first heard, and the attack generally comes on towards night: the cough is loud and peculiar, and the respiration becomes soon noisy both with inspiration and expiration, the head is thrown back, the hands clutch at the throat, the face becomes red and swollen, the eyelids are closed and puffy, and the pupils dilated. The dyspnœa may be of some continuance, but is not really so extreme as it may seem; the auxiliary muscles of respiration are not called into service; there is no drawing in of the supraclavicular spaces, nor sinking of the epigastrium; the respiratory murmur may be weak, but is everywhere audible; there is no tracheal siffle heard by the stethoscope placed at the top of the sternum; the voice may be harsh and produced with difficulty, but is not much altered: a conclusion will be arrived at by conjoining these particulars with an observation of the general attitude, the arching backwards of the body, the tossing of the limbs, the age and sex of the patient, the absence of definite complaint, the impaired volition of hysteria, and the spasmodic muscular actions, the most obvious of which will probably be the grating of the teeth. In these cases, whatever other disordered functions may be associated with the attack, it is necessary to be satisfied that it is not owing to local irritation, and that there is no appearance of disease in the fauces.

Pathology.—Croup is essentially an inflammation of the mucous membrane at the commencement of the air-passages, and this, not of the superficial or catarrhal kind, but such as affects profoundly its texture, nutrition, and secretion. The determining cause of this, as of other more deeply-seated inflammations, it is difficult to estimate. What in one subject may excite but a passing irritation, in another sets up a definite course of diseased action, its phenomena modified by the conditions of the part in which it is developed. The vital importance of the parts which are here the seat of the disease, the special endowment of the glottis and larynx, and some peculiarity in their structure and development, determine the phenomena of Croup.

At the time of puberty the larynx rapidly increases to one-half more, or to double its previous calibre, according to sex; an increase in the capacity of the trachea has been going on for some time, but during childhood the air tube and its orifice are remarkably small; at this period the mucous tissue is capable of as much distension from turgescence of its vessels, and even of more swelling from submucous infiltration than accompany the inflammatory process in adults: hence a certain condition of the lining membrane might become a dangerous obstruction to the entrance of air in the one case, and not offer an alarming impediment in the other. The existence of submucous cellular tissue as far as to the vocal cords, conduces to this possibility. The upper part of the larynx is also finely organized for the exclusion of any foreign or irritating particles from the air-passages; its muscles are spasmodically excited directly these irritants from without come into contact with the mucous membrane covering them. In this disease, the irritant cause is from within; no sooner is the congestion of the membrane sufficient to interfere with the normal state of its surface, than a source of irritation is provided which cannot fail to induce the violent action of the muscles beneath: at first, probably, it is not so much the swelling as the dryness of the membrane, from check to its natural secretion, which is concerned in the causation of the spasm; the hoarse voice indicates turgescence of the mucous membrane as far as to the vocal cords; the tone of the cough and the intermitting laryngeal siffle show that swelling is not great, and that spasm is an integral part of the seizure. The further effects of the inflammatory action are very evident, whether its intensity be confined to its first site, or be extended further along the air-passages; in the first case inflammatory products are formed both beneath and upon the mucous membrane, in the second they are accumulated on the surface, and in either the results are commensurate with the symptoms, and

afford a sufficient explanation of the resulting apnœa.

A very small amount of exudation, added to the swelling and spasm of the larynx, may not only be fatal, but produce symptoms of greater urgency than would ensue from a considerable accumulation in the trachea, and yet few traces remain after death to show that the passage of air was precluded. In the trachea all inflammatory exudation must be found upon its surface, and may there remain with less obstruction to respiration than is possible in the larynx, though not without offering a further impediment to the entrance of air into the chest; the power of the cough is thus lessened, and it is further diminished by the imperfect closure of the swollen glottis, while the secretions of the surface over which the inflammation extends are altered, so as to be less easy of detachment until the inflammatory action yields, by which time all expulsive power may be lost, so that albuminous concretions in the air tubes are a frequent post-mortem appearance, and are often remarkable in the trachea; it does not necessarily follow that the inflammatory action has been more intense at this part, still less that this has been its starting-point.

The symptomatic fever which accompanies the disease often has its sthenic character modified at an early period by the imperfect aëration of the blood; an early indication of this is afforded by the cerebral phenomena: some of the symptoms most distressing to witness are not those most felt by the sufferer; movements at first instinctive become imperfectly coordinated, and with the impairment of consciousness purely convulsive; the bending backwards of the head in the latter stages is not most frequently observed when the trachea is most obstructed, but is referable to convulsive action; and spasm may be the immediate cause of death. Whether respiration be thus suddenly stopped, or more gradually abolished, the actual termination of the disease is always by apnœa.

Morbid Anatomy.—Intense redness of the mucous membrane is persistent after death; swelling is seldom found, though sometimes the aryteno-epiglottidean folds are considerably distended, so as to diminish the upper opening of the larynx. Swelling may also be noticed at the base of the epiglottis, in the sacculus laryngis, and at the superior vocal cords and ventricles of the larynx; the mucous membrane is not much thickened, and has rarely undergone softening; sections of the mucous folds sometimes discover serum, sero-purulent fluid, or even pus beneath; pus has also been found disseminated between the muscles and cartilages of the larynx; the surface of the membrane has not lost much of its smoothness or polish; small patches of semi-transparent lymph occur, or a soft whitish exudation rests on some parts of the surface or fills the ventricles, or, in its place, viscid mucus or pus may be found on the

[Fig. 7.

False Membrane in Trachea.]

upper edge of the glottis or filling the laryngeal pouch. Ulceration of the surface is rare, especially in acute cases; Dr. West has observed it in one case of idiopathic Croup not of the most sthenic type, and more frequently in cases of secondary Croup; in one such case he records[1] "an uneven granular appearance of the larynx, and ulceration of the epiglottis." In some cases of Croup secondary to measles, small aphthous ulcerations may occur above the rima glottidis coexistent with true aphthæ of the mouth; no deposit in the pharynx or slough of the tonsils is found in true Croup. Dr. Cheyne remarks[2] that "in other diseases inflammation and incrustation, extending from the fauces to the larynx, may often be observed, but in genuine Croup, as it exists in Scotland and Ireland, never." The formation of false membrane, more or less continuous, is a usual result of the inflammatory process in Croup; its most frequent situation is in the trachea, but its presence is not so invariable as to make it pathognomonic; in some cases shreds of lymph, or striæ of viscid mucus, or vivid redness only, are found in the trachea; in others a soft curdy lymph, or pus, is present in some

[1] Lectures on the Diseases of Infancy and Childhood, by Charles West, M.D. 2d edition. 8vo. Lond. 1852. P. 220, note.

[2] Cyclopædia of Practical Medicine. Article, Croup, by J. Cheyne, M.D. London, 1833. Vol. i. p. 499.

quantity and extends into the bronchi. Where a more fibrinous exudation completely fills the trachea, it is separated from its surface by a layer of pus or of muco-purulent secretion, some of which may be found on both sides of the lower part of the false membrane, but it is less abundant and more viscid at its upper part. The false membrane may extend into the bronchi; its transition to a soft and less tenacious substance is so gradual that it may become at any point indistinguishable from the muco-pus covering the surface, which seems to be undergoing the same inflammatory process. The false membrane of Croup differs from that formed during the specific inflammation of diphtheria, both in its chemical and physiological relations; it is not simply fibrine, but consists of effused lymph in which the presence of albumen can always be chemically demonstrated: microscopically it is a mass of cystoid corpuscles; it is not the result of an interstitial change in the substance of the mucous membrane, but an exudation from its vessels and glands, so that the structure of the membrane producing it remains singularly free from pathological injury. This point has

[Fig. 8.

False Membrane of Croup. From a Specimen in Dr. Gross's Cabinet.]

arrested the attention of many of the observers of Croup. Albers, of Bremen, records the absence of all traces of inflammation of the subjacent mucous membrane in some of his cases, and considers that the plastic exudation being accomplished, the inflammation not only diminishes but probably ceases altogether. He notices also cases of another kind, and it is probable that two classes of disease were before him. MM. Barthez and Rilliet,[1] who represent the French school, in applying the terms Croup and pseudo-membranous laryngitis only to cases of diphtheria, remark "that the mucous membrane beneath the false membrane presents very various conditions — it is sometimes perfectly healthy; this is a fact we have ourselves established." And again, alluding to the number of cases of severe Croup on record where mere shreds of false membrane were found in the larynx and trachea, an instance of which they themselves observed, they ask, "May there not yet be a disease which presents so great a similitude to Croup that it has been confounded with it by most authors, and in which there is found after death either no alteration in the mucous membrane of the larynx, or a simple inflammation, without swelling enough to obliterate the air-passages?"

It is such a disease that we mean by Croup; and while in young children simple turgescence of the mucous membrane conjoined with spasm may be too soon fatal for the special products of inflammation to be found, and in others these products collected near the base of the arytenoid cartilages and in the sacculus laryngis may occlude the glottis at an early period of the disease; yet a more or less continuous membranous exudation may in other cases completely fill the trachea, and even extend into the bronchi; whether it is ever so closely identified with the mucous structure, or so purely fibrinous as is the special product of diphtheria, will require further observations to decide. In some cases the false membrane rapidly forms again after expulsion. Sir Thomas Watson relates a case[2] where after tracheotomy it was completely re-formed in six or seven hours: the rapidity of its formation indicates a developed specialty rather than an original intensity of inflammation, and numerous instances prove that the most vigorous antiphlogistic treatment has no effect in retarding its development, while in some acutely inflammatory cases of Croup going on to their fatal termination, no false membrane has been expectorated, and pus only has been found in the windpipe.

The bronchi show more or less trace of inflammation in their whole extent: in the upper tubes a whitish concrete exudation is often developed, and sometimes presents sufficient tenacity to be removed entire from one or two divisions; the smaller ramifications are often filled with opaque mucus but slightly aërated. Pneu-

[1] Traité des Maladies des Enfants. Paris. 8vo. 1843. Vol. i. pp. 320 and 336.

[2] Lectures on the Principles and Practice of Physic. 4th edition. Vol. i. p. 856.

monia both lobar and lobular frequently coexists, and vesicular emphysema is generally present in some part of the lung. The heart may contain dark blood, both

[Fig. 9.

False Membrane of the Bronchial Tubes.]

in its right cavities and in the left auricle, and the venous trunks and sinuses are similarly distended; there may be congestion, but there is no fibrinous deposit found in the liver, spleen, and kidneys. The brain may show congestion of its vessels or slight serous effusions within its ventricles. Numerous enlarged lymphatic glands are found beneath the thyroid on each side of the trachea; sometimes lymph has been effused both in this situation and between the sterno-hyoid and sterno-thyroid muscles.

Prognosis.—We have no data from which to judge the proportion the mortality from Croup bears to the number of the attacks. The fully formed disease is one of the most fatal to which childhood is liable, and the younger the sufferer the less favorable the prognosis. Were every case considered as one of Croup, in which hoarseness of voice and peculiarity of cough lead to the prompt and careful adoption of the means necessary to ward off the disease, we might under the most favorable hygienic conditions come near to the hopeful conjecture of Dr. Wood, of Philadelphia, that one case in fifty only is fatal. The mortality estimated by Jurine, of one in ten, is probably nearer to what we experience; where it has been placed as high as one-half, two-thirds, or four-fifths, cases of diphtheria are included; nor is it surprising that the most fatal complication of a very fatal disease should be thus mortal when, under a misconception of its nature, the course of treatment pursued has been such as would endanger the result of uncomplicated diphtheria.[1] The inherent fatality of secondary Croup is also increased by subjecting it to the course of treatment required in uncomplicated cases, and the favorable prospect of some of these is sometimes in jeopardy from the too prolonged use of tartar emetic. The slightest cases of Croup furnish grave cause for anxiety; for some of the most severe there is a hope in reserve, faint though it be, which is offered in few other acute diseases. Early treatment has a great effect on the favorable result of even the most severe cases. The most active treatment will often be of the least avail in the advanced stages.

The signs of a favorable progress are a softening in the tone of the cough, a diminution in the frequency and severity of the attacks, with a more free entrance of air into the chest, a less distress of breathing in the intervals, a return of the voice towards its natural tone, a loose or moist sound with the cough, and the possibility of a comfortable repose. This change in the symptoms must be continuous for twenty-four hours, and no lung complication present itself, before confidence is warrantable.

If the paroxysms be more frequent and more violent, and there be no diminution of the dyspnœa, or an increased restlessness in the intervals; if the voice be suppressed, and the cough less powerful or absent; if the expansion of the lung be reduced till the chest walls seem retracted or flattened, there is no hope of a favorable change taking place; and even if death be not sudden, it will come on more slowly, but not the less surely, with evidences of disordered consciousness and coma: where this stage is reached, though from some unexpected relief air has been again admitted into the lung, and some of the functions of life are resumed, and even continue for a time, recovery is scarcely possible.

Treatment.—Few diseases are so dependent for their relief on the prompt and careful adoption of a thoroughly antiphlogistic treatment at their very commence-

[1 When positively *membranous*, however, Croup is fatal in a considerable majority of cases.—H.]

ment as is true idiopathic Croup. Unless the first threatening symptoms have received all the attention they demand, a free abstraction of blood may be indispensable in checking its progress. The induction of vomiting in the early stages is often the most effective means of arrest, and one that must be resorted to in all stages of the disease but the last. The air to be respired will require its temperature elevating and made equable by day and night, and aqueous vapor should be diffused therein. The warmth of the surface and the application of warmth externally must be attended to, and diluents only allowed to be taken.

The exclusion of every possible irritant, as well as moderating the degree of irritation already existing, are of the utmost importance, either in preventing the progress of the threatened malady, or in favoring its arrest and subsidence.

However urgent the symptoms, attention must first be directed to their course and development, and their cause ascertained; before our special remedies are employed steps can be taken to secure those general aids which are indispensable. There must be a fire in the room, and a large quantity of hot water in readiness for a bath must be prepared; meanwhile some hot water can be brought to the bedside, sufficient perhaps for the immersion of the child's arms; the steam from it may be of some service, or sponges wrung thoroughly dry from it and repeatedly applied to the throat will afford some relief; the nurse or chief attendant upon the child must be retained in the room not only to assist in these measures, but to afford the necessary information as to the commencement of the attack and its antecedents, while at the same time the degree of embarrassment of the breathing, the state of the pulse, of the cervical glands, and of the surface generally, can be noted. An emetic should be in readiness, either the ipecacuanha wine, or the antimonial wine, or both; a combination of the two in equal proportions is well suited for the earliest stages, or a mixture of twelve grains of powdered ipecacuanha with a drachm of syrup, or of mucilage, and three drachms of water, will be of the same strength as the wine; an aqueous solution of the tartarized antimony of any strength can be made by means of hot water; some of this solution, containing at least a grain of antimony, must be added to the above mixture, the half of it given early to excite vomiting, and the remainder in less than half an hour, if that action has not commenced. In this interval an inspection of the throat should be obtained, and care must be taken to make it thoroughly efficient; a good light is required; if artificial light is needed, three candles cemented together will afford it; the child must be well secured in the nurse's lap, the head resting against her shoulder—sudden movements of the head, arms, or feet, must be guarded against, and by passing a firm unyielding instrument, or the broad handle of a tablespoon, to the back of the tongue, it can be depressed and at the same time brought forward so as to show the epiglottis. This inspection may be convenient before the first emetic dose is given, and if any doubt exist as to the nature of the attack, would then be advisable; whenever it is accomplished the diagnosis is complete, and the activity of the means employed can be proportioned to the severity of the disease, the stage at which it has arrived, and the condition of the patient. [In simple spasmodic "night croup," the prompt use of ipecacuanha alone, in powder, syrup, or wine, will often suffice to produce the needful relaxation and secretion. For a child four years old, 15 or 20 drops of the syrup of ipecac. may be used, to be repeated in a quarter of an hour unless relief to the breathing occurs. Vomiting is not in itself necessary, but the relaxant should be pushed until this follows, or until the respiration becomes soft and free. Ipecacuanha has the great advantage of doing no harm (simply acting as a mild purgative) if it should not be rejected by the stomach. The old remedy, "Hive Syrup," is objectionable, and, with young children, unsafe, on account of its containing tartar emetic.

A certain number of cases of night croup exhibit, in the recurrence of attacks, a predominance of the nervous element over the hyperæmic, so as to call for such remedies as assafœtida, hyoscyamus, or musk, in combination with ipecacuanha, and, if needful, with sinapisms and the warm bath.—H.]

Before repeating the emetic it will be advantageous to make use of the warm bath: this should be of a temperature of 98° or 99°, and maintained at the higher degree of heat, or even raised two or three degrees above it by the gradual addition of hot water; a warm blanket must be in readiness to envelop the child on removal, and hot towels to dry the surface completely. The air of the chamber should be raised to a temperature somewhat over 65°, and never allowed to fall lower; it can be made moist by placing a kettle of boiling water on the fire with a tin tube fixed to the spout, or a long roll of paper, to convey and diffuse the steam. After the action of the emetic there will generally be great relief to the distress of breathing, and quiet sleep will follow. It is now the time to listen attentively to the breathing; however complete the relief afforded by the emetic or the warm bath may be, the patient must be seen during

sleep, or visited again at night, not only to see that the improvement continues, but for the purpose of noting the state of the respiration; the stethoscope should be applied to the side of the neck or to the top of the sternum to ascertain the character of the tracheal bruit, to the upper part of the chest in front and to the lower part of the side or back, in aid of other observations as to the degrees of freedom with which the air is entering. If the result be satisfactory, it may be sufficient to prescribe ten grains of alkaline citrate of potash, or five grains of chlorate of potash, to be given every four hours, freely diluted with water or with milk and water, and a grain or two of calomel for its aperient action, which may be aided if necessary by castor oil in the morning; it is better to produce this effect by giving three or five grains of calomel at once, should the child have gone through a severe paroxysm of dyspnœa. If there be persistence of the febrile symptoms, of the cough, or of any laryngeal quality in the breathing, half-drachm doses of antimonial wine must be given with each dose of the saline, or a smaller dose at more frequent intervals, taking care with young children that it is given less frequently as the symptoms subside, and that it is omitted altogether as soon as relief is obtained. The emetic must always be at hand, so that in cases of a threatened paroxysm its full effect may be again induced. It is to be borne in mind that the paroxysm has a tendency to recur even when the disease is not advancing, and that the recourse to the warm bath may afford such relief as to enable the air to be drawn in again either with freedom or with lessening signs of obstruction.

The attack, when treated early, is not unfrequently arrested; the patient requires to be carefully watched, that any return of the croupy symptoms may receive timely attention; however favorable the progress, the child should be confined to bed for two or three days, the diet being gradually increased; the temperature of the room is to be maintained during this time, and great caution exercised before allowing the patient to leave it, the chest symptoms meanwhile being anxiously observed. Where the season is cold, or unusual susceptibility has been induced by repeated attacks of Croup, it may be necessary to restrict the patient for ten days or a fortnight to an apartment wherein the air is artificially moist and warm.

In the more severe cases a tent should be formed over the child's bed, to which steam can be admitted from the long spout of the kettle, which, filled with boiling water and placed behind the bed, can be kept hot by a spirit lamp or other means; the temperature within can be regulated from 70° to 75°, but at times it may be raised to 80°, and a larger quantity of steam be admitted with advantage. Calomel should be given from the first, and repeated frequently in small doses, interrupted occasionally for the repetition of the emetic; a grain, or half a grain of calomel, combined with the quarter or eighth of a grain of ipecacuanha, according to the age of the patient, is to be prescribed every two hours; if the bowels become disturbed, it can be persisted with in diminished doses until its characteristic effect on their secretions is obvious, nor is it then to be entirely discontinued. Antimony has great power in moderating the intensity of the first stage of the inflammation; it may, therefore, be used alone to cause vomiting, and as it will be necessary to repeat it with this object, it is better not to give it in frequent small doses, which, by inducing in the system a tolerance of its influence, render its emetic effect less easily obtained; for the same reason it is not to be the only emetic employed; where it is of service its good effect is soon observable, and can be secured by giving it in small doses with other emetics. In the later stages of the disease it is entirely inadmissible.

[Alum, introduced as an emetic in Croup by the late Prof. C. D. Meigs, of Philadelphia, appears sometimes to have decided efficacy in unpromising cases, when false membrane is being deposited. It is much less harsh than antimonial preparations.

Dr. James, of New York, has reported[1] a case of membranous croup in a child two years old, almost moribund, in which the hypodermic injection of $\frac{1}{10}$ grain of hydrochlorate of apomorphia was followed by vomiting, with ejection of a tracheal cast of membrane, and recovery. —H.]

In some cases, the necessity for bloodletting has to be considered in the earliest stages of the treatment: in certain districts, where the subjects of the attack are well nourished, and living much in the open air, the early abstraction of blood has been found by experience to be a main element in the favorable issue of the illness; there are also certain cases where the attack is of such severity that the influence of the emetic is better aided by the loss of blood than by the use of the warm bath. In these cases the bleeding should be practised before the action of the emetic has commenced, and the blood should be withdrawn rapidly, so that the system may at once feel the effect of the loss; the external jugular vein affords the requisite flow most readily; it may be obtained from the

[1] N. Y. Med. Record, April 26, 1879.]

arm, except in young children, and with them leeching is not so efficient a substitute as might be expected. The youngest children bear the loss of one or two ounces of blood better than abstinence from food, or the effects of depressing medicine; for a child of four or five years of age, bleeding to the extent of three or four ounces will suffice, either for present relief or for a check upon the advance of the disease; it is to be remembered that in the cases where it is most indicated, neither of these objects will be attained by this remedy alone; even where the immediate relief is great, in a few hours there will be a return of most of the symptoms which generally indicate its employment, and these will require for their control many of the means already mentioned, probably with the aid of local bleeding. General bleeding is only of service in the early stage of the disease, it is not to be repeated; loss of blood is only allowable while there is heat of skin, florid hue of face and lips, and firmness, as well as fulness of pulse, but these conditions do not of themselves demand it; it is to be sparingly resorted to among town populations; it is seldom advisable where the attack accompanies any marked deterioriation of health; and it is contra-indicated in almost all cases of secondary Croup.

Local bleeding by means of leeches is of great service wherever the disease is progressing towards its full development; the relief thus obtained is often very great, and may be afforded more than once if other considerations do not render such means of relief unadvisable; the influence of even a moderate loss of blood in this manner may either favor the subsidence of the disease, or the specific action of calomel upon its products, while in conjunction with the warm bath it may often replace with advantage the too frequent repetition of antimony. Leeches are best applied over the mastoid processes, or a little lower on the neck if a larger number are to be used or a free afterflow from their bites is desirable; in the former situation these can be readily closed by means of dry lint aided by pressure if required, in the latter they can be covered with a linseed poultice.

External warmth to the neck and chest is useful; it can be applied without the necessity of frequently disturbing the child, by cloths wrung out of hot water and covered with warm towels or by oil-silk and handkerchiefs, or by small bags containing heated bran, which can be accommodated to the child's changes of position. Care should be taken that no part of the surface is chilled, and dry, warm flannels should be from time to time applied to the body, legs, and feet.

Counter-irritation is of doubtful efficacy,[1] the application of tincture of iodine to the sides of the neck is of some service, and acts more beneficially when it is covered with water-dressing. Linseed poultices, not too moist, with which a little mustard is mingled, may be usefully applied to the back of the neck and shoulders, or even to the legs; where signs of bronchial irritation are found at any part of the chest, or there is less expansion at one part than another, the advantage derived from having that part covered with a large simple linseed poultice is very great; if the other symptoms are favorable, it may be sufficient to use stimulating friction over these parts three times a day when the poultice is changed. A blister may be required for more serious pulmonary implication, but must be strictly limited to a particular spot, and should be so dressed with cotton-wool as to give rise to no ulterior pain or discomfort.

Opiates are to be avoided; sleep is needful, and will naturally happen while the disease is within safe limits, then the easy respiratory movement and increasing roundness of the chest present a strong contrast to the flattening and retraction observable during restlessness and excitement; sleep will continue while that normal condition of the chest is maintained, if not, it is soon interrupted, and there would be danger in its being artificially prolonged.

The strength is to be carefully guarded during the necessary contest with disease, and requires early support; milk and farinaceous food as well as whey and barley-water, are soon necessary; if the attacks of dyspnœa have been severe, beef-tea or chicken broth may be given early; if distasteful, or tending to excite spasm in deglutition, they are to be given in small quantities by the rectum. The various meat essences, prepared after Liebig's formula, are very serviceable, as they contain the restorative salts of the flesh without any of the protein compounds requiring gastric digestion.

Alcoholic stimulants are injurious, until the primary obstruction to respiration is overcome, and some pulmonary complication is the source of danger. The stimulant expectorants are often required, and when repeated emetics are indicated in the decline of the disease a stimulant should be combined; ammonia, or the ammoniated tincture of valerian, may be added to the wine of ipecacuanha given for this purpose; the tincture of lobelia may also be cautiously used as an adjunct, but not if there be much obstructing secretions; senega is here of the greatest value,

[1] At an early stage, a sinapism applied over the sternal region may be decidedly useful.—H.]

either in large, repeated doses as an emetic, or in combination with ammonia and squills as an expectorant; for this purpose, when the occasion arises, a grain of carbonate of ammonia with two or three minims of tincture of squills and a dessert-spoonful of infusion of senega may be given every two or three hours, as prescribed by Dr. West, mixed with a very little milk and sweetened with treacle or coarse sugar; the infusion of senega should be made with an ounce to ten ounces of boiling water, or of double the pharmacopœial strength, and its pungency shielded as above, or by the addition of glycerine; it may be freely given for its emetic effect, after the first urgency of the attack has subsided, and it may be aided in its action by the addition of ipecacuanha wine and of tincture of squills. When emetics are most beneficial, care must be taken so to regulate their employment as not to interfere with the necessary absorption of nourishment from the stomach, as sustaining the integrity of the vital powers is an important element in calculating the hopes of a final recovery.

From a very early period in the treatment of Croup, from the earliest if the disease has made some progress before treatment has commenced, the necessity for affording relief by the operation of tracheotomy has to be carefully considered, and steadily kept in view; at any moment the best judged means of treatment may be rendered nugatory by threatened suffocation, and whenever this is imminent tracheotomy is to be performed. However insidiously the condition of apnœa may come on, if it be advancing and its course unvaried by temporary improvement, unless we are satisfied that the cause of apnœa is in the pulmonary tissue itself, and not chiefly in the primary air-passages, no period of the disease, nor the surrounding conditions of the patient, scarcely even the age of the sufferer, should determine us to withhold this chance of life. The extension of the disease beyond the point at which the trachea is to be opened does not prevent the success of the operation; actual inflammation or consolidation of the lung, which would preclude recovery, will have been ascertained in its course, and can be determined by physical examination of the chest at any period; any extension short of this cannot be so determined when the distress of breathing is at its height. Where a certain extension seems probable, the operation is not always contra-indicated; the admission of air restores vitality to the system, and affords a mechanical aid in expelling the morbid products, thus tending to prevent further change in the lung itself. The surrounding conditions that are essential in undertaking tracheotomy are the same that are necessary for the successful treatment of cases where this extreme means of relief is not demanded; some approach towards securing these can be made, whatever the social state of the patient. The treatment is not to be discontinued after the urgent signs of distress are obviated by tracheotomy; and though there be now no longer need for some of the medicines, greater attention is, if possible, required in regulating the state of the air to be respired. The most frequent cause of death after the operation, next to the use of too small a tube, and the risk of its becoming obstructed by secretions, is extension of the disease to the lung; this is the natural termination of the disease, whenever the state of the trachea allows it to run its course.

The age of the patient has hitherto been closely connected with the success of tracheotomy; the unfavorable result of this operation among young children has seemed to be from the difficulty at this period of life of counteracting the asthenic tendency of the disease for which it has been chiefly practised; some results of this operation at different ages are given by M. André,[1] when Interne at the Hôpital des Enfants Malades, from which it appears that, while of the cases beyond six years the recoveries are one-half, they are not one-fourth of those under that age, and of six cases under two years old there was not one instance of recovery. Most of the statistics relating to tracheotomy are drawn from cases of diphtheria, so that analogical deduction from them is unsafe; they show, however, one uniform result that is doubtless applicable to the disease and to this climate, that the relative proportion of cures increases with the frequency with which the operation is attempted. Dr. Buchanan of Glasgow has operated[2] twenty-six times with nine recoveries; of eleven cases operated upon by Dr. Cruickshank during two years in a wild country district in Scotland, eight were successful.[3] Mr. Spence[4] of Edinburgh has published some most interesting and instructive reports of cases of Croup, in eight of which he performed tracheotomy with three recoveries. The facts brought together by Dr. Fuller[5] in his valuable paper on tracheotomy in Croup, and the improvement in the tra-

[1] On Tracheotomy in Croup, by M. André, Bulletin de Thérapeutique. Paris, 1857. Tome ii. p. 471.

[2] Tracheotomy in Croup and Diphtheria (additional cases), by George Buchanan, A.M., M.D. Glasgow, 1866.

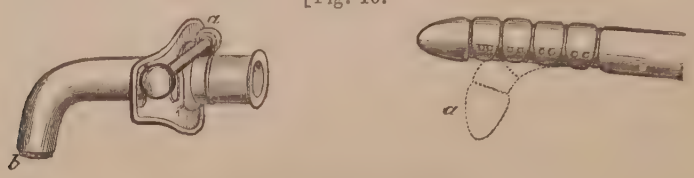
[3] The Science and Practice of Medicine, by W. Aitken, M.D. Vol. i. p. 587. (Third Edition.) London, 1864.

[4] Edinburgh Medical Journal, 1860, p. 693.

[5] Medico-Chirurgical Transactions, vol. xi. p. 50. London, 1857.

cheal tube, recommended by him and M. Gendron de l'Eure, contribute powerfully to this conclusion. The want of success with children under three years of age in this country ought not to discourage the operation. Mr. Henry Smith[1] details two instances, one a child of eleven months, and the other two years, wherein the difficulty of maintaining free the opening into the trachea seemed the only obstacle to recovery. It is somewhat remarkable that in the first three successful opera-

[Fig. 10.



Durham's Canula and Pilot.]

tions in this country for this disease no tracheal tube was used: two of these cases are recorded in the 3d and 6th vol. of the Medico-Chirurgical Transactions; the third was by Mr. Carmichael of Dublin, in 1820,[2] who maintained the opening for a week by the aid of tin retractors on the edges of the external wound. One of Dr. Buchanan's most urgent cases was brought to a successful termination without the use of a tube.[3] Dr. Wood, in his treatise on the Practice of Medicine, 5th edition, vol. i. p. 865, mentions three cases in which Dr. Pancoast, of Philadelphia, "removed a small piece of the trachea itself, thereby superseding the necessity of the canula, and avoiding irritation from that source;" two of these were successful. The fear of subsequent narrowing of the trachea renders this objectionable; passing a ligature through each edge of the tracheal wound might keep it open with safety for a short time; or a piece of wire bent like an eye-speculum, as made for me by Coxeter, might be used to separate the edges. No tube with less than a quarter of an inch diameter is sufficient to carry on respiration; at a year old such a tube cannot be introduced into the trachea; it would not be tolerated at two years old, so that at these ages other means must be looked for to secure a passage for the air: if it should prove that in a certain class of cases the mere opening of the trachea is sufficient, and that recovery is frequently possible without the introduction of the tube, then there would be room to hope for success even in the youngest children. [In Dr. J. Solis Cohen's monograph on Croup and Tracheotomy,[4] statistics are given of this operation in different countries. Those from American sources include 325 cases of tracheotomy, with 84 recoveries; or rather more than 1 success for 4 operations. Bouchut, Bergeron, and Barthez have reported not very different results in France. In eight London Hospitals (to 1859) 170 operations were recorded,[1] with 57 recoveries. The most favorable results for a considerable number of cases appear to have been those of Trousseau, in Paris. In private practice he is reported as having had, between 1851 and 1854, 24 operations, with 14 recoveries. Trousseau advocated *early* operations; the prognosis of which is no doubt better than that of those postponed until a moribund state is reached. In this country, the uncertainty of the operation itself, and the possibility of the false membrane being detached so as to allow recovery without operation, have made it generally difficult to avoid postponing it until a very late stage. All statistics show particularly unfavorable results from tracheotomy with patients under two years of age.

In regard to the operation itself, Trousseau, C. West, and R. W. Parker[2] particularly urge the importance of cleaning out the trachea, with a feather or otherwise, before inserting the tube. Besides its not being too small, the tube should have a curve not too great; imitating rather (to use Parker's language) the Gothic than the Roman arch. If, afterwards, the inner tube should be found to be dry, West and Parker advise spraying into it a solution of sodium bicarbonate, ten to twenty grains to the ounce of water, from time to time. —H.]

One great cause of non-success from this operation has been owing to cases of diphtheria being mistaken for Croup. The striking difference in the character of the two diseases, and in the treatment they require, struck me forcibly during the observation of some cases at the Hôpital des Enfants Malades, under the care of Trousseau, in the summer of 1850; the want of success in the earlier operations

[1] Medical Times and Gazette, 1853. Vol. i. p. 244.

[2] Transactions of the King and Queen's College of Physicians, Ireland.

[3] Glasgow Medical Journal, January 1st, 1862.

[4] Philadelphia, 1874.]

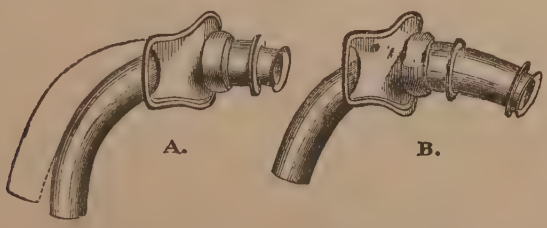
[1] Med. Times and Gazette, Oct. 15, 1859.]

[2] Lancet, Nov. 30, 1878.]

at that institution may partly be attributed to such cases being treated as Croup in our sense of the word. The favorable result in some cases recorded by Dr. Conway Evans[1] may be attributed to his recognition of their true relation in this respect. During the period 1850–53, but few of the croupal affections that came under my notice at the Marylebone Infirmary were cases of diphtheria; during the last ten years the majority of cases in London requiring tracheotomy have been cases of this kind.

For the operation a good light is indispensable; also, two assistants, one of whom may be the nurse, but two in addition are preferable; an unyielding cushion to support the shoulders, so that the head may rest well back upon the table beneath. An incision not less than an inch and a half in length is to be made exactly in the middle line; three ink-spots on the skin may be made to indicate this, and further, to avoid any superficial vein, the skin should be pinched up on both sides, and transfixed with the knife cutting outwards. Each layer of fascia is to be divided on a director, the knife-edge always being turned from the sternum, the isthmus of the thyroid is to be pulled upwards by a blunt hook, and the cellular tissue at its inferior border parted by the director, with as little use of the knife as possible; nevertheless the trachea should be bared over the line at which it is to be opened; the edges of the external wound are to be held apart by blunt hooks or wire retractors; small sponges affixed to stems are useful; all hemorrhage is to be restrained before the trachea is opened; a sharp hook may be used to fix the trachea, or it may be seized below with the artery-forceps. I have seen no sharp-pointed director suited for perforating the trachea and guiding the knife securely; having the thyroid drawn well up and shortening the knife in the hand with the edge directed upwards suffices to enter the trachea safely and to secure a well-placed opening. The expanding forceps or dilator is a useful aid in the introduction of the tube. However retracted the chest walls, some escape of air and mucus generally follows the knife, and then a full inspiratory action restores at once the normal outline of the chest; if respiration have ceased, it would be desirable by means of a tube to make suction from the trachea before commencing artificial inflation of the lungs or the auxiliary respiratory movements. Chloroform has been used safely in this operation on several occasions, but it may be better in some cases to abstain from any addition to

[Fig. 11.



Bryant's Canula. A. Full length. B. Shortened.]

already existing causes of apnœa, although it much facilitates the operation.[2] The tube introduced should always be the spring-sided, bivalve one; the inner tube should have an opening in its upper convex surface, as used by Liston; after a time a valve fitted to one of this kind, as recommended by Mr. Thomas Smith,[3] to admit of inspiration through it, and of expiration through the larynx, is a valuable addition; the collar recommended by M. Trousseau advantageously intervenes between the outer extremity of the tube and the skin.

[It is important, after tracheotomy, for the atmosphere breathed by the patient to be warm and moist. Some practitioners assert the advantage, in all cases of serious croup, whether operated on or not, of maintaining a temperature around the patient of 90° or 95° Fahr., kept in a high degree of humidity by the generation of steam. Inhalation of the vapor of lime-water, and of that of a solution of lactic acid, is reported to promote the dissolution of the membranous exudation. Such remedies are, at least, safe and worthy of trial.—H.]

After the operation free use is to be made of nutrient and remedial enemata; liquids can sometimes be swallowed with ease, at others they require always to be given in the form of sop.

Secondary Croup requires support or

[1] Edinburgh Medical Journal, 1860. Vol. v. p. 400.

[2] Ether is undoubtedly safer for this use.—H.]

[3] The Obstacles to the Re-establishment of Natural Respiration after the Performance of Tracheotomy, by Thomas Smith, F.R.C.S. A paper read before the Medico-Chirurgical Society, June 27th, 1865.

stimulation from the first; no emetics more depressing than ipecacuanha wine are available for repetition; this one answers very well when they are only required to meet the nocturnal remissions, at other times this interferes too much with the desire for nourishment; small doses of sulphate of copper in solution, as proposed by Hoffman, act well as an emetic on repetition; in some cases alum, as recommended by Dr. Meigs of Philadelphia, teaspoonful doses of the powder being given in honey or syrup every ten or fifteen minutes; it is rarely necessary to give the second dose, and the emetic effect may be obtained several times a day without exhausting the patient. An occasional dose of calomel is of service; there is no necessity for the continuous use of mercury, and its full influence is prejudicial. Great benefit often results from the administration of full doses of perchloride of iron. Salines are only admissible when Croup complicates the early stage of measles; at other times and in the other exanthemata the mineral acids are better adjuncts. The air to be respired need not to be kept as warm and moist as in primary Croup, but there must be the same care as to its purity. Close attention is to be given to the state of the fauces; local applications are of service here both as a stimulus to the mucous membrane near the entrance of the larynx, and for the removal of the mucosities which offer some impediment both to respiration and deglutition. If nourishment and wine are not readily swallowed, nutrition is to be supplemented by the injection per rectum of small quantities of beef-tea, to which a little brandy must occasionally be added.

Varieties.—There is great difficulty in defining the varieties of Croup that have been described: the greater number of them depend either on the inclusion of other diseases, or on a misapprehension of the nature of this; the terms spasmodic and nervous have been applied to the most acute inflammation of the trachea, as well as to spasm of the glottis; while pseudo-membranous, inflammatory, or asthenic have probably included more cases of diphtheria than of Croup, Catarrhal Croup has been applied to all these varieties when they fortunately have had recovery as their one common result. Croupal catarrh may exist independently, the inflammation being superficial and, under favorable circumstances, rapidly passing off: stridulous laryngitis, as used by M. Bretonneau, may be taken as its type; though not requiring the most energetic treatment, it must never be neglected. The temperature of the patient here affords the most valuable indication of the gravity of the disease. In merely spasmodic attacks there is little or no elevation of temperature. The stridulous laryngitis or false Croup of M. Guersant includes many of the slighter cases of Diphtheria as well as of Croup; he has remarked[1] the frequency with which it occurs among the upper classes of Paris rather than among the poor, and that it is sometimes observed in connection with exudation on the fauces,[2] a complication which he justly considers as "fort embarrassante pour le diagnostic;" these circumstances prevent the terms False Croup and Catarrhal Croup from being considered equivalent. Epidemic Croup is strictly diphtheria; when that disease prevailed epidemically in England at the end of the last century, any fresh outbreak of it was so spoken of; an outbreak at Chesham, in Buckinghamshire, in 1793, carefully described by Mr. Rumsey, leaves no doubt on this point; sometimes on its appearance in a fresh locality it was simply called Croup, and the word excited as much terror then, as diphtheria has again given us reason to associate with the disease it now designates.

[Allusion has been made above to the subdivision of croupal affections into three varieties, besides the laryngo-tracheal lesion of diphtheria. A sufficiently complete classification of the disorders in which *laryngeal dyspnœa* occurs as a leading symptom, may be the following:—

Laryngismus Stridulus;
Spasmodic Night Croup;
Croupal Catarrh;
Pseudo-membranous Laryngitis;
Laryngeal Diphtheria.—H.]

[1] Archives Générales de Médecine, tome xvii. Croup et Pseudo Croup. M. Blache, p. 493.

[2] Ibid. p. 507.

DISEASES OF THE RESPIRATORY SYSTEM.—*Continued.*

B. Diseases of the Thoracic Organs.

1. Emphysema.
2. Asthma.
3. Phthisis.
4. Cancer.
5. Acute Pneumonia.
6. Chronic Pneumonia.
7. Syphilitic Affections of the Lung.
8. Brown Induration of the Lung.
9. Cirrhosis.
10. Apneumatosis.
11. Bronchitis.
12. Pleurodynia.
13. Pleurisy.
14. Hydrothorax.
15. Pneumothorax.

EMPHYSEMA OF THE LUNGS.

By Sir William Jenner, Bart., M.D. Lond., D.C.L. Oxon., F.R.S.

Definition.—Relative excess of air in a part or the whole of the lungs.

The relative excess of air may be the result of increase in the quantity of air in the vesicles, of diminution in the solid tissues of the lung, or of the presence of air in lung structures which in health do not contain air.

Pulmonary Emphysema may be divided into—

1. Interlobular, extra-vesicular, or extra-alveolar Emphysema.
2. Vesicular or alveolar Emphysema.

This division, first made by Laennec, has been adopted by all subsequent writers on the subject.

Interlobular, Extra-Vesicular, or Extra-Alveolar Emphysema.

Definition.—Air in the connective tissue of the lung.

The connective tissue of the lung is seated chiefly, at least, between the lobules and under the pleura. The air in extra-vesicular Emphysema occupies the meshes of this connective tissue.

When air is present in the connective tissue between the lobules, it accumulates in small bubbles of tolerably equal size, separated from each other by bands of tissue, so that the surface of the lung looks as if streaked or crossed by rows of small beads. When air is in the sub-pleural tissue it forms air-blebs, sometimes of very large size.

Air may be formed after death in the connective tissue of the lungs by decomposition; it may be generated there during life by gangrene; and it may be extravasated into the same tissue in consequence of rupture of the normally air-containing structures of the lung. When formed by decomposition after death, the gas is usually seated in the interlobular tissue; when generated by gangrene, in the sub-pleural tissue; and when extravasated from the air-vesicles, it commonly occupies both situations.

Air extravasated into the connective tissue of the lung occasionally finds its way into the posterior mediastinum, and thence into the subcutaneous tissue of the neck, face, trunk, &c.

Rupture of the normal air-vesicles may be the result of injury inflicted from without, or of the pressure of the air on their inner surface during violent expiratory efforts made when the glottis is closed, *e. g.* during cough and parturient efforts. The distension of the air-vesicles by inspiratory efforts is never great enough to cause their rupture.

Interlobular Emphysema is a condition of little importance. When the air finds its way through the connective tissue of the posterior mediastinum into the subcu-

taneous tissue, the air is quickly absorbed, and in a few days no trace of the Emphysema is to be detected.

With the exception of the cases in which the air reaches the subcutaneous tissue, the diagnosis of pulmonary extra-vesicular Emphysema is impossible, and even in these cases there are no pulmonary signs or symptoms to indicate the existence of the local lesion.

Should the existence of extra-vesicular Emphysema be ascertained, no treatment is needed.

Pulmonary Vesicular Emphysema.

Definition.—Increase in the capacity and size of the air-vesicles of the lungs.

Pulmonary Vesicular Emphysema is a very common, and frequently a grave disease.

Causes of increase in the capacity and size of the air-vesicles.—All forms and degrees of Pulmonary Vesicular Emphysema have their origin either in destruction of the partitions between the air-vesicles, or in over-distension of individual air-vesicles. In the former case, two or more air-vesicles are, by the primary lesion, thrown into one; in the latter, each air-vesicle is, by an over-distending force, increased in capacity and size.

It is improbable that nutritive changes in the tissue of the walls of any hollow viscus ever lead directly to expansion of its cavity. But changes in the walls of a hollow viscus may weaken their resisting power and so favor the expansion of its cavity; and again, changes in the walls of a hollow viscus may cause a dilatation to be permanent, which otherwise would have been temporary.

Changes in the walls of a hollow viscus, which strengthen their resisting power, may, at the same time, weaken their contractile power. Walls so changed may resist a dilating force longer than healthy walls, but should the dilating force be sufficient to stretch them, the dilatation of the cavity they inclose is permanent. The walls are indeed stronger, but then the cavity is more likely to suffer *permanent* dilatation.

The causes of increase in the capacity and size of the air-vesicles of the lungs are then divisible into:—

1. The forces which determine their over-distension;
2. The conditions which favor their over-distension;
3. The conditions which render their over-distension permanent;
4. The lesions of structure by which two or more vesicles are thrown into one.

Although this division should always be kept in view when considering the causes of Pulmonary Vesicular Emphysema, it will be better, in an article such as this, to consider the causes included in the second and fourth divisions incidentally, as occasion arises, when treating of the causes included in the first and third divisions.

Determining causes of over-distension of the air-vesicles.—Pressure of air on the inside of the air-vesicles is the force which directly causes their normal expansion; increase in that pressure is the immediate cause of their over-distension.

Excess of pressure of air on the inside of the pulmonary air-vesicles (of the whole or of a part of the lung) may be brought to bear,

(*a*) By excessive expansion of the chest-walls;

(*b*) By normal expansion of the chest-walls, when disseminated portions of the lung are shrunken, and no longer admit air;

(*c*) By unequal compression of the lung at the moment when there is impediment to the free escape of air from its air-containing parts.

(*a*) In health, inspiration is effected by muscular effort, ordinary expiration chiefly by the elasticity of the thoracic parietes and of the lung textures. The muscular effort of inspiration overcomes the resistance to the entrance of the air into the air-vesicles, offered by the elasticity of the lungs and of the walls of the thorax; the muscular effort ceasing, the elasticity of these parts is sufficient to accomplish the ordinary expiratory act.

The elasticity of the ribs and of their cartilages diminishes considerably as age advances, while in a large number of cases the muscles of inspiration continue as powerful as, and are sometimes more powerful than, in early life. The result of inspiratory expansion of the chest being constantly accomplished by the action of the muscles undiminished in power and activity, and of expiratory diminution of the chest being constantly performed incompletely and imperfectly by thoracic parietes, the elasticity of which is diminished, is gradual expansion of the chest-walls, increased capacity of the chest, and dilatation of the air-vesicles of the lungs. The capacity of the chest not being reduced to its normal size during expiration, the inspiratory effort is made on a chest retaining too much air in the lung-vesicles, and thus, especially if there be repeated and powerful calls on the inspiratory power, as from cough or great muscular effort, the result is considerable over-distension of the air-vesicles.[1]

In the same way lessened elasticity of

[1] Dr. G. Budd, in a paper on Pulmonary Vesicular Emphysema, published in the Med.-Chir. Soc. Trans. for 1840, clearly pointed out the part which loss of elasticity of the lung plays in the production of Emphysema.

the lungs from age-degeneration, or other cause, without loss of power in the muscles of respiration, leads to increase in the capacity of the thorax, and over-distension of the air-vesicles. The excessive expansion of the thorax, and therefore the dilatation of the air-vesicles in this, as in the last case, is determined by extreme muscular inspiratory action—the necessary result of deficient ordinary expiratory power.[1]

Another cause of increased expansion of the thorax has been described by Freund. He says that persons of all ages, from twenty years upwards, the well-nourished as much as the withered and decrepit, are liable to a chronic disease of the cartilages of the ribs, which results in their hypertrophy and increased firmness and rigidity, and in diminution of their elasticity. As this increase in the size of the cartilages takes place in all directions, the ribs and sternum are separated from each other more than they are in health; the ribs being forced outwards and upwards, and the sternum forwards and a little upwards. The capacity of the thorax is thus (Freund says) increased, and the lungs proportionately over-distended. It has been contended by later writers that Freund exaggerated the frequency at least of this affection; that he supposed changes in the cartilages resulting from their stretching to be the primary affection—in fact, that he took the *effects* of the action of the determining cause for the determining cause itself. For the capacity of the thorax to be increased under the conditions named by Freund, the diaphragm must continue to be at the termination of ordinary expiration on as low a level as in health. Usually, however—and perhaps always when the cartilages lengthen—they bend so as to form an angle, with its concavity upwards, about their centre.

(*b*) It is evident that if disseminated portions of lung are from any pathological condition diminished in size and no longer admit air, and that if, at the same time, the chest-walls expand during inspiration to the same amount as in health, the air-admitting vesicles must be over-distended in proportion to the number of cells into which no air enters, and the degree to which the airless vesicles are diminished in size. Thus, in certain cases of bronchitis, disseminated lobular collapse is common. The collapsed lobules are smaller in bulk than are the air-containing lobules—their vesicles admit no air during inspiration. The necessary result is, that if the chest-walls expand to the same degree as before the establishment of collapse, and so inspire an equal quantity of air, the capacity of all the air-vesicles still pervious must be increased.[1]

(*c*) If a lung removed from the body be moderately inflated, and the bronchus leading to it be tied, and then the substance of the organ be compressed at one part, over-distension of the air-vesicles of the uncompressed part is produced. Should the compressed part be large, and the compression considerable, even rupture of the air-vesicles of the uncompressed part may result. Under the conditions supposed, air is forced from the compressed parts of the lung into the air-admitting structures of the uncompressed parts of the lung.[2]

The conditions essential to the over-distension of the air-vesicles here present are:—

(*a*) Inflation of the lung.

(*b*) Closure of the natural passage for the escape of air from the lung.

(*c*) Unequal pressure on the lung.

(*d*) Unequal support of different parts of the lung.

During violent cough and great muscular effort, these essential conditions are fulfilled:—

(*a*) Preparatory to cough and to great muscular effort, a deep inspiration is taken, *i. e.*, the lungs are inflated.

(*b*) Then the glottis is closed, *i. e.*, the air is prevented escaping by the natural channel.

(*c*) Then, by the action of the expiratory muscles, the lungs are strongly compressed, and an examination of the structure of the thoracic walls at once shows that the compression must be unequal.

(*d*) Examination of the structure of the walls of the chest also shows that the support offered to the lungs by those walls is very different in degree at different parts.[3]

Again, when blowing a wind instrument the chest is expanded to its utmost, and then the chest-walls compress the inflated lungs—the air cannot escape as freely through the instrument as through the open glottis, and the mechanical effect is over-distension of the air-cells of the least compressed and least supported parts

[1] See p. 75.

[1] This point has been excellently well brought out by Dr. W. Gairdner.

[2] This expiratory theory was first advanced by Mendelssohn, in a very able paper, "Der Mechanismus der Respiration und Circulation," 1845. The writer of this article was unacquainted with Mendelssohn's paper when he advanced the same theory in 1857, and so far as he knows the existence of Mendelssohn's paper was unknown in this country, and rarely, *if ever*, referred to abroad till Biemer's article appeared in 1867.

[3] For details on this point see the author's paper on the Determining Causes of Pulmonary Vesicular Emphysema, in the Med.-Chir. Soc. Trans. 1857.

in proportion to the impediment to the escape of air and the force with which it is attempted to drive the air forward.

The over-distension of the air-cells thus effected will be in proportion to the amount of inflation of the whole lung, to the firmness with which the glottis is closed, or the smallness of the aperture of the wind instrument, or other obstacle to the free escape of air;[1] to the extent, degree, and difference in the force of compression exercised on the several parts of the lung at the same moment; and to the deficiency of support afforded to the less compressed parts by the thoracic parietes. The greater and the more extensive the compression of one part of the lung, and the less the compression of the other, the greater will be the distension of the air-cells in the less compressed part; and the less the imperfectly compressed parts are supported by the thoracic parietes, the greater will be the distension of their air-vesicles.

It would at first sight appear that the over-distension of the vesicles should be in all cases limited to the less compressed and the less supported parts of the lungs, but on further examination it will be seen that this opinion is erroneous. Thus, if from some change in the walls of the chest or of the air-vesicles, the latter continue over-distended after the force which directly determined their over-distension has ceased to act, or in other words, if there be permanent dilatation of the air-cells, then the size of the chest and of the lungs is permanently increased.

The portions of lung corresponding to the intercostal spaces are less compressed and less supported just before violent expiration than are the parts immediately under the ribs themselves. Now with every increase in the size of the lungs, or thorax, or both, the relative positions of the lungs and ribs are changed. As the chest enlarges, the ribs assume a more horizontal position, the lower intercostal spaces become wider, and their supporting power by so much diminished.

By these changes in the lungs and in the chest-walls their relative positions are being constantly shifted, and fresh portions of the lungs are being constantly brought to correspond to the ribs and to the intercostal spaces, &c., and thus, ultimately, the air-vesicles of the whole lung may be over-distended. But when the air-vesicles of the whole lung are thus over-distended, the dilatation of the vesicles at the apex and margin of the lung is in excess of the dilatation of the vesicles of other parts. Strong expiratory effort, while there is impediment to the free escape of air from a part or whole of the lung, is now admitted to be the most common efficient determining cause of over-distension of the air-vesicles.

Pulmonary Vesicular Emphysema is very common in horses, and for this reason, viz., that they are constantly making powerful muscular efforts with closed glottis. No one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort, the horse holds its breath, having previously inflated its lungs—no sooner, however, does the animal cease its effort than the glottis is opened and the air suddenly expressed from the lungs. The degree to which the air was compressed during the powerful effort (and the consequent strain on the less compressed and less supported part of the lung) may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapors is seen to be driven forth.

Permanence-securing causes, or the conditions which render over-distension, or increase in the capacity and size of the air-vesicles of the lungs permanent.—Whatever destroys the partitions between adjacent air-vesicles, and whatever permanently diminishes the ordinary or habitual respiratory power, must, to a like degree, be a permanence-securing cause of increase in the capacity and size of the air-vesicles. The permanence-securing causes, therefore, are:—

1. Direct injury to the elasticity of the walls of the air-vesicles;

2. Permanent diminution of the power of supporting or compressing the lung, at any one part, during violent expiratory efforts;

3. Changes in the structure of the parietes of the thorax, which permanently diminish their elasticity, and therefore their ordinary or habitual expiratory power;

4. Chronic changes in the structure of the lungs, which permanently diminish their elasticity, and therefore their expiratory power;

5. Atrophy of the septa between the air-vesicles of the lungs, by which two or more vesicles are thrown into one.

1. If the forces which expel the air from the air-vesicles, viz., the elasticity of the thoracic parietes, and the elasticity of the walls of the air-vesicles, are at the termination of over-distension of the vesicles in a state of health, then the force determining their over-distension ceasing to act, the air-vesicles return to their natural size; but if, as very rarely happens, the air-vesicles have been *very* greatly over-distended, or kept for a very long time over-distended, or have been very repeatedly over-distended, then the elasticity of the walls of the air-vesicles may be permanently injured, and the over-distending force ceasing to act, they

[1] Dr. Budd's case.

do not recover their normal dimensions. They are under the circumstances supposed permanently over-distended. The elastic structures of their walls have been directly injured by the over-distending force. So great even may be the force by which their over-distension has been effected, that the partitions between adjacent vesicles may be destroyed, and two or more vesicles thrown into one; or even, as has been previously mentioned, the destruction may have reached further, and air have been extravasated into the interlobular tissue.

2. The observations of Ziemssen on a case in which there was loss of muscular power in the four upper intercostal spaces, proves that this loss may be a cause of Vesicular Emphysema. In Ziemssen's case, during violent expiratory effort, these intercostal spaces no longer affording their normal support to the lung were forced outward so much as to stand above the level of the ribs. When the muscles of either intercostal spaces were stimulated to contract by faradization, then the bulging during expiratory efforts of that intercostal space ceased, thus proving that want of muscular contraction at any part during expiratory effort is a cause of over-distension of the air-vesicles of the lung at that point; and if the want of support be permanent, then certainly the over-distension will be permanent.

3. The degenerations of the ribs and cartilages incident to age diminish their elasticity, and consequently diminish the expiratory power of the chest-walls. If, as was previously pointed out, the inspiratory muscles act perfectly when the expiratory force resulting from the resilience of the ribs and cartilages is diminished, dilatation of the thorax, over-distension of the air-vesicles, and enlargement of the lungs are determined.

As age-degeneration is a permanent lesion, the loss of elasticity resulting from it is permanent; the dilatation of the thorax, over-distension of the air-vesicles, and the enlargement of the lungs, is permanent. Age-degeneration of the ribs and their cartilages is, with perfectly acting inspiratory muscles, therefore a permanence-securing cause of Large-lunged Vesicular Emphysema.

The disease of the cartilages of the ribs described by Freund, once established, is permanent, and therefore, the over-distension of the air-vesicles due to the expansion of the chest resulting from it, is also permanent.

4, 5. Whatever changes in the lungs diminish their elasticity, to the same degree render permanent the over-distension of the air-vesicles determined by any of the forces previously enumerated.

Diminished elasticity of the lung may be the consequence of those changes in texture which result from repeated or long-continued congestion. After a part has been the seat of long-continued or of repeated congestion, it is, if an organ, indurated and toughened; if a tissue, toughened and thickened. If death occur long after the outset of the congestion, then a certain amount of wasting of the original structures is found to have taken place. In some cases, certainly, these changes are due to the formation among the normal anatomical elements of the part, of imperfectly developed connective, fibrous, or fibro-cellular tissue.

All degenerations of texture incident to age are attended by more or less loss of elasticity.[1]

The degenerations incident to age,[2] as they affect the lung, may be divided thus:—

(*a*) Atrophy, or waste of all the anatomical constituents of the lung, with general diminution in its size. As the partitions between the air-vesicles atrophy, two or more vessels are thrown into one. This form of atrophy has been supposed to be preceded by fatty degeneration.

(*b*) Thickening of the fibrous element of the lung, with more or less waste of some of its anatomical constituents. When the subject of this form of degeneration the size of the lung is often increased, and it may be considerably so.

In this latter form of age-degeneration there is, at the outset at least, no atrophy of the inspiratory muscles; while in the former, the muscles on the outside of the chest are wasted and pale, and the diaphragm is thin, lax, and in folds. In both, the ribs and cartilages are the seat of degenerative changes attended by loss of elasticity.

So, also, when the ribs and cartilages lose elasticity from age-degeneration, the lungs rarely preserve their normal elasticity; they too, commonly, like the ribs and cartilages, are suffering from age-degeneration.

The conjunction of diminished elasticity of the lungs and of the parietes reduces the ordinary or habitually employed expiratory force to a minimum. Now, this

[1] Diminution of elasticity is one of the most marked effects of the changes in nutrition incident to advancing age, *e. g.*, of the skin, giving the aged look; of the arteries, causing them to become tortuous or S shaped, at first when the part in which they are placed is shortened, as in flexion of the limbs, and then permanently; of the intervertebral cartilages, of the elastic structures in the sole of the foot, the joints, the bones, &c.

[2] Those changes of nutrition which are the characteristics of age, and in fact constitute old age, may occur, generally or locally, at an unusually early age. Thus, one man grows prematurely old as regards his jaws, another as regards his hair, another as regards the heart, &c.

being the case, if the muscles of inspiration and of expiration retain their normal power, then frequent cough, habitual straining at stool, moving heavy weights, climbing hills, blowing wind instruments, or other causes of repeated and powerful inspiratory efforts, followed by violent expiratory compression of the inflated lungs, with impediment to the escape of air, will be followed by great and permanent increase in the size of the thorax, and corresponding over-distension of the air-vesicles.

Changes in the lung, attended by loss of elasticity, said to be independent of age and of congestion, have been described by various authors.

M. Villemin thinks that the true anatomical structure of the walls of the air-vesicles is a network of capillary vessels, with a nucleus filling each intercapillary mesh, and elastic fibres on the inside of the vesicles crossing over the capillaries and intercapillary nuclei. "In Pulmonary Vesicular Emphysema," M. Villemin says, "the nuclei in the meshes of the capillary network hypertrophy, compression and atrophy of the capillaries follow; then the enlarged nuclei undergo fatty degeneration; they fall from their places in the walls of the air-vesicles, destruction of the elastic tissue and of more capillaries occurs; apertures are formed between adjacent vesicles, and finally, two or more vesicles are thrown into one."

"There is then," M. Villemin says, "a first stage of Emphysema, a true hypertrophy of the elements of the vesicular membrane; from this there naturally results an extension of that membrane, and an increase in the capacity of the vesicles."

It does not, however, necessarily follow, even though M. Villemin's anatomical observations be correct, that there is an increase in the size of the lung, as he supposes, because the walls of the air-vesicles are lengthened; for they might, under such circumstances, be folded on themselves. Moreover, the accuracy of these observations has been doubted. The so-called intercapillary nuclei are said by some observers to be epithelium on the inside of the air-vesicles.

"Changes in the nutrition of the lung," Freund says, "necessarily follow on the changed conditions of the respiratory movements due to the lengthening of the rib-cartilages, and these changes are attended by loss of elasticity, and the other changes in the walls of the air-vesicles which follow on their continued over-distension."

Dr. Waters while admitting that his investigations do not enable him to say what is the nature of the degeneration which leads to Emphysema, and that his microscopical researches on this point have yielded no results, adds, "I do not entertain the slightest doubt that the disease in its severer forms is of a constitutional nature."

Varieties of Pulmonary Vesicular Emphysema.—As over-distension of the air-vesicles may occur in perfectly healthy lungs, and in lungs the seat of any of those pathological changes which impair their elasticity, and as, moreover, the dilatation may affect the air-vesicles of the whole, or of a great part of the lung, or may be limited to the air-cells of a small part of the lung, Pulmonary Vesicular Emphysema has been divided into varieties.

The various forms of Pulmonary Vesicular Emphysema may be described under the four following heads:—

Acute Vesicular Emphysema.

Chronic Local Emphysema.

Large-lunged (or Hypertrophous) Emphysema.

Small lunged (or Atrophous) Emphysema.

Although perfect and uncomplicated specimens of each variety are common, cases of Pulmonary Vesicular Emphysema are frequently seen in practice and in the dead-house, in which these several varieties are conjoined in the same lung, and, again, cases which cannot at the time when they come under observation be referred absolutely to the one or the other group. The reasons for this are manifest when the etiology and the pathology of the affection are considered.

ACUTE VESICULAR EMPHYSEMA.—By Acute Pulmonary Vesicular Emphysema is signified acute over-distension of previously healthy air-vesicles.

The part of the lung, the air-vesicle of which are over-distended, is puffed up, is paler than it should be; the vesicles themselves, seen through the pleura, are manifestly larger than natural. The pallor is due solely to the excess of air in the vesicles stretching their walls, and so separating the capillaries further than should be from each other. The meshes of the capillary network on the walls of the air-vesicles are widened. Acute Vesicular Emphysema may be produced by too much air being drawn into the over-distended air-vesicles by inspiratory effort; or by too much air being driven into the air-cells of parts of the lungs by extreme compression of other parts by expiratory efforts, while the escape of the air by the natural outlet is prevented or retarded, *e. g.*, by closed glottis, narrowing of the trachea or bronchi.

Both these forces conspire to determine the occurrence of acute over-distension of the air-vesicles in acute bronchitis. In that disease disseminated collapse, and the consequent diminished bulk of lung

and increased desire for breath, lead to violent inspiratory efforts and over-distension of the pervious air-vesicles; while the frequent and violent expiratory efforts with closed glottis (preparatory to cough), determine over-distension of the air-vesicles of the less compressed and less supported parts of the lung.

When the ribs are greatly softened, as in some cases of rickets, the anterior margin of the lungs is the seat of Acute Vesicular Emphysema. The over-distension of the air-vesicles is produced partly by the compression of the lung at a little distance from its margin by the recession during inspiration of the ribs at their junction with their cartilages, but chiefly by the great advance of the sternum and rib-cartilages during inspiration, these parts being thrust forward by the impressing ribs.

In Acute Pulmonary Vesicular Emphysema, the rule is that the air-vesicles resume their normal size as soon as, or very soon after, the over-distending force ceases to act. The walls of the air-vesicles and the adjacent tissues being healthy, they contract to their normal dimensions.

In comparatively rare cases, the over-distension is so great, so long-continued, or so frequently repeated, that the over-stretched walls of air-vesicles are injured, their elasticity is impaired, and the air-vesicles continue permanently larger than they should be.

It is in this way that severe and prolonged hooping-cough in children appears to produce Chronic Pulmonary Vesicular Emphysema. The over-distension especially affects the air-vesicles of the apex and anterior margin of the lungs, the air being forced into those parts during the violent expiratory efforts which precede the cough.

Symptoms.—If widely spread, and extreme, Acute Pulmonary Vesicular Emphysema causes increased resonance of the chest; the symptoms due to the lesion are masked and altogether lost in those proper to the disease to which it is secondary.

It requires no special treatment.

CHRONIC LOCAL EMPHYSEMA is characterized by extreme permanent over-distension of a few vesicles. The large vesicles are formed by the coalescence of several smaller. The largest may be as large as a poulet's egg, are not unfrequently the size of hazel-nuts, though more commonly not larger than peas. In the same group, vesicles are often found varying in size from a pin's head to a hazel-nut.

The walls of these large vesicles are never healthy; they are thick, opaque, wanting in elasticity, and vessels of some size frequently ramify on the larger. Threads composed of obliterated bronchi, the remains of vessels or of lung tissue, cross the cavity of the larger vesicles. Sometimes these vesicles communicate with a small bronchus; at others the bronchus leading to them is occluded.

The most common seat of Chronic Local Emphysema is the apex of the lung, then the anterior margin, and the margin of the base of the lung. At the apex, the Emphysema is often conjoined with the remains of old tubercle.

The pathology and mechanism of the production of Chronic Local Emphysema is best studied as it occurs at the apex of the lung, when that part is the seat of obsolescent or of calcified tubercle.

When tubercles obsolesce or calcify at the apex of the lung, a considerable portion of lung-tissue in their vicinity is usually the seat of chronic congestion and exudation of lymph. This portion of lung loses its porosity, becomes tough, inelastic, and puckered, *i. e.*, irregularly contracted. Here and there, however, portions of the lung-textures are damaged, not destroyed, so that some air-vesicles still admit air.

In health the inspiratory and expiratory force are at a minimum at the apex. But during expiratory efforts with closed glottis, as in severe cough, the air is driven from the more compressed parts into the little compressed apex, and thus the vesicles still pervious to air are over-distended; and, as their walls have, from the previous changes in the tissues of the apex of the lung, lost much of their elasticity, the over-distension is permanent. Every paroxysm of cough must add to their dilatation. The diminution in size of the apex assists, as a permissive cause, in the production of extreme Chronic Local Emphysema at the apex.

Thus in proportion to the loss in the elasticity of the air-admitting textures, to the frequency and the violence of the expiratory efforts with closed glottis, and to the permanent diminution in the size of the apex, will be the degree and the rapidity with which Local Emphysema at that part will be established.

The anterior margin of the lung, the margin of the base, the anterior inferior angle of the superior lobe of the left lung, are, like the apex, very imperfectly compressed and supported during expiratory efforts, and so air is forced powerfully into the vesicles of those parts; and should the texture there be damaged at any time so as to diminish its elasticity, the result will be great dilatation of a few vesicles. The margins of the lungs are thus sometimes fringed with large vesicles.

Chronic Local Emphysema is always a secondary lesion. Its formation at the apex is the consequence, not the cause (as some have fancied) of the obsolescence of tubercles. Coincident with the obsoles-

cence is damage to the air-admitting textures of the lung, and it is that damage which renders the Chronic Local Emphysema with large vesicles possible.

Symptoms.—The development of Emphysema at the apex of the lung, when that part is the seat of chronic consolidation with contraction, diminishes the depression of the shoulder, and of supra- and infra-clavicular regions, and increases the resonance of the same parts; the dilated vesicles often projecting above and surrounding the solid textures. The dilatation of the vesicles may be so extensive and considerable as to cause supra-clavicular bulging either permanently or during cough.[1] It is unattended by other symptoms.

Treatment.—From the nature of the lesion, it will be understood that no treatment is required.

LARGE-LUNGED VESICULAR EMPHYSEMA.—By this name it is proposed to designate those cases in which there is over-distension of the air-vesicles of the whole, or of a large section of one or of both lungs, great increase in bulk of the lungs, or of the affected part of the lungs, and corresponding increase, local or general, in the capacity of the thorax. The term Hypertrophous Pulmonary Vesicular Emphysema has been used to describe the same set of cases.[2]

General Large-lunged Vesicular Emphysema is a very serious disease. The symptoms directly due to it are grave; the diseased conditions dependent on it for their origin are very frequently fatal.

Thus a large proportion of cases of heart disease have their starting-point in Large-lunged Vesicular Emphysema.

It rarely occurs in a marked form before the middle of life, and it more commonly affects those disposed to accumulation of fat in the subcutaneous tissue and internal parts. Lungs, the subject of this form of Vesicular Emphysema, are larger and drier than healthy lungs.[1]

The parts uncolored by pigment are paler than healthy lung.

The lungs overlap the pericardium to a considerable extent, and meet above it even to near the top of the sternum; they have a down-cushion-like feel, and retain the impression of the fingers. When the thorax is opened they contract less than healthy lungs do under like circumstances.

Large-lunged Vesicular Emphysema is, in the great majority of cases, preceded by attacks of bronchitis, by congestion of the lungs, by dry winter cough, or by chronic bronchitis; that is to say, by diseases having as immediate consequences toughening and thickening of the tissues of the lung,[2] and severe cough; in other words, diminished elasticity of the lungs, and powerful expiratory efforts with closed glottis.

By far the most common determining cause then of the over-distension of the air-vesicles in Large-lunged Vesicular Emphysema is powerful expiratory effort with closed glottis; and the most common permanence-securing cause is the changes in the texture of the walls of the air-vesicles resulting from excess of blood in their capillaries.

The next most frequent determining and permanence-securing causes of Large-lunged Vesicular Emphysema are diminished ordinary or habitual expiratory force, dependent on age-degeneration of the bones and cartilages in the thoracic parietes, without loss of full muscular inspiratory power, occurring alone, or more commonly conjoined with thickening of

[1] The bulging part is resonant and cannot, therefore, be confounded by a tolerably careful observer with the prominence of the same part due to distension of the veins during severe cough.

[2] Large-lunged is by far the better of the two names, because it involves the expression of no opinion in regard of disputed facts. Many observers regard Pulmonary Vesicular Emphysema as atrophic from its outset—no matter how it originates. And it must be admitted that even when the disease has been hypertrophic when first established, the lungs may be greatly wasted in regard of their essential anatomical constituents before death. And again, in some cases of Large-lunged Vesicular Emphysema, as in those in which the occurrence of the disease is determined, and its continuance secured, by increase in the capacity of the chest from age-degeneration of the ribs and cartilages without diminution in the power of the respiratory muscles, the wasting and rarefaction may not be preceded by hypertrophy of any anatomical constituent of the lung.

[1] When Vesicular Emphysema follows on bronchitis, congestion of the lungs, and similar pathological conditions, the lungs, at the very outset of the disease, weigh more than in health, and would continue to do so were it not for the waste of the normal anatomical constituents of the lung—blood, bloodvessels—epithelium, or intercapillary nuclei—which follows on over-distension of the air-vesicles, and on the lesions which secure the permanence of their over-distension.

[2] To comprehend the relation between bronchitis, the changes following it in the walls of the air-vesicles, and the frequency with which bronchitis supervenes on Pulmonary Vesicular Emphysema, it must be remembered that the blood of the bronchial arteries is returned to the heart chiefly through the pulmonary veins, and that many good observers affirm that the bronchial mucous membrane is in great measure nourished by the blood of the pulmonary artery, and that anastomoses exist between the finest divisions of the bronchial and pulmonary arteries.

the tissues and diminished elasticity of the lungs—changes also due to age-degeneration.

As bronchitis, winter cough, and congestion of the lungs are common at advanced periods of life, *i. e.*, at the period of life when, without loss of muscular inspiratory power, age-degeneration of the bones and cartilages of the thorax and of the lungs is common, it is manifest that violent expiratory efforts with closed glottis, habitually defective expiration, and therefore excessive inspiratory dilatation, changes in the lung due to congestion, and changes in the lung-tissue due to age-degeneration, must in a very large number of cases conspire to produce Large-lunged Vesicular Emphysema.

Fig. 12.

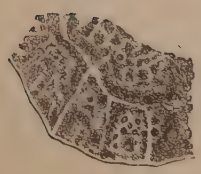
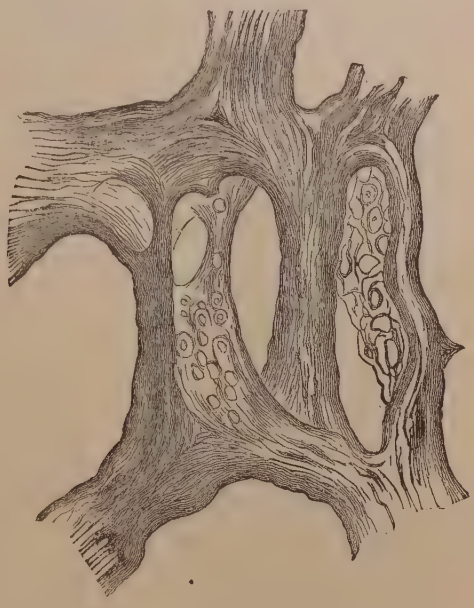


Fig. 12 shows increase in the thickness of the walls of the air-cells. Magnified two diameters.

Fig. 13.



A portion of the above magnified 400 diameters.

The changes which occur in the texture of the lung, in consequence of continued congestion, have been admirably described and figured by Rokitansky.

When the lung is congested, as from disease of the left side of the heart, an increase in the quantity of the connective tissue occurs, the walls of the air-vesicles are thickened, the parenchyma appears thicker and swollen and unusually resistant.

On section of the lung, the margins of the lung-vesicles are thicker than in health, and the cavity of each vesicle more visible than it should be, because its thickened walls prevent collapse. Sometimes the cavity of each vesicle is increased, and the lungs are larger than they should be; in other words, the substance of the lung is toughened and thickened from the formation of tissue, and enlargement of the lung, with dilatation of the vesicles, follows when any of the determining causes of over-distension of the air-vesicles come into action.

But, however produced, permanent over-distension of the air-vesicles is followed by various pathological changes in their walls. Some of these changes are the direct mechanical result of their over-distension; some are the result of degenerative changes in the structures thus mechanically injured; some of defective nutrition consequent on the injury inflicted on the capillaries of the walls by their stretching; some of altered nutrition due to the alterations in structure; some are due to the pathological states to which the permanence of the over-distension is owing. So that when the disease is far advanced, and has existed for some time, not only are individual air-cells enlarged, but the partitions between many are perforated; between others they are reduced to mere ridges; at places they have altogether disappeared; and at places they are greatly, though it may be irregularly, thickened by imperfectly constituted fibrous tissue formed in and about the normal structures. And so, ultimately, atrophy of some structures is conjoined with increase in size and thickness of others; and rarefaction and condensation may affect adjacent parts of the same lung.

If a portion of lung in an advanced stage of Vesicular Emphysema be in-

flated, dried, and then cut across, the cut surface appears to be made up of spaces varying in size from a millet-seed to a hemp-seed, while near to the apex and margin of the lungs may be a few much larger spaces. These spaces or small cavities are separated and intersected by septa and by threads of very variable degrees of thickness.

Fig. 14.

Section of lung in an advanced state of Chronic Vesicular Emphysema. (From Rokitansky, Lehrbuch der Pathol. Anatomie. B. iii. 1861.)

Black pigment accumulates in considerable quantity on the inner surface of the dilated vesicles, and amid the fibrous and other solids. This black pigment owes its origin in part to the conversion of the hæmatin in the partially destroyed capillaries into melanin.

When the whole of both lungs are emphysematous, the changes just described are much more advanced at the margins and apices than they are elsewhere; and, as a rule, they are more advanced at the base of the left than they are at the base of the right lung; these being, in the great majority of cases, the parts first to suffer in Large-lunged Vesicular Emphysema, because they are the parts least compressed and least supported during expiratory efforts with closed glottis.

Effect of over-distension of the air-vesicles on the circulation.—The capillaries of the pulmonary artery distributed on the walls of the air-vesicles are first stretched in proportion to the over-distension of the vesicles, and then, the over-distension continuing, some of the stretched vessels give way and are obliterated.

The passage of blood through the capillaries lengthened by stretching must be attended by increased friction, in proportion to the lengthening and narrowing of the vessels.

Destruction of the capillaries diminishes the number of channels through which the blood can pass, and so impedes, in proportion to the number of capillaries torn, the passage of the blood from the right to the left side of the heart.

Impediment to the flow of blood through the lungs is the cause of the greater number of the primary and secondary symptoms of Large-lunged Vesicular Emphysema.

The several causes of impediment to the flow of blood through the lung and their modes of action are—

1. Deficient extent of chest-movement in ordinary respiration; especially deficient ordinary or habitual expiratory movement:

2. Violent expiratory efforts with closed glottis; by the pressure brought to bear on the heart and great vessels, as well as on the air in the anterior of the air-vesicles, and so on the capillaries in their walls:

3. Diminished resistance from loss of elasticity of the lung; by disturbing the normal proportion borne by the pressure of the air on the inner to that on the outer chest-walls:

4. Lengthening of the pulmonary capillaries; by increasing the friction:

5. Destruction of pulmonary capillaries; by diminishing the channels for the passage of the blood from the pulmonary artery to the pulmonary vein.

As the establishment of an efficient collateral pulmonary circulation is anatomically impossible, any impediment to the flow of blood through all, or nearly all, the pulmonary capillaries, must have as direct result impediment to the escape of blood from the right ventricle.

The first effect of difficulty to the passage of blood through the pulmonary capillaries must be, in accordance with general laws, increased efforts, and so hypertrophy of the walls of the right ventricle; increased pressure on the inside of the right ventricle, and so dilatation of its cavity.

At the outset, the impediment to the onward passage of the blood may at parts of the lung be trifling, compared with the impediment at other parts; in such case these parts suffer from the increased blood-pressure, become hyperæmic, and, it may be, œdematous.

The impediment to its onward passage is soon felt by the blood in the right auricle, and in the whole systemic venous system, of which the right heart is merely the terminus. When the auricle and ventricle are dilated, the right auriculo-ventricular orifice is dilated, and the result of increase in its circumference, without corresponding increase in the size of the tricuspid valve, is incompetence of the valve to close the dilated opening, and regurgitation of blood during the ventricular systole, from the right ventricle to the right auricle, and veins opening into it.

But the impediment to the flow of blood through the pulmonary capillaries is not only followed by over-distension of the venous system, but ultimately the blood passes from the systemic capillaries into the veins with difficulty, and so an impediment arises to the escape of blood from the arteries, and from the left side of the heart, which is merely the head of the general arterial system.

That such impediment to the escape of blood from the arteries does exist when there is strong impediment to the flow of blood through the lungs, is manifested by placing the finger on an artery when a patient suffering from General Pulmonary Vesicular Emphysema coughs violently; the artery instantly becomes full and tense, and, for the second of violent expiratory effort, ceases to pulsate.

Over-filling of the capillaries of an organ or tissue with retardation of the flow of blood through them never continues for any length of time, and is never repeated frequently without inducing changes in the structure of that congested organ or tissue.

The changes of the several organs, resulting from mechanically-induced congestion, are considered at length in the articles on diseases of special organs. Only such changes of special organs as give rise to the more important symptoms in bad cases of Large-lunged Pulmonary Vesicular Emphysema will be here considered.

Speaking generally, if an organ be the seat of mechanically-induced intermitting congestion, the earliest result is increased nutrition and enlargement of the organ. When the dilatation of the capillaries has reached a certain degree and becomes permanent, then wasting of the structures of the part with increase in connective tissue, especially of imperfectly formed connective tissue, may result. The formation of the latter may precede, and greatly preponderate over the wasting of the natural structures of the part.

A large number of free granules, of olein and protein, are found among the proper anatomical elements of the part almost from the very commencement of the congestion; and fatty degeneration of the normal structures frequently precedes their disappearance.

The parts that especially suffer in Large-lunged Vesicular Emphysema are—

The Heart.—First and most certainly, the heart.

The first effect of the impediment to the passage of the blood through the lungs is increase in the muscular tissue of the right side of the heart; then follow accumulation of blood in the ventricle, and some dilatation of its cavity. The right auricle next suffers in the same way, and soon the whole venous system: the veins of the heart suffering over-distension in common with the other veins.

Mechanically-induced congestion of the walls of the heart, with increased action of the organ, leads not only to hypertrophy, but ultimately to induration and toughening of the walls. When these changes have occurred in its muscular tissue, the heart loses its power of close contraction, and permanence of the dilatation produced by the pressure of the blood on its inner surface is the result.

Free granules of olein and protein are found between the muscular fibres; and, after a longer or shorter time, fatty degeneration of the damaged muscular tissue follows.

When distension of the veins has reached a certain point, the blood escapes from the systemic capillaries with difficulty, and increased action of the left ventricle follows. As the walls of the left side of the heart suffer from the same mechanically induced congestion as the walls of the right side, when impediment to the escape of blood from the left ventricle is established, its walls and cavity experience, though in a less degree, the same changes in texture, &c., as the right side of the organ, viz., hypertrophy, induration, toughening, and permanent dilatation.

The Liver.—The radicles of the hepatic vein, then the terminal twigs of the portal vein and finally its radicles, suffer congestion from the same cause as the systemic capillaries, *i. e.*, from the impediment to the escape of blood from the inferior vena cava.

In consequence of the impediment to the circulation, the liver is first enlarged from mere congestion, and in this stage a variety of "nutmeg liver" is found after death.

When the congestion has continued for some time, the organ is more or less enlarged, indurated, and toughened, and free granules of olein and protein infiltrate all its tissues; then its natural structures waste, especially, it is said, the cell element, and a certain amount of granular atrophy is the final result.

Ascites very rarely occurs before the hepatic structure is organically injured,

and rarely to any great amount from those changes only which follow directly from the impediment to the circulation here considered.

The Kidneys, in common with other organs, suffer congestion in cases of extreme Large-lunged Vesicular Emphysema.

This extreme congestion is evidenced during life by the presence of albumen, and sometimes of blood, in the urine.

The kidney suffering from mechanically-induced congestion is at first larger, darker, and moister than in health. Granules of olein and protein are scattered through all its structures. After a time, induration and toughening of the organ follow. A slight amount of granular atrophy of the previously enlarged kidney is the ultimate result.

The Connective or Cellular Tissue throughout the body suffers from its mechanically-induced congestion. Its texture is toughened and thickened, and serosity is effused into its meshes.

Anasarca is one of the earliest consequences of over-filling of the venous system from impediment to the flow of blood through the lungs. The anasarca is frequently attributed to the regurgitation of blood through the right auriculo-ventricular orifice; but both the regurgitation and the anasarca are really due to a common cause, *i. e.*, to the state of the pulmonary capillaries. As a rule, however, before over-distension of the veins is so great as to relieve itself by letting out serosity into the cellular tissue, the pressure on the inside of the right ventricle and auricle is sufficient to dilate the auriculo-ventricular orifice to such an extent that the tricuspid valve is incompetent to its closure, pulsation in the jugulars is perceptible, and the anasarca is then erroneously attributed to a tricuspid regurgitation, as it is often called.

Blood and General Nutrition. — Niemeyer has pointed out that congestion of the venous system from mechanical impediment to the onward flow of blood through the lungs, or right heart, cannot exist without causing impediment to the escape of its contents from the thoracic duct. To this Niemeyer attributes the deficiency of fibrine and of albumen in the blood in cyanosis dependent on mechanically-induced over-filling of the venous system, and to it he also attributes the general emaciation which occurs in advanced cases of Pulmonary Vesicular Emphysema.

The Vessels of the Lungs.—In the last stages of the disease, after the left ventricle has suffered hypertrophy and dilatation, secondary lesions of the lung not unfrequently occur—thus the lungs may become greatly congested, and œdema of the lungs or congestive pneumonia follow. The mechanical impediment to the flow of blood through the pulmonary capillaries has told back through the systemic capillaries on the left side of the heart, and so on the radicles of the pulmonary veins.

Symptoms of Large-lunged Vesicular Emphysema. — The chief direct symptoms of Large-lunged Vesicular Emphysema are:—

(*a*) Increase in the size of the thorax;

(*b*) Increase in the resonance of the thorax, and prolonged expiration;

(*c*) Shortness of breath.

(*a*) The lungs are larger than in health, and the capacity of the thorax is in proportion to the size of the lungs.

The increase in the circumference of the thorax is effected chiefly by diminution in the natural obliquity of the ribs.

By this alteration in the direction of the ribs, the lower intercostal spaces are very considerably widened.

The sternum is carried forward.

The lower latero-dorsal bulging of the thorax is increased.

The enlargement of the circumference of the chest thus gained is made still greater by posterior curvature of the lowest cervical, the dorsal, and upper lumbar part of the spinal column. The patient stoops, he grows round-shouldered and round-backed.[1]

Increase in the capacity of the thorax from above downward is produced by lowering of the diaphragm. At the termination of expiration in extreme cases of Large-lunged Vesicular Emphysema the diaphragm lies very low, so that it is not in contact with the inner surface of even the lowest rib.

When the air-vesicles of the upper half of the lungs are the first to suffer over-distension, or are much more affected than are the air-vesicles of the lower part of the lung, the upper part of the thorax is disproportionately larger. When the determining cause of the over-distension has been violent cough from bronchitis, then the disproportion in size between the upper and lower part of the thorax is sometimes increased by imperfect expansion of the lower part of the lungs; the condition of the bronchial mucous membrane and the contents of the bronchial tubes preventing the free and ready entrance of the air into the air-cells of these parts of the lung.

The increase in the capacity of the thorax is determined by the forces which determine the over-distension of the air-vesicles, viz., by repetition of full inspiratory efforts, expiratory efforts with closed

[1] Whenever the depth of the chest, from before backwards, requires to be increased, *e. g.*, in dilatation of the heart—effusion into the pericardium—the patient instinctively rounds his back and elevates his shoulders.

glottis, and diminished elasticity of the thoracic parietes, or of the lungs, or more commonly of the two conjoined.

(*b*) Of the physical signs, after those furnished by inspection of the thorax, by far the most constant and important in regard of diagnosis is increased resonance on percussion — clear full sound. The abnormal clearness on percussion is due to the relative increase in the quantity of air in the chest, and to the tension of the chest-walls.

As the large lungs overlap the heart, the region of precordial dulness is diminished, and, as the diaphragm is flattened, the hyper-resonance extends posteriorly even to the twelfth rib, and in front often as low as the margin of the thorax, even on the right side, the liver lying altogether under the abdominal parietes.

Expiration is, in extreme cases, considerably prolonged in consequence of the diminution in the resilience of the chest-walls and lungs, and of the large size of the latter. At the same time, the inspiratory murmur is short and feeble. But when this form of Pulmonary Vesicular Emphysema is limited to a part of the lung, the only physical signs are local bulging and hyper-resonance.

(*c*) Shortness of breath is always present in Large-lunged Vesicular Emphysema. At first, the shortness of breath is only felt on exertion; the patient cannot mount a hill as he did. Then, when walking on level ground, he requires to stop, from time to time, to take in breath — he breathes too frequently, and pants a little; or it may be that he "suffers with his breath" after a full or an indigestible meal, when the descent of the diaphragm is impeded by a distended stomach. However the shortness of breath is induced, the subject of Large-lunged Vesicular Emphysema is, from a very early period, conscious that his "wind" is no longer what it was.

As the disease advances, the shortness of breath is experienced on the least exertion, *e. g.*, ascending a few steps, or a gentle slope; and finally, even when sitting on a chair. The patient is always panting.

By the altered position of the ribs and the diaphragm, a considerable increase in the capacity of the thorax is, as has been shown, obtained; but it is obtained at the expense of the inspiratory capability. The chest-walls are constantly expanded, and when the disease is far advanced, the capacity of the chest may be greater at the termination of expiration, than, in the normal condition of the lungs and chest-walls, it should be at the termination of inspiration. As Dr. C. J. B. Williams has tersely expressed it, "Breath is taken as it were on the top of breath."

The lowering of the diaphragm may be so considerable, it is said, as to cause its physiological action to be reversed. In place of increasing the capacity of the thorax by its contraction, the diaphragm may draw, it has been said, the lower ribs inwards, and so diminish to a slight extent the capacity of the lower part of the chest at the end of inspiration.

The diaphragm may be forced downwards by the expiratory efforts, which determine over-distension of the air-cells, but it probably never lies very low till the elasticity of the lungs is considerably impaired.

The great natural agent in effecting the ascent of the diaphragm, after it has been lowered by its own contraction, is the elasticity of the lungs. The muscles relax at the termination of inspiration, and the diminution in the size of the lungs resulting from their resilience greatly aids in determining the passive ascent of the diaphragm. When the lungs, from loss of elasticity, no longer diminish in size as much as they should, at the termination of inspiration, the ascent of the diaphragm is less than it should be, and it begins to act at the commencement of each inspiration from a lower and lower level; consequently, the increase in the capacity of the thorax obtainable by its contraction is always lessening, till finally, it is perhaps just possible that its normal physiological action may be, as above stated, reversed.[1] When the ordinary muscles of inspiration are, in consequence of the permanent expansion of the chest, unable to dilate it sufficiently to take in a proper supply of air, all the extraordinary muscles of inspiration are habitually employed in breathing; hence the muscles of the neck, back, &c., capable of aiding inspiration, are, after a time, considerably hypertrophied, the shoulders are raised, and the enlargements of the muscles of the neck, the scaleni especially, give a peculiar breadth to the neck.

Imperfect aëration of the blood resulting from the damaged state of the pulmonary capillaries, and the changes which take place in the walls of the air-vesicles

[1] The common cause of recession of the lower part of the chest during inspiration is some impediment to the free entrance of the air into the lungs, and the pressure of the external air for this reason being brought to bear with undue force on the outside of the thorax by the powerful action of the inspiratory muscles. The lower parts of the chest-walls are there most yielding, and are therefore pressed inwards by the weight of the atmosphere. On this and other points connected with the deformity of the chest in Pulmonary Vesicular Emphysema, the reader is referred to Dr. Sibson's elaborate and most able paper in the thirty-first volume (1848) of the Medico-Chir. Soc. Trans., "On the Movements of Respiration in Disease."

after they have been long over-distended, add greatly to the shortness of breath; while the dilution of the air taken in at each inspiration by the large quantity of residual air left after expiration, must still farther distress the breathing by interfering with aëration of the blood.

The shortness of breath, then, in uncomplicated Large-lunged Vesicular Emphysema, is due to the small extent of movement of the chest-walls, including the diaphragm, during respiration, to the impurity of the air in the thorax at the termination of inspiration,[1] to the state of the capillaries of the pulmonary artery, and to the structural changes in the substance of the walls of the air-vesicles.

If bronchitis in any form, or asthma, supervene, the distress of breathing is greatly increased; and in some cases in which the distress of breathing has been unusually great, fatty degeneration of the heart has been found after death (Virchow).

General description of the symptoms in a case of advanced Large-lunged Vesicular Emphysema. — The thorax is barrel-shaped; the antero-posterior, lateral, and vertical diameters are increased; the sternum is arched; the lower cervical, dorsal, and upper lumbar spine is curved, concavity forward; the ribs are too horizontal; the intercostal spaces are widened, and but little, if at all, depressed below the level of the ribs; the posterior bulgings on either side of the vertebral column are greater than they should be; the costal angle is larger than in health, and as the diaphragm is flattened and the lower part of the sternum is forced forward, at the same time that both lungs are enlarged, the heart is at once less covered than in health by the sternum, thrust downwards by the forces that over-distended the air-vesicles, and carried downwards by the contraction of the diaphragm, and can, in consequence, be felt and seen beating below the ensiform cartilage. The heart, and especially the right ventricle, is dilated, and hypertrophied—its impulse is heaving, and its dilatation and hypertrophy render the epigastric pulsation very perceptible.

The shoulders are raised, and the muscles of the neck and shoulders, especially the sterno-cleido-mastoidei, the scaleni, the omo-hyoid, and the trapezii, stand prominently out.

The fossa behind the clavicle is frequently deepened; when, however, there is excess of emphysema above the level of the first rib, there may be post clavicular bulging. Under all circumstances, when the patient coughs there is undue prominence, or bulging even of the post-clavicular fossa, and of the intercostal spaces, the air being forced from the more to the less compressed and supported parts by the expiratory efforts preceding the opening of the glottis.

The neck is broad from hypertrophy of its muscles, and its veins are unduly prominent. As the obstruction to the circulation increases, the veins of the neck pulsate synchronously with the beat of the right ventricle, and fill from below when emptied by the pressure of the finger. The whole venous system is manifestly dilated, the larger veins of the upper extremities have a knotted appearance from over-distension just above their valves, the hemorrhoidal veins are enlarged, thickened, &c., and often bleed—it may be to the great relief of many of the discomforts from which the patient is suffering. The face has a coarse, bloated, dusky, and, on exertion, even livid aspect; the alæ of the nose, and the lips, especially the lower lip, are thickened. The eyes are prominent, the conjunctivæ injected, occasionally yellowish, and the eyelids puffy—drowsiness, mental dulness, and headache are common symptoms. Emaciation is sometimes very considerable. The legs are œdematous, or the whole cellular tissue the seat of anasarca. Orthopnœa is often present, because in the recumbent position the extraordinary muscles of inspiration can have only imperfectly supported points, in place of fixed points, from which to act; and again, because the weight of the body in the recumbent position interferes with the expansion of some part of the chest-walls, and the position and weight of the abdominal viscera with the descent of the diaphragm.

In this stage of the disease the urine frequently contains albumen, and now and then blood and blood-casts of tubes.

The abdomen generally is fuller than natural. The spleen and liver are increased in size, and the latter organ is frequently so much depressed by the determining cause of the distended lungs, by the enlarged and distended heart, and by the flattened diaphragm, that its upper convex surface can be distinguished by eye and touch through the abdominal walls.

When from supervention of bronchitis, or other cause, the impediment to the

[1] Although the capacity of the chest is greater in Large-lunged Vesicular Emphysema than it is in health, spirometrical observations show that its vital capacity, as measured by the quantity of air that can be expelled after deep inspiration, is diminished. The residual air must therefore be much greater than it should be. Speaking of the difficulty of breathing in Emphysema, Magendie (Leçons, 1825, tome i. p. 169) observes: "The tissue of the lung has lost some of its elasticity, and no longer reacts with sufficient force on the air which has penetrated into its parenchyma."

pulmonary capillary circulation is temporarily increased, the liver and spleen may be proved, by percussion and touch, to be larger than before, and to resume their former size, as the circulation through the lungs becomes freer, and the mechanically-induced congestion is in consequence lessened.

The pulse in Large-lunged Emphysema is often small and weak, from the small quantities of blood which pass through the lungs and therefore into the left ventricle.

The urine is, speaking generally, that of imperfect respiration, and of congestion of the kidneys and liver. At times it is very abundant, pale, clear, and of low specific gravity; at others it is scanty, high-colored, and loaded with lithates, which as the urine cools form a heavy brickdust-like sediment. This deposit is not in all cases due merely to the concentration of the urine, there may be an absolute increase in the quantity of uric acid. Owing to the imperfect aëration of the blood there is a scanty supply of oxygen distributed through the system, hence the products of tissue metamorphosis are in a lower stage of oxidation, and uric acid is formed to some extent in place of urea. Parkes thinks it is only when bronchitis is superadded to Emphysema that there is such deficient oxidation as to lead to excess of uric acid in place of urea. J. C. Lehmann, in a carefully-observed case, found the urine after each attack of difficulty of breath deficient in urea and very acid. It contained oxalic acid and allantoin. To this Parkes objects, that in Lehmann's case bronchitis complicated the Emphysema, and refers to a case of uncomplicated Emphysema so severe as to cause cyanosis and constant dyspnœa, observed by Ranke and himself, in which very little uric acid and a full quantity of urea were present in the urine. Biemer says, after quoting the observations of Lehmann and Ranke, that he has more often been able to detect small quantities of bile pigment in the urine.

A trace of albumen may, when the disease is far advanced, be constantly present in the urine; the quantity being increased with every increase of the impediment to the flow of blood through the lungs. When the congestion of the kidneys is suddenly greatly increased, or attains, even slowly, an extreme degree, the urine contains blood and blood-casts of tubes. Much albumen, with little evidence of impediment to the flow of blood through the lungs, renders it probable that organic disease of the kidney is present.

It is not uncommon for the symptoms to be very trifling for some years, and then for a year or more, to see the graver symptoms only when the patient has an attack of bronchitis; with the cessation of the bronchitis the œdema of the legs, the albumen in the urine, and the jugular pulsation frequently disappear. The over-distended heart and veins having their walls, as yet, to any serious degree, undamaged, contract nearly to their normal dimensions, when the extra impediment to the flow of blood through the lungs, due to the acute attack, has passed away. But the improvement is only for a time, another attack of bronchitis renews the serious symptoms, and after one or more such renewals, they are permanently established.

The variations in severity of the chief symptoms of Large-lunged Vesicular Emphysema may be summed up thus: the increase in size of the thorax varies from that obtained by a slight diminution in the natural obliquity of the ribs, or trifling local bulging, to the utmost expansion of the chest-walls: the hyper-resonance on percussion, from slightly increased clearness to the fullest clear sound; the prolongation of expiration, from an amount difficult to appreciate, to that in which it considerably exceeds in length the inspiratory sound. The impediment to the flow of blood through the lungs varies, from just enough to give, when the patient coughs, undue prominence to the great veins of the neck, to sufficient to cause hypertrophy and dilatation of the right side of the heart, jugular pulsation, and knotting and enlargement of all the superficial veins, anasarca, albuminuria, enlargement of the liver and spleen, dilatation of the systemic capillaries and arteries, hypertrophy and dilatation of the left side of the heart, and finally organic changes in the structure of all the organs in the body, and of the connective tissue generally. The shortness of breath varies from a mere "touch in the wind" to inability to move without great distress of breathing.

The imperfect aëration of the blood varies from just sufficient to cause a slightly dusky hue of the lips on exertion, to enough to give the patient the purple or leaden hue of cyanosis.

When Large-lunged Vesicular Emphysema is limited to a lobe or part of a lobe, as not unfrequently happens, then local fulness or bulging, and hyper-resonance with trifling shortness of breath, are commonly the only evidences of the disease.

Small-lunged (or Atrophous Pulmonary) Vesicular Emphysema.—After Large-lunged Vesicular Emphysema has lasted some time, and the over-distension of the vesicles is extreme, a certain, it may be considerable, amount of wasting of the tissues of the lung ensues; and thus a form of Atrophous Emphysema is established.

But in the variety of Emphysema designated Small-lunged or Atrophous Pulmonary Vesicular Emphysema, atrophy of the lung-tissue is the primary disease, or it supervenes on trifling primary over-distension.

Small-lunged Vesicular Emphysema is confined to persons well past middle-life. Those who suffer from it are commonly thin. Withered-looking, shrivelled, old persons frequently have their lungs damaged by this form of Emphysema. It is a far less troublesome and less grave affection than is Large-lunged Vesicular Emphysema.

In primary general Small-lunged Vesicular Emphysema the whole of both lungs suffer. There is waste of tissue, true atrophy. In some cases fatty degeneration has been said to precede absorption, or the final disappearance of tissue.

In this form of Emphysema the separate vesicles are not dilated; but the partitions between adjacent vesicles with their pulmonary capillaries and other structures disappear or are reduced to mere threads, and two or more vesicles are thus thrown into one. No over-distending force is necessary to determine the increased capacity of the vesicles.

Lungs the subject of this disease are smaller, lighter, and drier than are healthy lungs. They would, from the destruction of the capillaries, be pale, but the pallor from this cause is commonly concealed by the large amount of black pigment spread through them. They weigh much less than healthy lungs, because they have lost much of their natural structures. The air-vesicles are large, but the lungs themselves are small.

The division between the superior and inferior lobe is more vertical than in health. The elasticity of the lungs is in a great measure lost—there is no resilience in them—they pit on pressure, and the pit remains. Their small size, their lightness, and the *very* small space into which they may be compressed are often most remarkable. They are occasionally so much wasted, that, on opening the thorax, they sink back at once toward the spine and posterior part of the thorax. When the lungs and air-passages are in health, death takes place at the termination of expiration. On opening the thorax, healthy at the moment of death, and permitting the pressure of the air to bear on the outer surface of the lungs, there is at once a slight diminution in their size. This diminution is due to the elasticity of the lungs. Before the opening of the thorax the complete elastic contraction of the lungs was opposed by the pressure of the air on the inner surface of the air-vesicles.

In Hypertrophous Pulmonary Vesicular Emphysema the resilience of the lungs is diminished, hence when the thorax is opened there is less contraction of the lungs, and therefore less separation of the lungs from the chest-walls, than there is when the lungs and air-passages are healthy. The quantity of solid tissue constituting the walls of the air-vesicles, &c., and the irregular thickening of that tissue, prevent any mere collapse of the lungs.

When the lungs are in a state of extreme Atrophous Vesicular Emphysema, they have not only lost in a great measure their elasticity, but a large quantity of their solid tissues has disappeared. The consequence is that, when the thorax is opened, and the pressure of the air on the internal and external surfaces of the lung is equalized, although little or no diminution may occur from its resilience, the weight of the lung may be sufficient to cause it to fall in like an inflated bag of wet paper.

If the subject of extreme Atrophous Pulmonary Vesicular Emphysema suffer from cough, then Local Emphysema with large vesicles is frequently superadded to the General Atrophous Emphysema. The elasticity of the lungs being diminished, the vesicles of the parts least compressed and least supported during expiratory efforts being permanently and greatly over-distended, atrophy of their walls throws several air-vesicles into one, and air being forced into the large cells so formed may lead to their extreme dilatation. For reasons previously assigned, these vesicles are found at the margins and apex of the lung.

Coincidently with the occurrence of the changes in the textures of the lungs which constitute Atrophous Vesicular Emphysema, the ribs and their cartilages experience degenerative changes by which their elasticity is diminished, but at the same time also the inspiratory muscles shrink and lose strength.

The chief direct symptoms of Small-lunged or Atrophous Pulmonary Vesicular Emphysema are—(1) shortness of breath; (2) diminution in the size of the thorax.

Shortness of breath in Small-lunged Emphysema is never felt to any notable degree, unless the patient makes exertion, and as the disease usually occurs in the aged, or in those wasted from other chronic diseases, persons indisposed and incapable of moving quickly, exertion sufficient to cause distressing shortness of breath is rarely made.

Primary Atrophous Vesicular Emphysema is commonly attended by general waste, and is therefore accompanied by waste of blood as well as of tissues; so that the capillary pulmonary vessels, although reduced in number, still suffice for the passage of the diminished quantity of blood.

Again, the lungs being small, the expiratory power is enough to drive out the air, and the play for inspiration is considerable. The patient, in place of, as in Large-lunged Vesicular Emphysema, always "taking in breath on the top of breath," in Small-lunged Vesicular Emphysema inspires from the bottom of his breath.

The chest in Small-lunged Vesicular Emphysema is diminished in capacity and all its diameters are less than in health. The diminution in the antero-posterior and lateral diameters is obtained by a great increase in the obliquity of the ribs. The upper intercostal spaces next the sternum are widened and depressed below the level of the ribs. So obliquely placed are the lowest ribs, that their cartilages almost reach the crest of the ilium, and the intercostal spaces are lost, the ribs themselves really touching. The cartilages between the ribs and sternum, as the ribs become abnormally oblique, bend so as to form an obtuse angle. Respiration is short, the thorax moves as a whole in inspiration, and the expiratory recoil follows quickly. The inspiratory murmur is short and feeble—the expiratory not prolonged. From the loss of elasticity in the ribs and cartilages, and the imperfect tension of the chest-walls, the resonance on percussion may be even less than in health, although the solids in the chest are diminished. As the lungs are small, the heart is less covered than it should be, and so the extent of precordial dulness may be increased, and that, although the heart itself may be partaking of the general atrophy. As Atrophous Emphysema is usually accompanied by waste of blood, and as the general muscular power of the patient forbids active exercise, there is commonly in Small-lunged Emphysema not only little distress in breathing, but no damming back of the blood in the right ventricle, over-distension of the venous system, dropsy, or hypertrophy, or dilatation of the heart.

Complications of Pulmonary Vesicular Emphysema.—The frequent conjunction of bronchitis and Pulmonary Vesicular Emphysema has been admitted from the time the latter was recognized as a special disease. Laennec considered the Emphysema to be in all cases the consequence of bronchitis, and especially of that form of bronchitis which was designated dry catarrh. And Louis, while denying the relation of the two diseases as cause and effect, admitted the frequency of their co-existence.

It is now established that bronchitis is the most common cause of the direct production of Pulmonary Vesicular Emphysema, and also that Emphysema may be developed independently of bronchitis; and that when so established, the subjects of Emphysema are very prone to suffer from bronchitis. So that although, as a rule, bronchitis is the cause of Pulmonary Vesicular Emphysema, it may be the consequence; and the Emphysema, in rare cases, may be unaccompanied from first to last by bronchitis.

One cause of the frequency with which bronchitis supervenes on Pulmonary Vesicular Emphysema is, that when a part of the lung only is emphysematous, and the passage of the blood through the capillaries of that part is consequently impeded, hyperæmia of the non-emphysematous tissues is the necessary result—the blood passing into the vessels of the part which offer it the least resistance.

Chronic general catarrhal bronchitis, with much violent cough and little expectoration, is the most common complication of general Large-lunged Vesicular Emphysema. Acute capillary bronchitis is one of the most common causes of death in the same form of the disease. If the heart be, as it so often is, dilated and hypertrophied, then the acute capillary bronchitis is usually attended with much congestion of the substance of the lung.

Chronic bronchitis with profuse purulent expectoration is less common in Large-lunged than it is in Atrophous Pulmonary Vesicular Emphysema. When the purulent discharge is considerable, the so-called amyloid degeneration of various organs is said to occur pretty frequently. In this case, amyloid degeneration is connected with the profuse suppuration and not with the Pulmonary Vesicular Emphysema.

Dilatation of the bronchial tubes is common in all forms of Pulmonary Vesicular Emphysema.

In severe Large-lunged Vesicular Emphysema, it is common to find an excess of fluid in the pericardium after death. It is only when congestion and œdema of the lungs have complicated the disease that an excess of serosity is found in the pleuræ.

The subjects of Large-lunged Vesicular Emphysema frequently suffer from disturbance of the digestive organs. The liver is loaded with blood, and the bile formed is thick and dark. The walls of the stomach are congested, and ultimately thickened. The result is notable disturbance of the stomach digestive processes. The patient suffers from visible distension of the epigastric region, and also from a sense of weight and fulness in the same part, especially after food, altogether disproportionate to the quantity and the quality of the food taken. Flatulence and acidity of stomach are troublesome symptoms.

The distension of the stomach is frequently so great as very decidedly to intensify, by the impediment it offers to the descent of the diaphragm, the habitual

dyspnœa. The derangement of the stomach is also a not infrequent exciting cause of asthma.

Spasmodic asthma is a very common complication of Pulmonary Vesicular Emphysema, especially of the large-lunged form of the disease. Attacks of extreme difficulty of inspiration supervene suddenly in the early morning, or at uncertain times, on the habitual shortness of breath. In addition to attacks of ordinary spasmodic asthma, the subjects of Pulmonary Vesicular Emphysema often suffer for weeks together from increased dyspnœa, out of all proportion to any catarrhal bronchitis which may happen to be present. This dyspnœa is, in a great measure at least, due to spasm of the bronchial muscular fibres.[1]

Phthisis.—One of the most marked anatomical characters of congenital tuberculosis, *i. e.* of an inherited disposition to the deposit of tubercle, is the small size of the lungs. It is by no means uncommon for a deposit of tubercle to take place in the apex of such lungs, and then for the tubercle to obsolesce or to calcify. The subjects of these changes frequently live to an advanced age. Chronic solidification with contraction of the apex of the lung, much black pigment in the solid tissue, and Local Emphysema with large vesicles, frequently follow the obsolescence or calcification of the tubercles.

After passing middle life, the subjects of these lesions frequently become affected with general Atrophous Pulmonary Vesicular Emphysema, rarely, if ever, with Large-lunged Vesicular Emphysema. Subsequently, the solid tissue of the apex of the lung may undergo molecular disintegration, and a cavity be formed; death, in such cases, is said to have occurred from tubercular consumption, when, indeed, there are no tubercles present. This is a form of senile phthisis.

Headache and drowsiness are common consequences of Large-lunged Vesicular Emphysema. These symptoms are due to congestion, the consequence especially of the mechanical impediment to the passage of blood through the lungs, aided in some cases by the imperfect aëration of the blood, and by those changes in the coats of the vessels so commonly seen in the subjects of the disease here considered. The veins and arteries, from changes in their walls, partly due to the congestion of their *vasa vasorum*, lose some of their normal reactive force, and hence the pressure of the blood on the inside of their walls leads here, as elsewhere, to their permanent dilatation. After death, we find not only increased capillary vascularity and excess of serosity in the meshes of the pia mater, but the arteries and veins in the same structure manifestly larger than they should be.

Diseases which in their progress are frequently complicated with Pulmonary Vesicular Emphysema.—All diseases accompanied by severe cough, by diminution of the whole or part of a lung, or by impediment to expiration, are accompanied by over-distension of the air-vesicles.

All diseases or changes in nutrition attended by damage to the elasticity of the lung, render permanent what would otherwise be transient over-distension of the air-vesicles.

Winter cough, catarrhal, and other forms of bronchitis, are undoubtedly the diseases most frequently conjoined with Pulmonary Emphysema.

Disease of the left side of the heart, by damming back the blood in the lungs, and so inducing changes in their texture, leads to permanent dilatation of the air-vesicles when their over-distension has been once determined by cough, &c.

Pneumonia is sometimes attended by Acute Vesicular Emphysema of the air-admitting vesicles, but as the walls of the over-distended vesicles are healthy, and their over-distension is neither extreme nor of long duration, they return to their natural size when the pneumonia subsides. After solidification has passed away, the textures of the lung damaged by the pneumonic exudation may be the seat of permanent Chronic Emphysema.

When one pleura contains sufficient fluid to render the lung altogether useless, the opposite side of the thorax expands more than it should, and is for the time larger than in health, and its air-vesicles are enlarged in proportion to the degree of the expansion of the side. Should the impervious lung be, from long compression or other cause, so damaged as never again to admit any quantity of air into its vesicles, then permanent over-distension of the vesicles of the opposite lung is the consequence. It rarely happens that the distension of the air-vesicles in this case is sufficient to interfere with the capillary circulation on their walls, or to induce atrophy or other serious changes of the vesicular septa.

In chronic phthisis, the bases of the lungs very frequently suffer from vesicular emphysema; this is especially likely to happen when there has previously been dry pleurisy with adhesions at the same point. Hyper-resonance of the bases of the lung is, therefore, frequently conjoined with tubercular dulness of the apices. The expiratory efforts of cough are the determining cause; the damage inflicted

[1] It is possible that in many cases asthma precedes Pulmonary Vesicular Emphysema, and the violent efforts to inspire are the determining causes of the Emphysema, and that in this as in so many other cases the effect has been mistaken for the cause.

on the textures by the pleurisy is a common cause of the permanence of the over-distension. The Vesicular Emphysema in this and similar cases, when tolerably limited in extent, is not attended with notable disturbance of respiration or circulation. It must, it is true, add a little to the shortness of breath, and a little to the impediment to the circulation through the lungs, but these additions are insignificant in comparison with the primary disturbances of respiration and circulation resulting from the original disease. Diseases attended by incomplete occlusion of the air-passages frequently have, as consequence, over-distension of the air-vesicles of the whole or of part of the lung.

In accidental occlusion of the larynx, Acute General Vesicular Emphysema is frequent. Thus, in the case of a woman, who in a state of drunkenness choked herself by thrusting the food with her finger from the over-full pharynx into the larynx, the lungs were the seat of extreme General Acute Emphysema.

In such cases, supposing the obstruction to be at first incomplete, the sufferer does what he would do if the obstacle were removable—he makes the deepest possible inspiration, and then coughs. The violent expiratory effort drives the air into the less compressed and supported parts. The local obstacle to the escape of air being irremovable, causes an excess of air to be retained in the lungs. A second deep inspiration follows, and so finally general over-distension of the air-vesicles is established.

The full normal distension of the lungs with air may be mistaken for Acute General Emphysema, if death occur while the lungs are distended by a deep inspiration, and a foreign body in the larynx prevent the expulsion of the air from the lungs by the natural death-expiratory act.

In croup, the false membrane in the larynx may, in rare cases, act for a short time as a valve, admitting the air to pass into the lung, but opposing its escape, and so cause Acute Vesicular Emphysema.

In croup, again, pulmonary lobular collapse and lobular pneumonia are both common, and, when present, some of the Acute Vesicular Emphysema found after death may be secondary to those lesions of structure.

But more commonly than in either of the modes just enumerated, the Acute Vesicular Emphysema found after death in croup is produced during the expiratory efforts of coughing; that it is so produced is proved by the situations it occupies, viz., those parts of the chest which in these cases are seen during life to recede during inspiration, and to advance during expiration.

In hooping-cough, Vesicular Emphysema is a constant result of the violent expiratory efforts of that disease. When the over-distension is extreme, and is frequently repeated, the elasticity of the walls of the vesicles may be diminished, and then a certain amount of over-distension remains a permanent lesion. As bronchitis is a frequent complication of hooping-cough, disseminated lobular collapse may in some cases aid to a slight extent in the production of the Vesicular Emphysema.

Hereditary nature of Pulmonary Vesicular Emphysema.—Jackson[1] found that 18 of 28 subjects of Pulmonary Emphysema were born of parents one of whom was affected with the same disease; while of 50 non-emphysematous patients 3 only came of emphysematous parents; and Niemeyer remarks, "I have known at least one family in which, without catarrh preceding, all the members for three generations suffered from emphysema."

These facts afford some support to the theory that Pulmonary Vesicular Emphysema is hereditary. But Pulmonary Vesicular Emphysema is far too common a disease for Jackson's few oft-quoted observations, or for solitary badly stated facts, such as that of Niemeyer, to *prove*, or even to render it highly probable that it is really hereditary.

If the cases be excluded in which the disease owes its origin to inherited predisposition to bronchitis, heart disease, asthma, premature age-degeneration, &c., the facts adduced in support of the hereditary nature of Pulmonary Vesicular Emphysema will be reduced to an insignificant figure. It is not denied that the disease may be hereditary, but it is without question in the writer's mind that the evidence adduced in support of its being hereditary in the sense in which tubercle and cancer are hereditary, is altogether insufficient for the proof.

Diseases of the lungs to which Pulmonary Vesicular Emphysema indisposes.—Pneumonia with exudation of lymph, croupose pneumonia as it has been called, rarely occurs in the emphysematous parts of a lung. The destruction of the capillary vessels which occurs in Chronic Vesicular Emphysema, is supposed to afford a certain degree of exemption from this form of inflammation. Although a streak or two of blood in the sputa is not uncommon in the bronchitis from which emphysematous patients suffer, hemorrhage in quantity from emphysematous lungs is said to be rare. Those suffering from Atrophous Vesicular Emphysema alone enjoy a practical immunity from pneumonia and from hemorrhage.

Tubercle has been said to be excluded by Pulmonary Vesicular Emphysema.

[1] Quoted by Louis.

No doubt active congestion of a part accompanies the formation of tubercle, and active congestion rarely affects the emphysematous parts of a lung, and to the same extent Vesicular Emphysema of the lung indisposes to the deposit of tubercle. It is to the wasting of the vessels in Emphysema of the lung that the immunity, so much as it is, from tubercle is due.

The blood in Pulmonary Vesicular Emphysema is less fully aërated than in health. To this venosity of the blood, Rokitansky attributes the infrequency of tubercle in the subjects of Pulmonary Vesicular Emphysema. His theory, however, is opposed by facts.

TREATMENT OF CHRONIC PULMONARY VESICULAR EMPHYSEMA.—The treatment of Pulmonary Vesicular Emphysema may be divided into Curative, Palliative, including the treatment of its direct consequences, and Preventive.

Curative Treatment.—Some therapeutists have supposed that, by the lengthened administration of small doses of strychnine, and others, that by the skilful employment of electrical power, permanent contraction of the walls of the dilated air-vesicles may be obtained. But, although powerful agents for exciting muscular contraction, neither strychnine nor electrical action have any influence in restoring or increasing the elasticity of a tissue. In Chronic Pulmonary Vesicular Emphysema, it is the elasticity of the walls of the air-vesicles which is damaged. Experience, as might have been anticipated, has afforded no evidence in support of the value of the drug or the battery in the cure of Pulmonary Vesicular Emphysema.

Again, some therapeutists have placed persons suffering from Chronic Pulmonary Vesicular Emphysema in a chamber supplied with condensed air, in the hope that the breathing of the condensed air would cure the disease. Others have alleged that great attention to diet, and the administration of iron, and other blood, nervine, and stomach tonics, will, by improving the nutritive powers, cure the disease. But, if it be remembered, that to cure Chronic Pulmonary Vesicular Emphysema of severity sufficient to cause trouble to the patient, is not only to renew the elasticity of the walls of the air-cells, but also to restore the stretched vessels to their normal length and to their natural tone, to repair the apertures in the walls of the air-vesicles, and to replace the torn and otherwise destroyed capillaries by healthy vessels, it will be at once admitted that the cure of Chronic Pulmonary Vesicular Emphysema is impossible. Persons suffering from Emphysema have been greatly relieved by breathing condensed air; but the relief, as might be anticipated, has been temporary only; and while attention to diet, &c., is of no avail to cure the disease, it is of great importance in staying the progress of the disease and relieving distress.

Preventive Treatment.—The great factors of Pulmonary Vesicular Emphysema being—

Excess of pressure of air on the inside of the air-vesicles,

Age degenerative changes of the parietes of the thorax,

Changes of the texture of the lung, from excess of blood in it.

Age-degenerative changes of the lung, in order to prevent the disease and to stay its advance when established, care must be taken to guard against these, its determining and permanence-securing causes. Catarrhal, and all other forms of bronchitis being beyond question the most frequent excitors of the Pulmonary Vesicular Emphysema, the prevention of these diseases is of the very highest importance. To secure immunity from bronchitis, and to prevent its recurrence, clothing must be adapted to the season, and it is necessary that cold and wet, especially fog and cold winds, be avoided. A mild climate has a marked influence in preventing the attack of bronchitis, to which so many are subject during the winter in this country. Hence it is most important for those whose lungs are the seat of Vesicular Emphysema, to spend the winter in a mild, and not too dry air.

When chronic or subacute bronchitis is present, the freer the secretion from the bronchial mucous membrane, and the less violent the cough, the less likely is Chronic Emphysema to follow. Expectorants and opiates combined are the great medicinal agents. The expectorants selected when the cough is dry, should be those that promote secretion; when the secretion is abundant, those that favor its expulsion.

Violent and irritative cough—that is, cough out of all proportion to the matter to be expectorated, should be restrained by sedatives: Opium, belladonna, stramonium, conium, and prussic acid, are the chief sedative agents in this class of cases. These drugs are more efficacious when given with little water, and in a small quantity of mucilage and syrup. Sedative inhalations are particularly useful. The sedative should be placed on the sponge of Maw's inhaler, and the steam of hot water passed through the sponge. Chloroform vapor exhibited in this way is sometimes very serviceable.

When the secretion from the bronchial mucous membrane is too abundant and purulent, the mineral acids, quinine, iron, especially the tincture of the sesquichloride, and cod-liver oil, are invaluable; as is also the inhalation of mild stimulants,

e. g., iodine diffused in small quantity through the room. In these cases, a change to dry sea-air is often very serviceable.

All efforts which try the muscular powers, as carrying heavy weights, are injurious. All exertions which induce panting, or oblige the person to stop frequently to recover his breath, are calculated to inflict permanent injury. Rapid walking, hill climbing, and violent exertions of all kinds, are to be carefully avoided. Walking exercise should, as much as possible, be limited to level ground.

Many an old gentleman has been hurried to his grave by attempting to follow the birds as he did in his early days, and by striving to improve his health by active exercise, It is a great gain for length of life to take old age pleasantly. Those predisposed to the disease and, *à fortiori*, subjects of Pulmonary Vesicular Emphysema, should never attempt to play wind instruments.

When urging these points on a patient, it must never be forgotten that the permanence-securing cause being established, every single over-distension of the air-vesicles permanently increases their size. The increase on each occasion is indeed insignificant; but as every repetition of the over-distension adds to that previously existing, it follows that, should the over-distension be frequently repeated, a considerable amount of dilatation must be the ultimate result.

All the foregoing means which are of importance in preventing the occurrence of the disease, are practically still more important as preventing its increase when established. Many a man whose wind was merely touched, has become dropsical, &c., by attempts to renovate himself, by endeavors to climb, hunt, and shoot as he did before his "wind" began to go. Old age has commenced on his chest; he is but between fifty and sixty, and he won't admit the existence of it. He strives against its inevitable consequences, and dies from the effects of the struggle years before he would have done had he shunned the contest.

All measures which oppose the supervention of the degenerative changes of age, are to be sedulously employed, with the hope not only of specially retarding age-degeneration of the lungs and thoracic parietes, but of the body generally.

Diet, carefully regulated exercises, and, of drugs, iron and cod-liver oil, especially the former, are among the most potent means for effecting the object in view.

Palliative Treatment.—The distress from which the subjects of Large-lunged Vesicular Emphysema suffer, is due—

1. To shortness of breath;

2. To congestions of distant organs produced mechanically by the impediment to the flow of blood through the pulmonary capillaries;

3. To the abnormities of blood which result from the functional and structural changes of the liver and kidneys especially, consequent on their congestion.

1. The remedies for the shortness of breath vary according to its direct cause. Having regard to the treatment, the causes of shortness of breath may be summed up thus:—

(*a*) Organic changes in the walls of the thorax, in the walls of the air-vesicles, and in the capillaries in the walls of the air-vesicles, and dilution of the air received into the air-vesicles at each inspiration, by the excess of air retained in them at the termination of expiration.

Breathing condensed air, it appears probable, temporarily relieves the distress of breathing due to the dilution of the air. Whether it does more than this is doubtful.

(*b*) Catarrhal and other forms of bronchitis.—For the shortness of breath arising from these affections, expectorants which both favor free secretion and expectoration are the great remedies. Ipecacuanha and carbonate and muriate of ammonia, squills and senega are the most potent remedies (see art. Bronchitis).

(*c*) Asthma.—Free secretion and expectoration from the bronchial mucous membrane, affords the most efficient relief in continuous shortness of breath from this complication. Ipecacuanha, squill, ammonia, and senega alone, or combined with sedatives and antispasmodics, are the agents best calculated to attain the desired end.

It must not be forgotten that disturbances of the digestive organs, the liver, stomach, and bowels are common in Large-lunged Vesicular Emphysema, and are also frequent exciting causes of attacks of spasmodic asthma in that disease.

(*d*) Congestion of the liver, accumulation of flatus in the stomach and bowels, and loaded bowels by interfering with the descent of the diaphragm, are common causes of shortness of breath.

It is in consequence of this that a full dose of blue pill, or calomel and colocynth, followed by a brisk, warm, saline aperient, so often affords marked relief to the dyspnœa of Large-lunged Vesicular Emphysema.

Blue pill occasionally, aromatic saline antacid aperients, taraxacum with soda, or nitro-hydrochloric acid with aromatics, and attention to diet, are the means best calculated to ward off shortness of breath from these causes.

2. In treating the congestions of organs, two objects have to be kept in view.

1st. To remove the impediment to the flow of blood through the lungs.

2d. To relieve directly the local congestions.

The impediment to the flow of blood due directly to organic changes in the walls of the air-vesicles and in the pulmonary capillaries, is irremediable. Catarrhal and other forms of acute and chronic bronchitis increase the impediment to the capillary circulation through the lungs: and, therefore, to relieve those affections, is to relieve the congestion of the venous system. Free secretion and expectoration from the bronchial tubes, is the most efficient agent for affording relief in these cases.

Violent cough again impedes the flow of blood through the lungs, and so produces congestion of the venous system.

Sedatives, therefore, by checking cough, become means of relieving local congestions.

Free secretion from the kidneys, liver, and intestinal mucous membrane, relieve the local and general over-distension of the capillaries and veins of those organs, resulting from impediment to the flow of blood through the lungs.

Of diuretics, the ordinary salts of potash, with small quantities of iodide of potassium, are, as a rule, the most efficacious. This class of remedies should be preceded by one or more doses of blue pill, with squill and digitalis.

It is common for diuretics not to act till the tension of the venous system has been, to some extent, taken off by other means. Hence should diuretics fail when first given, aperients may be employed, and their use be followed by diuretics with advantage.

Blue pill, and other cholagogues, followed by hydragogue aperients, such as cream of tartar with jalap, effect the desired object by promoting a full flow of secretions from the liver and intestines, and so especially relieving congestion of the portal radicles and terminal branches.

A natural relief is occasionally afforded to a congested organ by spontaneous hemorrhage from it. Cerebral congestion is relieved by epistaxis; congestion of the lungs by hæmoptysis; of the liver and intestines, by hæmorrhoidal bleeding; of the kidneys, by hæmaturia; of the stomach, by hæmatemesis. The blood thus lost may not only relieve the vessels of the organ from which it escapes, but the venous system generally.

When congestion of an organ is extreme, the application of dry-cups, or the removal of a small quantity of blood by cupping-glasses, is sometimes very useful. When the distension of the whole venous system is extreme, the removal of a little blood from the arm gives marked and sudden relief when judiciously performed.

The stomach dyspeptic symptoms are due chiefly to congestion of the stomach following on congestion of the liver. They are best treated by occasional doses of mercurials, saline aperients with mineral acids, and mustard poultices to the epigastric region. These remedies may be followed by small doses of strychnine, and light aromatic bitters.

Aromatics, with alkalies, afford temporary relief to the sense of distension and weight.

3. The congestion of the kidneys is sometimes accompanied by the retention of urinary elements in the blood, congestion of the liver by slight jaundice, and finally by organic diseases of these organs and then all the abnormities resulting from those diseases follow.

For the special treatment of the conditions of blood dependent on the diseases of the liver and kidneys, see the article on those diseases.

Here it is only necessary to say that the treatment before recommended for the relief of the congestion of these organs, is that best calculated to secure the removal from the blood of the elements retained in it.

In cases of Atrophous Pulmonary Vesicular Emphysema, the great object is to support the failing general powers. Iron is one of the most important tonics in this class of cases. A moderate supply of stimulants is useful.

When accompanied with profuse purulent expectoration, mineral acids, especially the sulphuric, with small doses of quinine, tincture of the sesquichloride of iron, cod-liver oil, and mild sea-air, are the great remedial agents. Stimulating inhalations are sometimes serviceable.

WORKS CONSULTED.

Laennec, Traité de l'Auscultation médiate et des Maladies des Poumons et Cœur. Paris: 1826.

Magendie, Leçons, 1825. Tome i.

Louis, Recherches sur l'Emphysème des Poumons. Mémoires de la Société d'Observation. Tome i. Paris: 1837.

Dr. G. Budd, Remarks on Emphysema of the Lungs. Med.-Chir. Transactions, 1840.

A. Mendelsohn, Der Mechanismus der Respiration und Circulation. Berlin: 1845.

Dr. Sibson, On the Movements of Respiration in Disease. Med.-Chir. Trans., vol. xxxi. 1848.

Dr. Gairdner, On the Pathological States of the Lungs connected with Bronchitis and Bronchial Obstruction. Edin. Monthly Journal, 1851.

Ranke, H., Beobachtungen und Versuche über die Ausscheidung der Harnsäure. Munich: 1858.

Parkes, On the Urine. London: 1860.

Donders, Beiträge zum Mechanismus der Respiration und Circulation in gesunden und krankheiten Zustande. Zeitschrift für Rat. Med., 1853, p. 287.

Freund, Der Einfluss der primären Erkrank-

ungen des knorpeligen Thorax auf Entstehung gewisser Lungen Krankheiten. Würzb. Verhandl. 1859.

Ziemssen, Ueber die Pathogenese des substantiven Lungen-Emphysems. Deutsche Klinik, p. 157, 1858.

Biermer, Lungen-Emphysem. Virchow's Handbuch der Pathologie und Therapie, vol. v.

Rokitansky, Lehrbuch der Pathol. Anatomie, Bd. 3, 1861.

Dr. A. T. H. Waters, Researches on the Nature, Pathology, and Treatment of Emphysema of the Lungs, 1862.

Niemeyer, Ueber Emphysem der Lunge. Berlin. Med. Wochenschrift, 1864.

Villemin, Recherches sur la Vésicule pulmonaire et l'Emphysème. Archives Générales de Méd., Oct. and Nov. 1866.

Hourmann et Déchambre, Archives Générales, 1835.

Rainey, Medico-Chirurg. Soc. Trans., 1848.

Rossinol, Rech. anat. sur l'Emphysème, 1849.

ASTHMA.

By Hyde Salter, M.D., F.R.S.

Definition.—Asthma may be defined as dyspnœa of peculiar urgency and violence, generally paroxysmal and recurrent, often periodic, not necessarily attended by cough or expectoration, accompanied usually by dry râles, and compatible with easy and healthy respiration in the intervals of the attacks.

The History of Asthma may be divided into the History of the Paroxysms and the General History of the Disease.

A. Symptoms of the Paroxysms.—Before the attack itself sets in, it is not at all uncommon for the asthmatic to be aware that it is impending, by certain premonitory symptoms with which his experience has made him familiar, and of which it has given him the infallible interpretation. It is, however, more common for there to be no distinct premonitory symptoms, but for the first slight traces of the attack to be the only warning of its approach. When premonitory symptoms *do* occur, they are generally such as are referable to the nervous system; as, for example, an unusual buoyancy of spirits and mental excitement, or depression, lethargy, and an irresistible sleepiness. One very common symptom is profuse diuresis, the patient passing, some hours before the attack, a large quantity of clear pale urine, almost as white as pump-water—identical, in fact, with what is called nervous water, or hysterical water. As a rule, however, as has been mentioned, the patient has no warning, no sign whatever to guide him; and this is one of the peculiar features of Asthma, and imparts to it that uncertainty and ever-possible nearness which makes it so disqualifying a disease and one so destructive of the engagements of life and the duties of an active career. It always threatens because it *never* threatens. Nay, more, from the time at which it is most apt to occur—the early morning—the patient has not even the warning of the *initiatory* symptoms, but sleeps through them till he is awoke to find himself with the attack full upon him. He goes to bed every night uncertain whether his next return to consciousness may not be among the full developed horrors of the asthmatic struggle.

The *Initiatory Symptoms*, when they are not slept through, and when they show themselves by day, consist in a faint development of the characteristic dyspnœa of Asthma—a slight sense of constriction across the chest, a short dry cough, a tendency to wheeze, and an indisposition to exertion. The asthmatic's friends notice that he walks about with his shoulders higher than usual; and he complains of flatulent distension which makes the girth across the epigastrium greater than usual, so that he unbuttons his waistcoat to give himself ease and room. This is not a mere sensation, the circumference of the chest is really considerably increased, and the patient usually attributes it to distension of the stomach with wind. No doubt, in many instances, there *is* a large development of gas in the stomach at the commencement of an asthmatic paroxysm; but in the great majority of instances this increased girth of chest and abdomen is probably due to that enlargement of the cavity of the chest which always accompanies the asthmatic state, and is a part of it, of which more will be said by and by, and which enlarges the girth of the chest by the elevation of the ribs, and of

the abdomen by the depression of the diaphragm.

These initiatory symptoms may hang about for some time, even for some days, before they culminate in an attack, the asthmatic creeping about with a gradually increasing sense of constriction across the chest, a more perceptible wheeze and a greater and greater incapacity for exertion. At other times the paroxysm is at once so fully developed that it can hardly be said to have any initiatory symptoms at all.

There is nothing more uniform in Asthma than the time at which the attack is apt to come on. In nineteen cases out of twenty, this is in the early morning, from two to four o'clock. So uniform is this, that it is the exception to find it otherwise. Each case, as a rule, has its own particular time: one will always be awoke at two, another at three; and so unvarying is the time, that the patient often knows exactly what o'clock it is by his asthma waking him. There are, however, two circumstances that vary the time: one, the hour at which the patient goes to bed—the earlier this is, the earlier does the attack come on; and the other, the intensity of the exciting cause—the more powerful this is, the earlier is the attack likely to follow it: thus a supper in a favorable air may bring on the Asthma at three o'clock; in an unfavorable air as early as one. Occasionally the Asthma will remain in abeyance as long as sleep lasts and develop itself immediately after waking; but this is rare. A not at all uncommon time for the attack to come on is about two hours after dinner. In some, it always appears on getting into bed at night. There cannot be a doubt that the part of the four-and-twenty hours the most free from it is the forenoon, from breakfast to luncheon or early dinner; many asthmatics who suffer more or less at all other times are free then.

When the paroxysm is fully developed, the appearance of the patient is very characteristic. He sits in a fixed position, unable to move, generally leaning forward with his hands or elbows planted on something in order to raise his shoulders; sometimes he *stands*, leaning over some piece of furniture, finding this position easier than sitting: kneeling up in bed, leaning over the pillows, or kneeling on the floor against a bed or chair, is, in many cases, the easiest position. He is pale, or, if very bad, dusky in complexion; the shoulders are raised, the back rounded, and the sweat often pours off the face from the violence of the respiratory efforts. These efforts are so great that the body is quite convulsed by them, the shoulders are thrown up, the head thrown back, and the mouth opened at each inspiration; and all the muscles mediately or immediately connected with the chest thrown into violent action. The patient cannot bear anything tight around his body, all his clothes must be loosened; all curtains drawn around him, all bystanders crowding about him, seem to increase his sense of suffocation. Sometimes he will sit by an open window the whole night in the coldest weather, so great is the desire for fresh air; he feels as if death was impending, as if his chest were bound with iron, and as if the only thing that would give him relief would be to cut it open. The extremities are often cold, especially the lower extremities, although the perspiration may be running from the face at the same time; the pulse is small and quick.

If the patient is stripped and the chest watched, it will be seen that, although the respiratory efforts are so violent, there is very little real movement, the muscles tug at the ribs, but the ribs refuse to rise—they strive to compress them, but they refuse to subside. Although violent the respirations are not often hurried, not exceeding the natural number; but although the number may be natural, in every other respect the respiratory rhythm is disturbed; the inspiration is short and jerky; the expiration inordinately long, often wound up with a sudden pumping out of the last quantities of the expired air; and there is no post-expiratory rest.

On listening to the patient's chest, everything seems lost in loud musical râles of high pitch, and mostly sibilant; among these is also often heard sonorous rhonchus. These sounds are multitudinous, of all pitch, and utterly discordant, squeaking, chirping, mewing, whistling, cooing, snoring, and fifty other sounds. They are almost invariably louder at expiration, and sometimes confined to it. The *typical* sounds of Asthma are of this dry character; occasionally, however, moist râles are heard either from the Asthma being complicated with bronchitis, or from the attack approaching its termination, when mucus is being poured out.

But there is one auscultatory phenomenon in Asthma, which, although negative in its character and apt to be overlooked, is far more important and significant than these noisy manifestations of bronchial stricture; it is the almost complete or total absence of the respiratory murmur: this is not only not heard because it is drowned by the other sounds, but because it is really, for the time being, in abeyance; for even when the musical râles are absent, as they sometimes are, the respiratory murmur is equally defective.

The reason of this absence of respiratory murmur is that the bronchial spasm prevents the air reaching the recesses of

the lungs in sufficient quantity to generate it. The more severe the Asthma, the more complete is the loss of the respiratory murmur, and no sooner does the spasm yield than the normal sound is reinstated.

Not unfrequently, the physical signs of Asthma, above described, are partial in their distribution, the respiratory murmur is not totally absent but crops up tolerably clearly here and there; the sibilant and sonorous râles, too, are patchy—some parts of the chest, especially those where the respiratory murmur is best heard, being free from them.

There can, I think, be but one interpretation to this patchy distribution of the physical signs of Asthma; namely, that the Asthma itself has a patchy distribution—that the tubes are in some parts affected, in others free. In those parts where, from the absence of spasm of the tubes, the access of air is free, we often hear a respiratory murmur of strongly-marked compensatory character, the inrush of air remarkably clear and loud. This is just what might be expected, and depends upon the whole of the violent inspiratory efforts being spent upon those parts of the lung where the absence of bronchial spasm renders its inflation possible.

There is yet another fact to be observed; namely, that the physical signs of Asthma change their seat with considerable rapidity: we may hear a patch of sibilus or rhonchus one minute, and the next it may be gone; we may find complete absence of respiratory murmur, and in a quarter of an hour, in the same place, it may be quite audible. In fact, for no two consecutive hours does the asthmatic chest present the same physical signs at the same place. This fugitiveness and migration of the normal and morbid sounds show that the bronchial spasm itself is constantly changing its seat, that constricted tubes are constantly becoming relaxed, and patulous ones contracted. Occasionally, the physical signs show that the Asthma is lateral, confined to one lung, or nearly so, the râles will occupy one lung and normal breath-sound the other. The patient himself is sometimes quite aware that only one of his lungs is affected, and knows which it is. In most of those cases in which Asthma is thus restricted to one side, it always occurs more or less on the same side. In such cases there is probably some organic cause, such as emphysema or bronchitis, for its localization.

Percussion, during a paroxysm of Asthma, is always exaggeratedly resonant; in this, the chest distension, and the loss of respiratory murmur, the conditions bear a striking resemblance to emphysema: in one respect, however, besides the temporary duration of these signs, there is a marked contrast; in emphysema, the intercostal spaces are not depressed, even in strong inspiration, while in Asthma they are drawn in to an extraordinary degree; and not only the intercostal spaces, but all yielding parts bounding, or lying contiguous to, the chest cavity, as the supra-sternal and supra-clavicular fossæ, and the scrobiculus cordis. The surface in these situations is literally sucked in at each inspiration, and nothing more strikingly suggests than this appearance the real nature of the difficulty in asthmatic breathing, nothing more strikingly proves that it must depend on a condition which temporarily renders the lung incapable of following the inspiratory enlargement of the chest wall.

Before the paroxysm ceases there is commonly some expectoration. In many cases the fit never ceases without it, and not until the expectoration is established will the spasm give way, however long it may be delayed. It is this circumstance that has given rise to the theory that the material discharged has been the cause of the preceding attack, and that the violent respiratory efforts are merely the mechanism which nature adopts to get rid of it. This was Bree's theory, and expressions frequently used by patients show that such an idea is still very prevalent. They say, "If I could once get the phlegm up, the spasm would give way." No doubt, the phlegm, when there, is a source of irritation and an additional cause of dyspnœa, and no doubt, if it were discharged, the patient would be, *pro tanto*, under better circumstances. No doubt, too, in many cases, the spasm will not give way till the expectoration takes place, but only because the expectoration will not take place till the spasm begins to yield. Till that takes place, the mucus is locked up behind the constricted tubes, and cannot be discharged partly because its channel of egress is too narrowed, and partly because sufficient air cannot be introduced behind the constricted tubes to produce efficient cough. That the pituitary theory of Asthma—the theory of a material irritant present in the secretion of the tubes—is incorrect, is shown by the fact that there are many cases in which there is no secretion first or last, the chest sounds being dry throughout, and the spasm going off without any expectoration. And even in the cases where the attacks always terminate with more or less spitting, they begin dry. At first there is no cough, and nothing but a dry wheeze; by and by, to the wheeze, rattling is added, and cough begins to appear, and before the paroxysm is over moist sounds may be heard all over the chest, and the cough may be incessant. The mucus has evidently been gradually

developed as the attack has progressed. In fact, the spasm is the cause of the secretion, and not the secretion the cause of the spasm. This is not at all inconsistent with the discharge of the secretion, when once formed, being attended with relief.

The material itself is peculiar and very characteristic. True asthmatic septum, where there is no bronchitis, consists of little pellets of gray pearly mucus, like pieces of tapioca, or very firm arrowroot. It is free from pus, free from either stringy or watery mucus and not frothy.

Rarely, another material is expectorated during the attack—blood. In most instances where this occurs it is only characteristic of the severest paroxysms; in some few cases I have known every attack attended with more or less blood-spitting. It is generally small in quantity, in streaks and patches; sometimes it amounts to a profuse hemorrhage. It evidently depends upon the rupture of the over-distended bronchial venules and capillaries, due to the congestion into which they are thrown by the partial asphyxia of the asthmatic paroxysm.

The *duration* of a fit of Asthma is a thing about which there is no rule—it may be over in a few minutes, it may last many weeks. But though there is no rule for the *disease* there generally is for the *case;* each case has, as a rule, its own length of attack although the uniformity may not be rigid. A very common length is for the attack to begin at three or four o'clock in the morning, and be over by breakfast time, or gradually clear off at nine or ten o'clock in the morning. In many cases, the attack involves a single day, never more or less. From two or three days is not an uncommon duration. In some cases, the duration is complex, each attack extending over many days and consisting of a succession of shorter paroxysms with easy breathing between; and then, the bout being over, many months may be passed before another attack occurs. This is especially the case in those instances in which the Asthma is due to some cause that occurs at distant intervals, such as hay Asthma. Here the asthmatic state will last, more or less, throughout the whole of the grass-flowering season, although consisting throughout that time of short paroxysms, each not lasting above an hour or so, and perhaps confined to the night.

The *method of termination* of an attack depends very much upon two circumstances: one, the length of time the attack has lasted; and the other, whether it yields spontaneously, or in obedience to remedies. If the attack has lasted long it is always slower and more protracted in its departure; for the lungs are left so congested that it may be days before their circulation can resume the condition it was in before the fit. For the same reason, the expectoration after a prolonged attack is more profuse and continues longer. Again, if an attack is left to die out by itself, its departure is often tedious, and it may show many partial remissions before it takes its final leave. If, however, some powerful influence is brought to bear upon it, it may yield almost instantaneously—the patient may be one minute struggling for breath and the next without a trace of dyspnœa, as for example where the Asthma is suddenly arrested by some violent emotion, as fear, or where it yields to the influence of some powerful depressant, as tobacco. Unless the paroxysm has been of very short duration it generally leaves the patient with a sense of clogging and stiffness at the chest; he feels himself more than usually incapable of exertion, and is easily winded: this gradually gets less and less, and in a day or two may have completely passed away. The expectoration often continues for several days after the attack is quite gone; at first, it occurs throughout the day, is then confined to the morning, and finally ceases altogether.

There is one peculiarity in the state of the asthmatic after the paroxysm that is especially worthy of remark, and that is the diminution of the asthmatic tendency that he then experiences—the almost certain immunity, for the time being, from a repetition of the attack. There are many things that he dare not do at other times without the certainty of bringing on a paroxysm, that immediately after an attack he may do with perfect impunity. It seems as if each fit were a sort of "clearing shower," as if the tendency to fall into the asthmatic state accumulated in the intervals, and was, so to speak, discharged by the paroxysms. Certainly the fact, which we frequently see in Asthma, that the longer the time that has elapsed since the last attack the more particular must the asthmatic be in not exposing himself to the ordinary exciting causes of his disease, and the more sensitive of their influence does he become, is compatible with this idea. We see just the same thing in epilepsy.

B. Such is the history of an asthmatic paroxysm. But, besides the features of a paroxysm, there are certain points in the history of the disease that deserve notice. Of these I will especially advert to three:—First, the periodicity that the disease so frequently exhibits; secondly, the change of phase that time impresses on many cases; and thirdly, the influence which sex and age appear to exercise on the liability to the disorder.

1. *Periodicity.*—That Asthma is markedly periodic no one who has watched it

can doubt. Although in some instances there appears to be no particular interval at which the attacks are apt to occur, yet, in the majority of cases, the interval is well marked, and in many, minutely and singularly regular. This is one point, among many others, that vindicates for Asthma a place among the neuroses. But while each case preserves its own periodicity with more or less regularity, there is no particular period for *Asthma* itself; for, while in one case the attacks will occur regularly at the same time every night, in another they will occur once a month, in another once a year. So regular is the time of recurrence in some cases that the asthmatic knows exactly when to expect an attack. The periodicity of Asthma is clearly divisible into two kinds, *intrinsic* and *extrinsic*. Of the former, which is the only true essential periodicity, we see examples in those cases in which the attack comes on after the same interval irrespective of external circumstances. Of the latter we see examples in those cases where the regular return of the attack is simply due to the regular return of the exciting cause. The period in the last cases is almost always that of some natural interval. Thus the annual periodicity of hay fever is of this kind; so is the monthly periodicity of hysterical Asthma, and the diurnal periodicity of cases in which sleep and the recumbent posture induced the attacks. Indeed, in any case in which the periodicity affects some natural interval I should suspect that it was extrinsic, and dependent on the periodic recurrence of the exciting cause. Thus, in all cases in which the attack comes on every Saturday, or every Sunday, or every Monday morning, I believe the attack is due to something having a weekly recurrence, something in which Saturday and Sunday differ from other days—a suspension of the usual employment; difference of food, or the time of taking it; sleep after food; the taking of supper. That the periodicity of such cases is not inherent or essential is shown by the fact, that if the exciting cause is made to recur at irregular intervals the attacks become correspondingly irregular, and all periodicity is lost.

2. *Change of Type by Time.*—It is not at all uncommon to see the features of a case change considerably as time advances, and ultimately differ very much from what they were at first. And there is a certain type of change that commonly obtains, so that it is possible in any given case to predict what the effect of time will probably be. As a rule, the attacks are the most violent in the early history of a case, and gradually becomes less and less severe. It is very common for patients to say, "I never have those awful attacks now that I used to have, they seem to have quite left me." But while the attacks become milder they often become more frequent, so that a monthly periodicity may be exchanged for a weekly, or diurnal one. At the same time, another change is generally going on—the breathing in the interval is getting less and less free; certain slow organic changes are gradually being impressed on the lungs by the repetition of the attacks, by which their functional integrity is increasingly impaired; so that while at first the attacks are severe and distant, and the breathing in the interval like that of a healthy person, after a time the paroxysms become so slight and frequent, and the breathing so embarrassed, even at its best, that there can hardly be said to be any distinct attacks, and the disease has ceased to be paroxysmal.

3. *Age* and *Sex.*—It is a commonly received opinion that Asthma is a disease of advanced life. Nothing can be more erroneous. It is confined to no age; and so far is it from being peculiarly a disease of the old, that I find a larger number of cases take their origin in the first ten years of life than in any subsequent equal period.

During youth, from ten to twenty, few cases originate; but, from that time up to fifty, the asthmatic tendency regularly increases. From that time forward, fewer and fewer cases take their origin. No doubt, many old people are asthmatic; but that is simply because many asthmatic people reach old age. It must be borne in mind, too, that with respect to old people the word "Asthma" is very loosely used: three-fourths of the "Asthma" of old people are due to chronic bronchitis.

Men are liable to Asthma, in relation to women, in the proportion of two to one.

C. VARIETIES OF ASTHMA.—All cases of Asthma fall, I think, under one of two main divisions—Idiopathic or Primary, and Symptomatic or Secondary. Idiopathic Asthma is the inherent and essential form of the disease that occurs independent of and uncomplicated with any other affection. The best marked, most typical, and characteristic cases, and I may add the most severe are of this kind. In these cases we generally get considerable intervals between the attacks, those intervals being marked by perfect freedom of breathing, and the attacks by a regular periodicity. There are many points of resemblance between this variety of Asthma and epilepsy. Both of them affect the same nervous temperament, both of them are markedly periodic, and in both each paroxysm seems to act as a sort of thunderstorm, and to discharge, or work off, some particular state which constitutes the liability to the condition, and which accumulates in the intervals, and

reaches its maximum immediately before the fit. So close a relation, indeed, exists between this form of Asthma and epilepsy, that I have seen two or three well-marked cases in which the one kind of fit took the place of the other. Most examples of Asthma in the young are of this idiopathic type.

Symptomatic or Secondary Asthma may be sub-divided into three varieties—peptic, bronchitic, and cardiac—that is to say, Asthma may have its origin in stomach derangement, in an inflamed condition of the bronchial mucous membrane, and in heart disease. Each of these varieties has something peculiar in itself, depending generally on the nature of its cause. Thus *peptic* Asthma is apt to come on two or three hours after taking food, may be entirely regulated by dietetic rules—brought on at any time, or kept off indefinitely at pleasure—according to what is eaten, and when. It is remarkably independent of the other recognized causes of Asthma, and is (through the stomach) more amenable to treatment, and more hopeful, than any other form. Bronchitic Asthma, perhaps the commonest of all, is distinguished from all other varieties by certain well-marked characteristics. It is only caused by the causes of bronchitis, especially cold. As a rule, the patient never has Asthma without bronchitis, and never has bronchitis without Asthma, so that we generally have really a complex condition to deal with, although the bronchitic element may sometimes be so slight as hardly to be detected. There is generally in these cases an abundant expectoration and a good deal of cough and moist breath sounds. Such cases are often very intractable, and from this reason, that we have two diseases to treat—bronchitis and Asthma: the bronchitis is intractable because it is so greatly aggravated by the Asthma, and the Asthma is intractable because its exciting cause, the bronchitis, abiding, any remedies that are brought to bear upon it are rendered inoperative or merely of transient efficacy. Indeed it so happens, that in one element of treatment, air, that which is best for the bronchitis is often worst for the Asthma, and *vice versâ*. Thus many of these cases lose their asthmatic tendency in London, where the bronchitis alone survives; while if you send them to the Mediterranean for the cure of the bronchitis, the asthmatic tendency is so much increased, that they are worse than ever, so that they have the alternative of bronchitis at home or Asthma abroad. Being dependent on the causes of bronchitis, such cases are generally worse in the winter; indeed, in many of them, the Asthma occurs *only* in the winter. I think most of the cases in which Asthma occurs every morning are of the bronchitic kind, the reason being that the inflamed condition of the bronchial mucous membrane constitutes an ever-present exciting cause, which, in a person with the asthmatic tendency, only requires sleep and the recumbent posture in order to bring it into activity. The third variety, the least common of all, is Cardiac Asthma, or Asthma complicating heart cases, and depending upon the heart disease. A great deal that goes by the name of Cardiac Asthma is not Asthma at all; it is simply cardiac dyspnœa, unattended with any bronchial spasm. Now and then, however, a heart case is met with in which paroxysms of true Asthma occur, attended with wheezing, prolonged expiration, and other characteristic signs of Asthma. In these cases, I have no doubt that the immediate exciting cause of the Asthma is the pulmonary congestion produced by the heart disease.

This last variety, and bronchitic Asthma, when the bronchitis has become chronic, may be classed together as "organic" Asthma; the peptic variety and the idiopathic, as non-organic.

D. Causes of Asthma.—The causes of Asthma may be divided into two classes—those affecting the air-tubes primarily and directly, and those applied to some remote part. Of those that are brought to bear directly upon the air-tubes there are three kinds: first, things inhaled; secondly, some offending condition of the blood; and, thirdly, an inflamed condition of the mucous membrane of the air-tubes.

There is an endless variety of materials which, when respired, will produce Asthma in those possessing the asthmatic tendency, and they produce it no doubt by virtue of that morbid sensitiveness of the bronchial mucous membrane in which the Asthma in these cases essentially consists. Some of these materials are such as will produce a certain amount of Asthma in many people, such as the smell of a lucifer-match, pitch, smoke, pungent vapors. Some are rendered asthmatic by dust, some by fog and damp. Particular smells will at once bring on Asthma in some people, such as that of flowers—roses, for example, and privet. The commonest vegetable emanation having this effect is hay—this form of Asthma being well known as Hay Asthma, and a part of that curious disease, Hay Fever. In some people animal emanations have a similar effect: some are at once rendered asthmatic by the presence of a cat, some cannot go near a stable, or even ride behind a horse, or go near those who have been riding; some the effluvium of rabbits renders asthmatic; some, guinea-pigs; some cannot go near a poulterer's shop where there are hareskins; some have their Asthma brought on immedi-

ately if they go to a menagerie; and some suffer immediately if a dog comes near them. A more subtle influence is that arising from change of weather, or particular winds, some persons being rendered at once asthmatic by an easterly wind. A more subtle influence still is that arising from locality. Almost all asthmatics are influenced to a certain degree by the air they breathe, but to many it is the one thing that regulates their Asthma. Some are best in a dry air, some in a moist, some high, some low, some inland, some by the seaside. In some there is only one place that will render them asthmatic, in others there is only one place at which they are free from Asthma; in some the peculiar character of air that offends is well known; in some it is utterly inscrutable. In some so slight is the peculiarity of air that will determine the supervention of Asthma, that they may be perfectly well in the front of the house, but cannot sleep at the back. As a rule, a dry air is worse for Asthma than a rather moist one, the air of a high locality than a low. Yet the most constant circumstance noticed in respect to air, is the superiority of urban air over that of the country. So common an incident is this, that it becomes an important element in treatment—many a case of Asthma is at once cured by living in a dense quarter of some smoky and crowded city.

The cases in which I am inclined to think Asthma is brought on by *an offending condition of blood*, are cases in which it is apt to come on a little time after the ingestion of certain articles of diet. Cases in which people are asthmatic about two hours after a meal (a very common circumstance) are of this kind. In some only certain articles of diet will give rise to the Asthma, as wine, beer, sweets; in some only what upsets the digestion; in some any food whatever. My reason for thinking that it is the condition of the blood circulating in the respiratory organs after the absorption of these ingesta, that produces the bronchial spasm, and not the irritation caused by the presence of food in the stomach acting on the gastric periphery of the pneumogastric nerve, is the time at which the Asthma comes on. If it were the presence of the food in the stomach that caused the Asthma the symptoms would appear immediately on taking it, whereas it is not until a couple of hours afterwards, at about the time when the results of digestion are entering the circulation, that the difficulty of breathing comes on. Moreover, the rapidity with which the Asthma will supervene varies as the rapidity with which the ingesta are absorbed; thus, after wine, which is rapidly taken up, the Asthma will quickly make its appearance.

Inflammation of the bronchial mucous membrane is one of the commonest causes of Asthma, perhaps the commonest of all, especially in people in advanced life. Such cases are really complex cases, being bronchitic as well as asthmatic; indeed the bronchitic may be said to be the fundamental and essential part of them, and their essential treatment is the treatment of the bronchitis. Take care of that and the Asthma will take care of itself. The only difference between such cases and cases of ordinary bronchitis, is that the bronchitis happens to occur in individuals in whom bronchial spasm is easily induced.

The immediate excitants of the asthmatic paroxysm to which I have already referred are such as act directly on the bronchial tubes; but there are some that produce bronchial spasm by application to some remote part. Such causes always act, I believe, through the nervous system; and they may either act through the organic nerves or the cerebro-spinal. We see an example of the former in cases where Asthma is at once produced by a loaded stomach, or a loaded rectum: of the latter, where Asthma is at once produced by cold feet, &c. In both these classes of cases the exciting cause is applied to the periphery of the nerves on which it acts; but this need not be the case, for sometimes, the irritant is applied to a nervous centre. Asthma, for example, has been known to be produced by organic disease of the brain; and that very common occurrence, the production of Asthma by violent emotion, is another example of the same thing; only here the irritant applied to the centre is psychical and not physical.

In speaking of the causes of Asthma I must not omit to mention those which lay the foundation of the asthmatic tendency. Perhaps the largest group of causes of this kind are conditions affecting the vascularity of the bronchial tubes, such as measles, hooping-cough, bronchitis. In many cases the sole predisposing cause appears to be some inherited peculiarity.

E. Diagnosis of Asthma.—It is of the utmost importance to be able to recognize Asthma with certainty, because there are several diseases with which it might be, and often is, confounded, and, because the treatment of these diseases and of Asthma is of the most opposite kind.

The three forms of dyspnœa with which Asthma is apt to be confounded, are bronchitis, emphysema, and heart disease. From bronchitis, Asthma may be distinguished by its sudden access, and often equally sudden departure, by the absence of cold as a necessary cause, and frequently by the absence of expectoration and of moist sounds. Moreover, when expec-

toration does occur, it is of a different kind; in bronchitis it is often purulent, in pure Asthma never. Again, the action of remedies distinguishes the two dyspnœas: the intensest asthmatic dyspnœa will often suddenly, almost instantaneously, yield to certain remedies; in bronchitis this is not the case; if the dyspnœa is severe, so as to be at all commensurate with Asthma, it always takes some time to subside.

From emphysema, Asthma may be distinguished by the paroxysmal character of the dyspnœa, by its violence, and by the absence of any dyspnœa whatever in the intervals. In emphysema, the cause of the difficulty of breathing is organic and unchanging, and, therefore, dyspnœa is never completely absent, and varies in amount only in proportion to the degree to which respiration is taxed. The presence or absence of the physical signs of emphysema will also, of course, materially aid the diagnosis.

The dyspnœa that Asthma is the most apt to be confounded with, and which it most resembles, is that of heart disease. The two resemble one another in that they are both paroxysmal, both intense, both apt to occur at night, both compatible with organic soundness of lung, and both intolerant, though not exactly in the same way, of the recumbent position, of exertion, and of sleep; moreover, in both of them the respiration may be perfectly normal between the attacks. It is not wonderful, therefore, that with so many points of resemblance, the two should sometimes be confounded. There is, however, no real difficulty in distinguishing them. In cardiac dyspnœa there is generally an absence of the characteristic signs of narrowing of the bronchial tubes, universally present in Asthma, such as wheezing, prolonged expiration, suppression of respiratory murmur, &c. The length of the attacks, too, is different, the asthmatic paroxysm being commonly longer than the time reached by an attack of cardiac dyspnœa.

F. Prognosis of Asthma.—This varies greatly in different cases, in some being unqualifiedly favorable, in some unqualifiedly unfavorable, and in some doubtful; it is principally influenced by the following considerations:—

1. *The presence or absence of an organic cause.*—If the Asthma is manifestly dependent on some organic cause, in its nature irremediable and irremovable, it is manifest that the resulting Asthma must be itself incurable. If, for example, it depends upon inveterate bronchitis it is clear that all treatment can be merely palliative, and that a final cessation of the Asthma can never be expected. If, on the other hand, the circulatory and respiratory organs are found to be perfectly sound, then, *quoad* this circumstance, the prognosis is favorable; for, though the absence of organic disease does not make the final cessation of the Asthma certain, it makes it possible; in other words, the absence of organic disease makes the prognosis negatively favorable though not positively so.

2. *Age* has great sway in influencing the prognosis of Asthma; the younger the individual the more probable is ultimate recovery: an asthmatic child of ten will probably lose his Asthma, an asthmatic man of forty will probably not; an asthmatic man of sixty you may say, will certainly not; in an asthmatic youth of twenty it would be difficult to say, as far as the circumstance of age goes, on which side the probabilities would lead. The reason for this fact appears to be, that Asthma has much more commonly an organic basis in advanced life than in early life, that the tendency of Asthma to lay the foundation of organic change is much greater in advanced life than in early life, that the loss of a constitutional peculiarity is much less probable in advanced life than in early life.

3. *The frequency and severity of the attacks* very much influence the prognosis; for, if the attacks are very severe and very frequent, the lungs are unable to recover in the intervals from the injuries inflicted by the attacks, and certain organic changes are probably and speedily induced. If, on the other hand, the attacks are light, and the intervals between them long, the lungs are able perfectly to recover from the temporary derangement produced in them by the paroxysms; and such a case may go on for an indefinite time without the development of any organic changes.

4. The state of the patient in the intervals is of great importance in influencing our views as to prognosis. If the lungs and heart appear to be anatomically and functionally sound, if the breathing is perfectly natural and free, and there is no wheezing, cough, or expectoration in the intervals, the prognosis is infinitely more favorable than if the reverse is the case. Persistent difficulty of breathing in the intervals of the attacks is a very bad prognostic sign; indeed, I think the state of the respiration in the intervals of the attacks is of more importance than either their frequency or severity.

5. Lastly, the history of the case often greatly influences our prognosis, because the past often implies the future; if we find that the tendency of the case—the direction it appears to be taking—is towards an alleviation of the symptoms, the attacks becoming lighter or less frequent, or in any way mitigated, we have strong warrant for a favorable prognosis;

If, on the other hand, the attacks have been becoming more frequent, more easily induced, more violent, or protracted, or in any way aggravated, then a favorable issue becomes exceedingly improbable.

G. PATHOLOGY OF ASTHMA. — Our views respecting this must be greatly influenced by our views of the immediate condition in Asthma. My belief is that the immediate and essential condition of the asthmatic paroxysm is a state of contraction of the bronchial tubes. What proof have we of this? In the first place, the sudden induction and remission of the asthmatic paroxysm is consistent with its depending on muscular spasm; in the second place, there is abundant proof that the air in the lungs is locked up, and can neither be got in nor out: there is evidently plenty of air in the chest, percussion is even hyper-resonant; the patient is as unable to drive air out as to draw it in, can neither inspire nor expire, cannot discharge breath enough to whistle or blow out a candle, or blow his nose. The muscles of respiration tug and labor to fill and empty the chest, but the chest walls remain almost immovable: the inspiratory muscles cannot raise them, the expiratory cannot depress them. On listening to the chest we find corroborative evidence of the stagnation of the air. The respiratory murmur is in a great degree lost. This absence of respiratory sound, accompanied by violent respiratory effort, is one of the most striking and suggestive of the facts of Asthma. How can we explain it, except by supposing that there is some bar to the ingress and egress of air; and what can this bar be, unless it is spasm of the bronchial tubes? It cannot be inflammatory thickening of the mucous membrane lining them; for the sudden, almost instantaneous, establishment and remission of the dyspnœa is incompatible with this. It cannot be mucous plugging of the tubes; for the attack will often come and go without any expectoration whatever. But we have still more positive and precise evidence of circumscribed narrowing of the air-tubes in the musical sounds that are present in asthmatic breathing. This symptom has all the certainty and precision that characterize physical phenomena, and shows that the air-tubes are the seat of constrictions that throw the air passing through them into vibrations, and convert them into musical instruments; and since these musical sounds are multitudinous the points of constriction must be many; and since they are constantly varying in locality and character, the constrictions of the tubes must be undergoing similar change. Lastly, the effects of remedies and their nature tell the same tale, and point to muscular spasm as the immediate essential condition. The most powerful remedies of Asthma are what are called cerebro-spinal depressants, such as emetics, tobacco, &c.—remedies whose direct effect is to relax muscular spasm.

If, then, the immediate condition is muscular spasm, the presumption is, that the primary and essential condition is an affection of the nervous system; with very few exceptions we may lay it down as a rule that perturbed muscular action points not to the muscular system, but to the nervous. What proof have we, then, that the nervous system is involved in Asthma? Some of the most striking proofs of this are derived from the nature of the causes of Asthma; and many of these not only show that the nervous system is the real seat of the morbid action, but they show also what portion of the nervous system is involved. The most numerous of the causes of Asthma are what may be called respired irritants, noxious materials of whatever nature contained in the inspired air. It is manifest that these can only be appreciated by the perceptive nerves distributed to the bronchial mucous membrane, and that they can only give rise to bronchial spasm by the irritation which they produce being propagated to the bronchial ganglia, and by them reflected to the motor filaments distributed to the muscular wall of the bronchial tubes. This, then, is the nervous circuit involved in these cases—extremely short, but still a distinct nervous circuit. In other cases an undigested meal will produce Asthma; here the nervous circuit is longer, and involves the gastric branches of the vagus as its afferent portion, and the pulmonary as its efferent. In other cases a loaded rectum, or uterine irritation, may be the cause of the paroxysm; here the circuit is still longer. In other cases, the sudden application of cold to the surface may at once induce bronchial spasm; here the circuit involves the cerebro-spinal as well as the ganglionic system of nerves. In other cases, some sudden emotion may at once throw the patient into a paroxysm of Asthma; here there is no true circuit, no reflection, but the stimulus is propagated direct from the centre to the periphery.

There are other circumstances that point equally clearly to the nervous nature of Asthma. The action of many remedies is not explicable on any other hypothesis. For example, emotion will not only cause Asthma, but it will cure it, and in the most sudden and complete way. Nervous stimulants, such as coffee, strong forms of alcohol, &c., are very powerful remedies; and nervous sedatives, such as stramonium, are among the best known and most efficient of our means of relief.

The view that the nervous system is essentially engaged in the asthmatic state, does not negative the fact that the foun-

dation of Asthma may be laid, and the asthmatic tendency determined, by something organically affecting the respiratory organs. All who are familiar with Asthma must have observed that it frequently takes its origin in childhood from measles, or hooping-cough, or bronchitis. Now, these are diseases disturbing the vascular condition of the bronchial mucous membrane; but a morbidly vascular mucous membrane is a morbidly sensitive mucous membrane, and, therefore, a mucous membrane whose irritation is likely to produce, through the nerves supplied to it, spasm of the muscular wall of the tube which it lines, just as we see spasmodic stricture of the urethra apt to occur in gonorrhœa.

H. Treatment of Asthma.—There are two things that the physician has to do—two problems suggested to him—in the treatment of any case of Asthma: one is to relieve the attacks when they occur, and the other is to prevent their occurrence; in other words, one is the treatment of the paroxysm, and the other the treatment of the disease. Of these two the latter alone deserves the name of curative treatment, the former is merely palliative. I shall first consider the treatment of the paroxysms.

On being summoned to a patient in an asthmatic paroxysm, the first thing that the physician has to do, is to remove any exciting cause, and to place the patient in the most favorable condition. If an offending meal or some error in diet appears to be the exciting cause, an emetic should be at once given; if a loaded rectum, a purgative should be administered, &c.; if smoke or dust, or any vegetable or animal emanation, is the cause of the attack, this cause should be immediately removed; free ventilation should be secured, and the crowding and officious ministrations of friends should be forbidden. The sufferer should not be made to speak; everything should be done for him, and done without the necessity of his requesting it. The position in which he is placed will make a great difference to him, not only to his comfort, but to the abatement of his symptoms. The best position to put the asthmatic in, as a rule, is sitting in a chair and leaning forward on something in front of him, so as to raise his shoulders. Sometimes he will find leaning on something in a standing posture the easiest position; but, generally, standing involves too much exertion. Sitting at a table and leaning forward so as to rest the elbows on it, or resting the elbows on the arms of an armchair, or kneeling up in bed, or kneeling on the floor and resting the elbows on the side of the bed or a chair, are positions that give the greatest relief.

I have known one patient who elevated the shoulders by placing under them two short crutches which rested on the side of her chair. The great object is, in some way or other, to raise the shoulders, and the advantage of doing so by these mechanical means is that it saves the muscles the labor and fatigue of so doing. The reason why it is necessary that the shoulders be in some way raised, is that the inspiratory muscles passing from the shoulders down to the chest wall may act with greater power as elevators of the ribs.

Having then placed his patient under the most favorable circumstances for the abatement of the spasm, the physician has next to select the remedies that he will employ. This selection is very much influenced by the patient's experience. From the constitutional nature of Asthma, and its persistent character, it is rare to see a patient in his first attack. The great majority of asthmatics that one sees are habitual sufferers from their disease, and have generally some knowledge, often a very accurate one, of the remedies that best suit their case. But in this there is the greatest variety, and the experience of one person would be no guide to the treatment of another. Indeed, the behavior of Asthma to remedies is marked by the most extraordinary uncertainty and caprice; that which is the most valuable in one case is inert in another; in some there are many things that will give relief,—the only question being which is the quickest and the most complete; in other cases all remedies are alike powerless.

The remedies of the asthmatic paroxysm may, I think, be divided into three classes:—Direct Depressants, Sedatives, and Stimulants.

Depressants.—I have already spoken of the value of emetics for the purpose of evacuating the stomach of unwholesome or undigested contents. No doubt, in this way, by the removal of an exciting cause, the paroxysm may often be relieved. But emetics also relieve Asthma very efficiently as depressants, quite independently of their emetic action. I may be asked, "How do I know that it is as depressants, and not as evacuants of the stomach, that emetics give relief?" For two reasons: first, because they will give the same relief when the patient has an empty stomach; and secondly, because the relief comes on when the first sense of nausea is experienced, and before any vomiting has taken place; in a moment, at the first sensation of faint sickness which gives warning of the approach of vomiting, the spasm will suddenly yield, and the patient pass into a state of tranquillity and ease. If this condition could be produced and kept up without giving rise to vomiting I think it would be just as well for the asthmatic, provided that

the paroxysm was not kept up by a loaded stomach. The emetic that I have most commonly given is ipecacuanha powder, in twenty-grain doses; it generally acts in a quarter of an hour; a tumbler of warm water should be taken before its first action and after each act of vomiting. I have lately thought the ipecacuanha wine preferable to the powder, from its action being sooner over; the powder, I think, sometimes sticks to the surface of the stomach, and keeps up a teasing and lingering retching. With a view to produce nausea, short of vomiting, I sometimes give ipecacuanha lozenges, directing the patient to take one at short intervals till a slight sense of nausea is experienced, and to return to the lozenges as soon as this passes off: this plan is often quite successful.

Tobacco.—As this is one of the most powerful depressants, so it is one of the most powerful remedies in Asthma. In those unaccustomed to its use, and in whom, therefore, its full physiological effects are most developed, it is almost impossible for the asthmatic paroxysm to resist it. If I were asked to name a remedy on which I should place the greatest reliance in subduing the most obstinate asthmatic spasm, I should say tobacco in those unaccustomed to it. I believe that the death-like collapse that it produces is something before which Asthma must go down. From this potency it is in obstinate cases a most valuable remedy, but it has three disadvantages: in the first place, it is peculiarly distressing—the sensation that it produces is as near like the worst form of sea-sickness as possible, perhaps a little worse; in the second place, it sometimes produces alarming, if not dangerous symptoms; and in the third place, it is in a large class of asthmatic patients, adult males, inoperative in consequence of its habitual use.

It should always be given with great care, and tentatively, especially to those who have never before tried it; and the mildest forms should be chosen. Like ipecacuanha, tobacco relieves Asthma independently of the vomiting it may produce. By careful management and experience, smoking may be carried just far enough to give rise to a sense of faintness and slight nausea, without its passing on to vomiting at all.

Another remedy, very efficacious, very commonly used, and very like tobacco in its action, is the *Lobelia inflata*. I find that different authorities have a very different estimate of the value of this remedy; and I myself am conscious that I have a much higher opinion of it than I had some years ago. The fact is, I now give it in a way that I believe tests its power much more fairly than the ordinary way in which it is administered; this is the plan recommended by Dr. Elliotson, of giving it in gradually increasing doses at short intervals, till its physiological effects are manifested. I generally start with twenty minims of the ethereal tincture, and tell the patient to repeat the dose every half-hour, making it five minims larger each time, till some slight nausea and feeling of faintness is experienced. By this plain the efficacy of the drug is fairly tested; by the ordinary plan of giving the patient fifteen or twenty drops every three or four hours, its value is not tested. I have on many occasions known as much as forty or fifty drops reached before any feeling of nausea was produced, and before the Asthma was relieved; but with the nausea came the relief. I should never feel the slightest confidence that Lobelia was valueless as a remedy, in any given case in which it had been administered in the ordinary way. I have many times, by changing the method of its administration, obliged patients to reverse their verdict of it. When a patient has found out his maximum dose, I advise him on the next occasion to start with that dose; it saves him the trouble and loss of time of gradually working up to it.

Sedatives.—The relief obtained in Asthma from this class of remedies, no doubt depends on their rendering the nervous system less irritable and less susceptible to sources of disturbance, and the presence of sources of irritation less likely, therefore, to issue in the production of spasm. Some of them appear to act locally, on the nervous system of the lungs alone, but most of them on the general nervous system. Those that experience has shown to have the most value in Asthma, are: tobacco, in sedative doses, stramonium, datura tatula, belladonna, conium, hyoscyamus, ether, and chloroform; and lastly, the fumes of burning nitre paper may be mentioned in the same category.

Tobacco, smoked in the ordinary way, is certainly of great service to many asthmatics. By its habitual use, they keep themselves much freer from attacks than they are without it, and are constantly able to check the asthmatic tendency when it shows itself. If at any time a little wheezy they resort to their pipe or cigar, and soon experience its soothing effects; the breathing quiets down and becomes clear, and they are soon themselves again. Many asthmatics have told me that they are sure that, if they left off smoking, their asthma would soon become troublesome; and that, as long as they smoked, they may do many things with impunity that, without their tobacco, would be sure to bring on their symptoms. But, while very useful in this way, I do not think that it is equal to the sub-

duing of a severe attack, unless pushed to what may be called a poison dose; and then it ceases to be a sedative and becomes a depressant.

The two species of datura—the *D. stramonium* and the *D. tatula*—certainly deserve a very high place among the remedies of Asthma: they are however of very variable efficacy in different cases; and that is the probable reason why different observers entertain such different opinions of their value; some thinking very highly of them and some regarding them as next to worthless. I find, in my own practice, that in the majority of cases they do some good, and in many are the one sovereign remedy. I have had some cases that I may say have been completely cured by them, and others in which, though they have not effected a final cure, the disease has, under their continual use lost all its horrors. It does not, however, do to speak of them together as if their operation was always alike. In most cases they differ in their effects; in some, one being the most powerful, in some the other. In some cases one will be completely successful, while the other is perfectly inert. Seeing that they not only belong to the same class of remedies, but are merely different species of the same genus, this diversity of their action is very extraordinary. Of the two, I think the tatula is the more powerful. I have, however, met with many cases in which it has been powerless where the stramonium has always given relief. They may be given in two ways—either by smoking the leaves in a pipe or cigar, or else internally as tincture, or extract; though I doubt if in these two ways exactly the same agent is given. I doubt whether the combustion in smoking does not produce something that did not before exist, as in the case of tobacco-smoking. Nevertheless it is certain that in both ways the daturas are of value. When smoked they are best used with one of two objects, or both—either habitually, at stated intervals—say night and morning—with a view of keeping off the attacks and making them less likely to come on; or having them always in readiness to fly to on the least approach of an attack, so as to check it at once and prevent its development. This latter plan often answers very well. The patient fills his pipe and puts it by the side of his bed over-night, with the means of lighting it, and when he wakes towards morning with the first traces of his Asthma upon him, he at once lights it and smokes away, the dyspnœa subsiding with each whiff that he draws; so that in a few minutes he is able to put it out, and lie down and go to sleep again. This is the story of many asthmatics, and they would rather not go to bed at all, than do so without their stramonium by their side. Internally I often give something with advantage, the extract of stramonium in a pill. I give it in a quarter of a grain dose generally, combined with an eighth of a grain of belladonna, and two or three grains of extract of conium. This pill, taken at bedtime, has sometimes the effect of preventing the development of the attack during the night; it guards the patient through the critical time, and tides him over it. But I must say that I think stramonium taken internally has not that general utility that it has when smoked, and I have known it quite useless in patients, who, when they have smoked it, have found it very efficacious.

Conium and *hyoscyamus* are sedatives that doubtless have some value in Asthma, and are very commonly employed; but, in the majority of cases, the relief they give is but slight and temporary, and they are not remedies on which reliance can be placed. I think I have seen them most efficacious when given in combination, in the form of tincture, with chloric ether. Of *belladonna* I have made an extensive trial since this article was first written, and I am satisfied of its great value in many cases. In not a few its employment has resulted in a complete and apparently permanent cure. I generally give it in the form of the tincture at bed-time, increasing the dose each night, until the asthmatic tendency ceases to show itself, or until, without such result, the physiological effects of the drug are well marked. [Huchard, after careful experimentation,[1] asserts that the most intense paroxysms of Asthma may be promptly relieved by hypodermic injections of morphia. He describes the influence of this remedy, also observed in other forms of dyspnœa, in the following words: "Morphia makes one breathe freely."—H.]

Chloroform.—There is, perhaps, no disease in which the wonderful power of chloroform is more shown than in Asthma. I have never seen a spasm that it failed to subdue. The worst of it is that its operation is often evanescent;—as soon as its physiological effects pass off, its remedial effects disappear too. This, however, is by no means always the case, the cure frequently remaining permanent after the stupefying effects of the agent have quite passed off. And even where the Asthma does return, it is no slight thing to be able to suspend its horrors for a time, and to give the sufferer a short respite. It has certain disadvantages that would induce me not to place it among the first remedies that I would try, but to keep it rather as a last resort

[1 London Medical Record, March 15, 1879.]

when everything else has failed:—in the first place, it is, as we know, not entirely devoid of danger; in the second place, it is often not safe to trust it in the patient's hands or those of his friends, and, therefore, can only be used in the presence of the medical attendant; in the third place, its habitual use is very apt to generate a liking for it, and to pass into a kind of dram-taking. I have seen two or three painful cases of this kind that make me always unwilling to begin its use, just as I am unwilling to begin the habitual use of opium in any chronic malady. In those rare cases in which Asthma never comes on during sleep it is of great value as inducing sleep. I have known ten drops in this way cure an attack and give the patient a good night, simply by just putting her off to sleep. I do not think that any amount of asthmatic dyspnœa is any reason against giving it, or constitutes in any degree an element of danger—supposing, that is, that the Asthma is pure, and that the dyspnœa is neither cardiac nor bronchitic. In either of these cases the dyspnœa, being organic and not of a nature which the chloroform would remove, would constitute a serious embarrassment: whereas the asthmatic dyspnœa would cease to exist, and therefore cease to be any source of danger, in just such proportion as the influence of the chloroform was established.

Nitre Paper.—This is, perhaps, now one of the best-known and best-established remedies of Asthma, as it is one of the most uniformly successful. So generally is it efficacious, that it is always a matter of surprise to me when an asthmatic tells me that it does him no good. I am not certain of the category in which I ought to place it, and I class it among sedatives, and am inclined to think that it acts as one, chiefly on account of the strong soporific influence that it exercises. It affects not only the patient in this way but the bystanders. On the very day that I am writing this, a lady has complained to me that she finds it almost impossible to administer the nitre fumes to her husband, on account of the irresistible sleepiness with which it overwhelms her; and I have mentioned in my work on Asthma the case of a lady who burnt the paper every night of her life in bed, but always had to wake her husband up, as soon as the fumes had relieved her breathing, because they made her so helplessly drowsy that she feared she might fall back while the paper was still burning, and set the bed on fire; she always *did* fall back asleep before the process was over, and her husband always had to take charge of the embers. What are the exact products of the burning of nitre paper I do not know, nor of those products what may be the remedial one, or ones. This is a subject that still waits investigation. The papers may be made by the patient (by dipping ordinary blotting-paper, white or red, into a warm saturate solution of saltpetre), or bought at any chemist's. The papers should be kept in a dry place, so as always to be fit for use. When employed, a piece about six or eight inches square should be torn off and lit at one corner. As the ignition fizzes along the edge of the paper, white fumes arise which are to be inhaled. I do not think it necessary or advantageous that the actual smoke itself should be drawn into the chest, but the air in its immediate neighborhood which is impregnated with it. It is a good plan to burn the paper in a small room, or confined space, so as to get the air thoroughly charged with the fumes: a cupboard, or closet, or four-post bed, with the curtains close drawn, answers very well; I have seen a patient make use of a large carriage umbrella for this purpose. There are two ways in which the paper may be advantageously used:—one habitually at stated periods, as a preventive, as, for example, every night and morning; and the other when the Asthma shows itself, with a view to its immediate relief. By using it in the former way, patients may often prevent the development of any attack for a long period. For example, many persons burn the paper every night in their bedroom on going to bed, and retire to rest with confidence and with the certainty of immunity through the night; whereas, if they go to sleep without first impregnating the air of their bedrooms with the nitre fumes, they are as certain to be disturbed with their Asthma. Others, with a view to its use in the latter way, always carry some nitre papers about with them wherever they go, and if their asthmatic symptoms appear burn a piece, and in a few minutes are relieved. Such patients never go to bed at night without having some of the paper by their bedside, that, if their Asthma disturbs them at night, they may immediately resort to their remedy. So rapid are its effects often in these cases, so complete is the relief, and so drowsy do the combined effects of the previous dyspnœa and the nitre paper render the patients, that they have not time, as I have already mentioned, to put the still burning paper in a place of safety before they fall back asleep.

Stimulants.—This is a class of remedies whose action is very different from, one may almost say opposite to, the action of those I have just mentioned, but which nevertheless exercise a most powerful influence over the asthmatic state. Among these, *coffee* is perhaps the best known, and the most generally efficacious. I find, in the majority of cases of Asthma

that come before me, that coffee has been tried, and that it has given relief. It should be made as strong as possible, *café noir*, taken as hot as it can be swallowed, without either milk or sugar. It should also always be taken upon an empty stomach; coffee taken with food not only does no good, but does positive harm, by impeding the process of digestion. I have known more than one case, as I have mentioned elsewhere, in which coffee made in the ordinary way, and taken immediately after dinner, had a strong tendency to induce Asthma, although, taken in the way I have above described, it had a very powerful beneficial influence.

Alcohol, in its various forms, is another remedy of this class, that my experience during the last few years has induced me to think highly of as a remedy for Asthma. In many cases it does not do much good, but in some it has a most powerful effect, and these I have noticed are frequently cases in which all other remedies have failed. In such cases I should certainly, if for this last reason alone, recommend its use; in any case where other remedies answered I do not think I should, on account of the many manifest objections there are to the habitual use of the stronger forms of alcohol. I have observed that it seems of little use unless given hot and strong—about half spirit and half boiling water; this circumstance seems to make more difference than the kind of spirit, or the actual quantity taken. Some asthmatics prefer brandy, some whisky, some gin; but in all, however small the quantity of spirit taken, it must be hot and concentrated. The worst of this remedy is that it is so apt to become habitual, and to require to be given in larger and larger doses.

While speaking of stimulants, I think I ought to mention the curious and striking remedial effect that sudden emotion has in Asthma. There is nothing that suspends the asthmatic state so completely and so immediately. At once, without any gradual subsidence, the patient will pass from the most violent paroxysm to a state of perfectly free and unimpeded breathing. And this is the case not only in emotional temperaments, but in all kinds of people, of both sexes, and at all ages. The emotion may be pleasurable or painful, but it must be intense, and I think it acts more powerfully if it is sudden. Did the length of this paper permit, I might relate some very curious and interesting cases in illustration of this point, but I must content myself with merely mentioning the fact. And surely, if it were wanted we could not have a more striking or convincing proof of the nervous nature of Asthma; I should myself want nothing more to establish this theory of the disease than this single therapeutical fact.

I have hitherto been speaking exclusively of the treatment of the paroxysms. But a very important part of the treatment of Asthma, indeed the only radical treatment of the disease, is the treatment in the intervals—that which is directed to the prevention of the attacks altogether. This is the only treatment that deserves the name of curative; the treatment of the paroxysms is but palliative.

There are, I think, three forms of treatment that have for their object this final cure of the disease by the prevention of the paroxysms. The first, the treatment by air—that is, by locality; the second, dietetic treatment and the regulation of the digestive organs; and the third, treatment by the avoidance of the excitants of Asthma, such as hay, animal emanations, &c. These different plans of treatment are applicable to different classes of cases; but if we examine them closely we shall see that they all really belong to one kind of treatment, that they all essentially consist in the avoidance of the provocatives of the attacks; their applicability depending upon what, in each particular case, is the special exciting cause.

Treatment by Air.—It has long been known to those who have either observed or experienced Asthma, that locality exercises a most remarkable control over the disease—that there are certain airs in which the asthmatic cannot breathe, and that there are certain other airs in which he enjoys a sure immunity from his malady; that, in fact, his being an asthmatic or not depends entirely on where he lives: if he lives in the one place, he is constantly suffering, but he might live twenty years in the other and never have an asthmatic sensation. There are some circumstances with regard to this curious fact that are constant, and worthy of note. In the first place, the effect is *immediate;* let the asthmatic be suffering ever so severely, he no sooner arrives at the air that, in his case, is curative, than he is at once relieved. In the second place, the effect is invariable for each particular case; there is nothing irregular or haphazard about it; the same thing may be repeated twenty times, and always with the same result; so much is this the case that the asthmatic knows he may calculate on it with the greatest safety. I have mentioned elsewhere the case of a gentleman who, let him be suffering ever so much at Cambridge, would accept an invitation to a dinner party in London, knowing that as soon as he arrived in town he would be well. And this may go on for a lifetime, and is as noticeable in the production as in the cure of Asthma. A person may have an attack of Asthma on going to a particular place; twenty years after he may revisit that place, and he will again be attacked. Again, the effect is

permanent; as long as the patient resides in the curative air, he is free from his disease, if it is for the rest of his lifetime, but only so long as he resides there; for the remedy does not eradicate the asthmatic tendency; the patient has only to be exposed to the same influences as before to have all his old symptoms return upon him in their original force, and that after any lapse of time during which they have been suspended. Another noticeable point in most cases is the inscrutable character of the atmospheric peculiarity on which this influence depends, and very often its extreme slightness: the fact only is known that in such an air the Asthma never appears; but what is the peculiar character of that air, or in what respect it differs from another in which the patient cannot breathe, neither the asthmatic, nor his friends, nor his medical advisers, can even guess.

But while the effect of locality is constant for each particular case, the experience of one case is not the slightest guide for another; on the contrary, there is the utmost diversity and contrariety with regard to this circumstance in different cases. The air that is a certain cure to one is death to another. One patient is best in the country, one in town; one is best in an elevated position, one in a low one; one is relieved by a relaxing air, one by a bracing; one is best at the seaside, one inland. But though there is this uncertainty and irregularity, yet on the whole, on the average, there are certain rules as to what is curative. Thus, in the great majority of cases, an urban air is the air that cures, and of a city air that seems to be the best which is the most urban—the densest and smokiest. As a rule the air of a low situation is better than that of a high one, and a relaxing air than one that is bracing. In some cases there is one place, and only one where the Asthma manifests itself. In such cases the circumstance has generally been discovered by accident—the asthmatic has suddenly been seized, soon after his arrival at some place that he never visited before, with strange and alarming symptoms which have turned out to be Asthma. These symptoms may never again appear except on a return to the same locality. It is, however, much commoner for there to be many places where the Asthma is apt to occur, and only one, or but few in which the asthmatic tendency seems to be in abeyance.

Treatment by food is the sovereign and final treatment of all those cases in which the Asthma is produced, and only produced through the stomach. There are many cases in which a late dinner, or a supper, is sure to bring on an attack, but in which nothing else will. In such cases the patient has only to abstain from food after an early dinner, and he will see no more of his Asthma for such time as he keeps up such abstention, if it is for the term of his natural life. Such a person may cease to be an asthmatic at pleasure—that is, he ceases to be an asthmatic *in esse*, not *in posse;* for immunity so obtained does not destroy the asthmatic tendency: let him at any time break through his rules and his Asthma will immediately reappear.

Treatment by the avoidance of special provocatives is, as I have already mentioned, but the application to other cases of the same principle as the treatment of peptic cases by dietetic rules. Some patients always have Asthma brought on by hay, some by the smell of flowers, some by emanations from particular animals, such as cats, or dogs, or horses. Such persons have merely to keep themselves out of reach of the especial exciting causes, and they may elude their disease for any length of time. The radical treatment of bronchitic Asthma belongs to the same category, and consists essentially in the treatment of the bronchitis. Place such a patient under such circumstances as preclude the bronchitis, and with the cause you preclude the result; send such a case to Australia, and there is an end of his Asthma, because there is an end of his bronchitis.

PHTHISIS PULMONALIS.

By John Hughes Bennett, M.D., F.R.S.E.

Definition.—By the term Phthisis or Consumption (from φθίω to waste or consume) has been understood from the earliest times a disease characterized by wasting or emaciation of the body. The cultivation of morbid anatomy having determined that this condition was frequently dependent upon the deposition of

little grains or nodules of a peculiar substance in the lungs, these received the name of tubercles. Thus the terms tubercle, tubercular disease, or tuberculosis, gradually came to be regarded as synonymous with Phthisis, which may now be said to comprehend all kinds of disease essentially connected with or dependent upon pulmonary tubercle.

It is this important morbid condition which we propose to describe in the present article, under the general heads of Pathology, Symptoms, Diagnosis, Prognosis, and Treatment.

I. Pathology of Tubercular Phthisis.

The pathology of Phthisis involves a consideration of the histology, chemistry, and general pathology of tubercle—of the morbid anatomy of the disease—of its causes—of its natural progress—and of the theory of its production.

Histology, Chemistry, and General Pathology of Tubercle.—The term tubercle literally implies a little swelling, and in this sense it still serves to distinguish a class of skin diseases. As applied to the peculiar deposits so frequently found in the lungs and other organs, it now means not only those products when they present a tubercular form, but when they are infiltrated in masses, or exhibit appearances wholly opposed to the original signification of the word. At present, by tubercle is understood a peculiar morbid deposit, sometime gray, but more frequently of a yellowish color, varying in size, form, and consistence, which sometimes softens, and causes ulceration in the surrounding textures, but which at others dries up, becomes cretaceous or calcareous, and produces induration and cicatrization.

The ultimate structure of tubercle varies according as it is soft or hard, or as it has been recently or for a long time deposited. If we mix a minute fragment of yellow, tolerably soft or cheesy, tubercle with a drop of water, and crush it between glasses, so that it may be thoroughly broken up, and capable of being examined with a magnifying power of 250 diameters linear, it may be seen to consist of a number of irregular-shaped bodies, and of numerous molecules and granules. The bodies are called *tubercle corpuscles*, and approach a round, oval, or triangular form. Their longest diameter varies from the four-thousandth to the

[Fig. 15.

Acute Phthisis.—Showing one of the alveoli filled with fibrinous exudation and leucocytes, and some cellular infiltration of the alveolar wall. × 200. (Green.)]

two-thousandth of an inch. They are solid, having a distinct external outline, and have embedded in them generally three or more granules and molecules, varying in size from a point scarcely measurable to the six-thousandth of an inch in diameter. Acetic acid causes partial solution and transparency of these bodies. Ether and alcohol produce little change. Ammonia and liquor potassæ cause them to break down and dissolve with varying rapidity. The molecules and granules differ greatly in various specimens of tubercle, sometimes being very minute, and at others half the size of the corpuscles themselves. Chemically, they may be albuminous and partially soluble in acetic acid—fatty when they are soluble in ether and potash—or mineral when they are dissolved by the mineral acids.

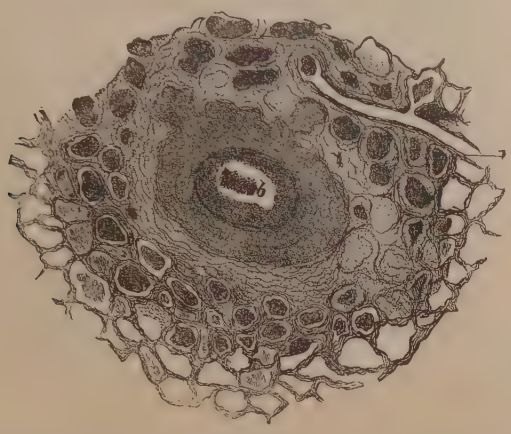
The corpuscular and molecular elements of tubercle are always present, but

in different proportions. Generally speaking, in indurated or gray tubercle there are few molecules, and the corpuscles are so compressed together as to be scarcely distinguishable. On the other hand, in soft tubercle the molecules are numerous, and the corpuscles easily separable. The more tubercle softens and becomes diffluent, the more the relative amount of the molecular element increases.

In chronic tubercle, and especially when it has undergone the cretaceous or calcareous transformation, the elements described become mixed with hard, gritty particles of earthy salts. These are of irregular form and size, and are large and numerous in proportion as the tubercle is more and more calcareous. They are often associated with crystals of cholesterine, and not unfrequently with black pigment granules and masses. When tubercle is converted into a mass of stony hardness, a thin section of it presents an irregular granular appearance, made up of a congeries of minute earthy particles without any distinct form.

Tubercle corpuscles may be associated with pus and granule cells, as well as those peculiar to glandular organs or mucous surfaces. From pus corpuscles they are readily distinguished by the action of acetic acid, which in them causes no

[Fig. 16.



Acute Phthisis.—A transverse section of a terminal bronchus (air-passage) and the surrounding alveoli. Showing the lobulated character of the pulmonary consolidation. *b*, cavity of bronchus containing a little mucus. *v*, a bloodvessel. × 50, reduced ½. (Green.)]

granular nucleus to appear. From the fibre or plastic cells found in recent lymph they may be separated by their irregular form, smaller size, and the absence of primitive filaments. With the granule cell they can scarcely ever be confounded on account of its large size, brownish appearance and granular structure. From gland or epithelial cells they are distinguished by their smaller size and the absence of nuclei. Cancer cells also are at once recognized by their size, transparency, and oval nuclei. The only elementary structures resembling tubercle corpuscles are those constituting the reticulum of cancer and the disintegration of fibro-nucleated growths. The former, although often, even to the naked eye, resembling tubercle, and under the microscope composed of irregularly-shaped nuclei, and numerous molecules, resulting from the histolysis of cancer, are almost always associated with the more recent cell-forms of that growth, while the fragments or presence of fibres serve to distinguish the latter. It should be remembered that all forms of exudation, and many kinds of growth, at an early period of development, present a molecular and nuclear structure throughout, and might by inexperienced histologists be confounded with tubercle. A careful consideration of all the circumstances connected with tubercle, and of the distinctive structures associated with it, however, will seldom deceive the skilful observer.[1]

Tubercle has been made the subject of special chemical analysis by numerous chemists, from which the following conclusions may be drawn: 1. That it consists of an animal matter, mixed with certain earthy salts. 2. That the relative proportion of these varies in different specimens of tubercle. That animal matter is most abundant in recent, and earthy salts in chronic tubercle. 3. That the animal matter consists principally of albumen, occasionally mixed with a small amount of fibrin. Fat also exists to a slight degree, and becomes more abundant

[1 See article on Scrofula, vol. i.]

as a constituent as the disintegration of tubercle progresses. 4. The earthy salts are principally composed of the insoluble phosphate and carbonate of lime with a small proportion of the soluble salts of soda. 5. That very little difference in ultimate composition has yet been detected between tubercle and other albuminous compounds.

From the preceding structural and chemical facts tubercle must be regarded as a morbid product, having a very low degree of vital power, seldom proceeding beyond an imperfect degree of nuclear formation, and having a constant tendency to fatty or mineral degeneration. It assumes four forms:—

1. *Miliary Tubercle*, when the morbid deposit is scattered throughout an organ, or on the surface of a membrane, in isolated grains like millet seeds. Sometimes they are sprinkled indiscriminately throughout a tissue; at others, they are in groups or clusters more abundant in one part than in another. Occasionally they are minute, of grayish-color, semi-transparent, and hard to the feel—the so-called *gray granulations* of Bayle. More

[Fig. 17.

Section of lung from a case of somewhat Chronic Phthisis. Showing the thickening of the alveolar walls by a fibro-nucleated adenoid-like tissue; together with an accumulation of epithelial cells within the alveolar artery. The latter are undergoing retrogressive changes. × 200. (Green.)]

frequently they are of a yellow color, about the size of a millet or mustard seed, and of soft consistence, so that they can be easily crushed between the fingers. In consistence they may vary greatly, being sometimes hard, or, as they are then called, crude, or they may be so soft as to resemble cheese and cream. They may have undergone the cretaceous or calcareous transformation, and still preserve their miliary form.

2. *Infiltrated Tubercle* occurs in diffuse masses, varying in size from that of a bean to that of the entire organ affected. Thus a lymphatic gland, or the lobe of a lung, may present a uniform deposition of the substance throughout its whole extent. Between these two extremes every variety in extent of deposition may be observed, masses being frequently formed by the agglomeration or condensation of miliary tubercle. Like it, also, this form of the deposit may be gray or yellow, crude or soft, and undergo the cretaceous and calcareous transformation.

3. *Nodular and Encysted Tubercle.*—This form of tubercle exists in rounded, isolated masses, varying in size from that of a small pea to a bean. It may present all the characters of the other forms, but is frequently seen to be surrounded by a capsule, more or less dense, of fibrous tissue.

4. *Cretaceous and Calcareous Tubercle.*—This form of tubercle is distinguished by its white appearance, and its putty-like, gritty, or stony consistence.

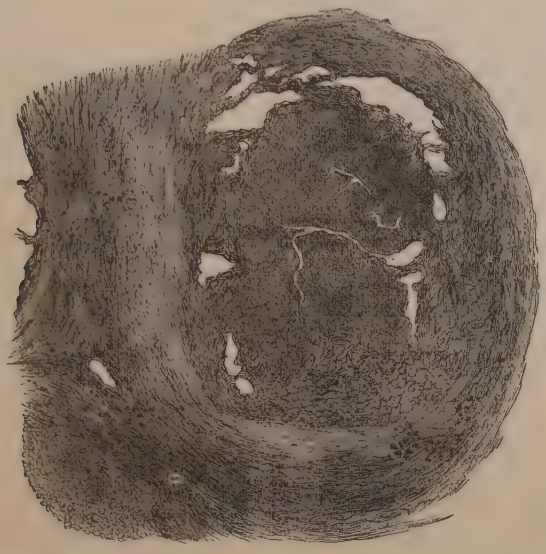
All these forms of tubercle run into one another, and may exist in the same individual, and often in the same organ, especially in the lungs. They indicate no further essential difference in the nature of the deposits than is concerned with its amount and extent, its hardness or softness, its color—whether white, yellow, gray, or black, or its being recent or old—miliary and infiltrated tubercle being generally new, while encysted and calcareous tubercles are always chronic. In the last the animal matter has been absorbed, while the mineral matter remains to form a concretion.

Great discussion has taken place as to whether tubercle is peculiar to any particular elementary tissue, and as to how it is produced. Like all forms of exudation, it may occur in every vascular texture, and readily coagulates in the minute spaces between or outside the textural elements immediately external to the vessels. Of this we may easily be satis-

fied by studying its special histology in various organs.

With regard to its mode of production, tubercular matter is first separated from the bloodvessels as a fluid exudation, forming by its coagulation a molecular blastema. The molecules of which it is composed then aggregate or melt into each other to produce the tubercular corpuseles. These, if compressed together and formed slowly, constitute the indurated dense granulations described by Bayle: but if separated by the soft molecular matter, produce the more common yellow miliary tubercles. The idea that these bodies are invariably the result of

[Fig. 18.



Chronic Phthisis.—Showing the new interlobular fibroid growth surrounding and encapsulating a degenerated and caseous portion of the consolidated lung. × 50, reduced ½. (Green.)]

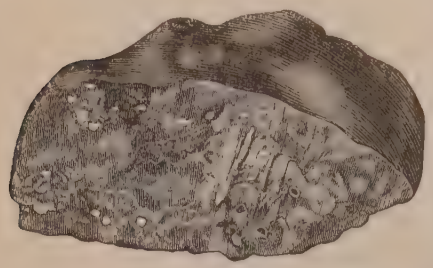
cell-proliferation originates from the erroneous hypothesis maintained by Virchow and his followers, viz., that all morbid products are derived from cells. In their attempts to maintain this view, they have mistaken the occasional enlargement and proliferation of fibre cells in areolar tissue first described by Lebert, as fibro-plastic cells, for tubercular granules, which they describe as the essential elements of the lesion. It is not in the pleura or peritoneum, however, where such fibrous growths are occasionally seen, that the real manner in which tubercle is formed can be well observed, but in the lung, where the disease is most common and best characterized. There, all observation demonstrates that it originates in a molecular exudation, which, in consequence of diminished vital power, seldom passes beyond the nuclear stage of growth. It is this low type of *hysto-genesis* that communicates to the exudation those essential characters which form the foundation of tubercular or phthisical disease.

MORBID ANATOMY OF PHTHISIS PULMONALIS.—Although tuberculization of the lungs is a constant and essential element of Phthisis, it rarely, if ever, happens that the disease proceeds to a fatal termination without affecting other organs. Nothing, also, is more common to find, during the examination of dead bodies generally, than that the lungs are often the seat of tubercle to a greater or less extent, although during life the presence of the disease has never been suspected. So common, indeed, is this lesion, and so many have been the able investigators of the alterations it produces in the various organs of the body, that all the anatomical facts connected with it may be said to be thoroughly known. We shall notice the morbid changes observed in cases of Phthisis in the different parts of the frame, *seriatim*.

The Lungs.—These are the organs in which, according to the researches of Louis, tubercle is sure to be discovered, if it occur in the body at all. This law, though now known to admit of some exceptions, especially as regards tubercular peritonitis, is still so generally true as to be one of the most valuable generalizations ever arrived at in pathological science. To the same distinguished physician we are indebted for another fact of no less importance, viz., that when tubercle occurs in the lungs it attacks the

apices of those organs first. The exceptions to this law are so few as in no way to invalidate its great practical value.

[Fig. 19.



Apex of Lung affected with Tubercular Pneumonia.]

The morbid changes found in the lungs of those who die laboring under Phthisis Pulmonalis vary according as the disease is acute or chronic, as it is advancing or retrograding, and as it is associated with other lesions. In acute cases miliary and infiltrated tubercles are more or less general in one or both lungs. The deposit is generally soft, and frequently diffluent, causing ulceration and irregular anfractuous cavities. The intervening pulmonary texture is often engorged with blood, is more or less pneumonic, while the bronchi are loaded with purulent matter. The acute disease in many respects resembles anatomically gray hepatization of the lung, and like it is more frequently most developed in the lower lobe.

In chronic Phthisis, constituting the vast majority of cases met with, all the forms of tubercle previously described are met with. The tubercle is most abundant at the apex, but may invade the greater portion of one or both lungs. In the latter case, it will most often be observed that one lung is more affected than the other, so that an examination of them displays all stages, either of the onward or retrograde progress of the disease; these, although often associated together in very chronic cases, are so distinctive anatomically as to require a separate description.

The appearances of the lung during the onward progress of the disease are—1. The presence of miliary tubercle to a greater or less extent. 2. The softening of this tubercle, so that it readily breaks down under the finger or a current of water, and forms small cavities or irregular ulcerations communicating one with another. 3. The existence of distinct ulcers, excavations, or cavities, as they are named. These vary in size from a pea until they involve nearly the entire lung. There may be one or several. They may be isolated or anfractuous, that is, communicating with one another. If recent, the internal walls are irregular and rough; but if chronic, the ulcerative process has dissected out the fibrous tissue, leaving irregular bands stretched across the interior, composed of bloodvessels, the bronchi, or indurated fibrous tissue. When very chronic, the interior is lined with a smooth membrane. These cavities may be filled with air and fluids in varying proportions; the latter being viscous, purulent, occasionally sanguinolent, and not unfrequently ichorous, of a dirty-green color and offensive odor. These changes in the lung may be associated in varying proportions with many other lesions to which the organ is subject. Pleuritic adhesions by means of fibrous lymph, are very common; the pleuræ, at the apices of the lung, often being united to each other by a dense, tough substance which renders their separation impossible. Bronchitis, in all its forms and stages, may exist together with more or less emphysema, dilated bronchi, and collapse of the lung. There may be pneumonia or extravasation of blood, involving varying amounts of lung tissue.

There is a disease frequent in coal-miners, called carbonaceous lungs or Black Phthisis, in which there is no tubercle, but a deposition and infiltration of lamp-black or carbon in a finely molecular form, and which gives rise to cavities and disorganization of the pulmonary tissue, also commencing at the apex. It is accompanied by black spit, and is generally fatal.[1]

[Fig. 20.

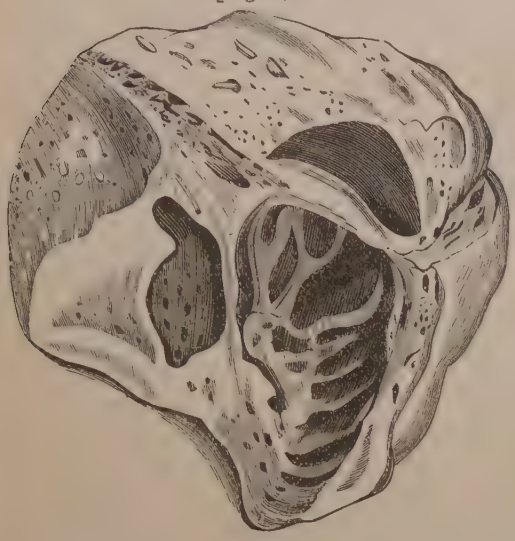
Apex of Tuberculous Lung.]

The retrograde progress of the disease

[1] See the author's Clinical Lectures, 5th edit. "On Carbonaceous Lungs," p. 756.

is characterized anatomically first, by the horny induration and cretaceous or calcareous transformation of the tubercular matter; secondly, by puckerings and cicatrices of the lung tissue; and thirdly, by contractions, loss of substance, and more

[Fig. 21.



Pulmonary Caverns. From a specimen in the cabinet of Dr. Gross.]

or less induration of the organ. It may be observed in about one-fourth of all those who are examined after death in our public hospitals, that the apices of the lungs contain one or more masses, varying in size from a millet-seed to a coffee-bean, of cretaceous or calcareous matter. That these masses were originally tubercle cannot be doubted by those who have had any experience in post-mortem examinations, the more so as in various cases such tubercle, whether in the miliary, infiltrated, or nodular form, may not unfrequently be seen to present the various stages of induration and horny hardness, approaching towards the calcareous substance. Such hard masses if dug out and allowed to dry, indeed, become cretaceous, the animal matter having shrunk away, leaving the mineral substance unaltered. In old persons above seventy years of age, it has been shown by Rogér and Boudet that the presence of these concretions in the lungs increases to the extent of from one-half to four-fifths of all those examined.

If these concretions or masses of indurated tubercle occur at the surface of the lungs, the pleuræ covering them and subjacent tissue are frequently drawn in and puckered. If they occur deeper, they are surrounded by indurated pulmonary texture, more or less tinged of a black color. Occasionally, also, linear and radiating cicatrices indicate the disappearance and closure of pre-existing ulcerations. Sometimes, however, tubercular cavities, instead of closing and forming cicatrices, remain permanently open and filled with air. They are lined by a smooth membrane, and almost always communicate with a bronchial tube. In this condition we discovered, in 1842, in such a case, associated with pneumothorax fungi growing in the infiltrated matter lining the chronic cavities, and have found them frequently in similar excavations since then.[1] At other times the bronchial tubes are permanently dilated, by the contraction and induration of the pulmonary tissue between them. This occurrence, conjoined with the other lesions referred to, gives rise to that condition described by Dr. Corrigan as cirrhosis of the lung.[2]

The various alterations now described may be associated with other lesions, especially chronic adhesions of the pleuræ, emphysema, chronic bronchitis, and dense pigmentary deposits. Not unfrequently it may be observed that whilst one portion of the same lung presents a marked example of the retrograde progress of Phthisis, another portion as decidedly shows the progressive changes. In such a case the former indicates tolerably well the older and more chronic transformations of the pulmonary tissue.

It would thus appear that there is nothing essentially destructive or necessarily fatal in Phthisis, and that in all stages of the disease it may be checked, and enable the individual affected to live many years subsequently, and die of old age or other disorders. Attention to morbid anatomy in recent times is demonstrating that this occurs far more frequently than was formerly supposed, and is due not only in many cases to the spontaneous efforts of nature, but in not a few to the direct interference of art.[3] This latter termination, however, is materially interfered

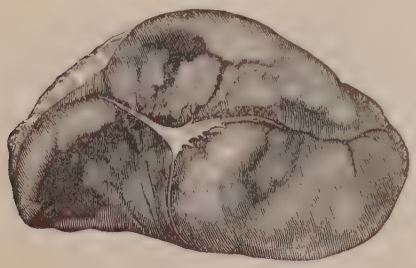
[1] Description of a Cryptogamic Plant found growing in the sputa and lungs of a man who labored under pneumo-thorax. Trans. of Royal Soc. of Edinburgh, 1842.

[2] See Dublin Medical Journal, vol. xiii. 1838; Laennec, vol. i. p. 201; Reynaud, Mémoires de l'Académie, tome 4me; Cruveilhier, Anatomie Pathologique, livraison 32, planché 5, fig. 3; and the author on Pulmonary Consumption, 2d edit. Case 3, p. 57.

[3] See the author's work on Pulmonary Consumption, in which several such cases are recorded, and the post-mortem appearances figured,—Figs. 21 to 26.

with should other organs participate in the disease; and the morbid changes observed in them, therefore, next demand our attention.

[Fig. 22.



Cicatrix at apex of Lung, from arrested Tuberculosis.]

The Pleuræ.—We have already pointed out that during the whole progress of Phthisis the pleuræ, as well as every other part of the lung, are apt to be affected. This, however, may not only be exhibited by adhesions more or less dense, but not unfrequently by the deposition of tubercle in a miliary or infiltrated form, the latter of which assumes a laminar or stratiform character. Effusions and exudations into the pleural cavity may also occur, giving rise to more or less hydrothorax and empyema. Further, the pulmonary pleuræ may be ulcerated and communications take place with tubercular cavities, or with the bronchial tubes, in which last case pneumo-thorax is the result.

The Trachea and Larynx.—The trachea and larynx are very commonly the seat of congestion and ulceration in cases of Phthisis. In the mucous membrane of the former the ulcerations are frequently small, numerous, and round, as if dug out with a small point; at others, they are larger, deeper, and lay bare the cartilaginous rings. In the larynx they are generally irregular, varying greatly in size, and sometimes involving both vocal cords and the whole interior of the organ. Their edges are occasionally studded with indurated tubercles, and sometimes there is thickening with œdema of the cellular tissue, tending to close the glottis. In chronic cases of laryngeal ulceration, which is often called *Phthisis laryngea*, caries and necrosis of the cartilages may occur.

The Bronchial Glands.—It is seldom in cases of chronic Phthisis that the bronchial glands escape being affected with tubercle, which assumes the infiltrated form, and causes in these considerable enlargement. On section they may be almost colorless, but they are sometimes more or less loaded with black pigment.

Heart and Pericardium.—It is very rarely that tubercle is deposited in the heart or pericardium, but when this does occur, it takes place in the nodular form. The heart, however, is very liable to become atrophied, and smaller than usual. In lingering cases of the disease, with extreme emaciation, it may be found after death not larger than a duck's egg. The bulk appears to be adapted to the diminished amount of blood in the body, and the little work it has to do.

Alimentary Canal.—Very rarely ulcerations may exist in the pharynx, but enlargement of the follicles is common. The œsophagus and stomach are organs which are remarkably free from tubercular disease; but, according to Louis, the mucous membrane of the latter viscus is liable to softening, mammillation, and attenuation, in the majority of cases. In the small intestine the glands of Peyer are very liable to enlargements and ulceration, especially in its lower third. The enlargements are owing to the deposition of tubercle in and around the solitary and aggregated glands, often accompanied by considerable redness and vascular congestion. It presents the miliary or granular forms, although occasionally it may exhibit small nodules the size of peas. Tubercular ulcerations of the small intestines are common in the last stages of Phthisis, and occupy the seat of the solitary and aggregated glands. In the first case they are rounded, with abrupt or tuberculated margins, with a yellowish or dirty-gray base. In the latter case they are oval in form, running transversely round the gut, so that they are readily distinguished from typhoid ulcerations, the long axis of which is in the opposite direction. Their margins are smooth, sometimes tuberculated; the base sunk, and covered with a purulent or dirty-grayish substance. Above the ileo-colic valve the ulcers have a tendency to run into one another and produce an ulcerated surface, more or less broad, surrounding the gut. These ulcers occasionally are so deep as to perforate the intestine. Similar tubercular granulations and ulcers may also be found in the large intestine.

Peritoneum.—In rare cases the intestinal ulcerations perforate the bowel, almost always giving rise to fatal peritonitis. Not unfrequently, however, chronic adhesions exist, to a greater or less extent, on the peritoneal surface outside these ulcers, uniting coils of intestines to one another, or to the abdominal walls. Chronic tubercular peritonitis may also occur when the intestines are covered and agglutinated together by coagulated exudation, studded throughout with miliary tubercle. This lesion, though it may accompany Pulmonary Phthisis, may, sometimes, exist as a primary and independent disease.

Mesenteric and other Lymphatic Glands.—These are very liable to be enlarged in Phthisis, when they may present greater or less induration and enlargement, according to the recent or chronic condition of the disease. Tubercle usually is present in them in the infiltrated form, at first yellow, cheesy or soft, afterwards white and indurated, and, in a few cases, cretaceous and calcareous.

Liver.—In children the liver is not unfrequently the seat of miliary and infiltrated tubercles, but in the adult this is very rarely observed. More commonly the organ is enlarged, a result previously supposed to be owing to fatty degeneration, but now known to depend upon a peculiar albuminous transformation known as the waxy disease, from its resemblance to beeswax. In this condition it may be so enlarged as to weigh eight or ten pounds. It presents a peculiar density to the feel, a pale fawn or yellow-brownish color; and on section the cut edges, when held up to the light, are semi-translucent. We were the first to examine this disease of the liver microscopically in 1845, and found the hepatic cells to be condensed together, shrivelled, colorless, and of peculiar transparency, with the nucleus absent, or evidently disappearing.[1] It has been supposed by some to be related chemically to starch, and therefore called amyloid degeneration. But it is never changed blue on the addition of iodine, although we have found that like certain other forms of albuminous compounds it possesses the property of fixing colors, such as the reddish-brown tint of iodine, or the peculiar pigments of indigo and carmine.

Spleen and Kidneys.—Both these organs, like the liver, in early life may become subject to tubercular deposits in the miliary form, which in the adult are very rare. The kidney further is liable to extensive tubercular deposits, causing abscesses, or what is known as scrofulous pyelitis. Like the liver also, it is commonly affected in Phthisis with the waxy degeneration, causing induration and enlargement of its substance, and the same translucent albuminoid degeneration of the cells and vascular elements.

Other textures and organs.—In the foregoing summary we have only shortly alluded to the morbid changes most commonly found in cases of Phthisis. It should be understood, however, that almost every vascular tissue in the body may, under particular conditions, be subject to tubercular deposits in conjunction with the disease of which we are treating, and thus, in special cases, the bones, muscles, the brain and its membranes, skin, the bladder, testes, &c. &c., may be occasionally involved.

[1] See the author's Clinical Lectures, 5th edit. Case clxi. p. 731. Also remarks on the waxy degeneration, Idem, p. 249.

Causes of Phthisis Pulmonalis.—The various circumstances which predispose to Phthisis have been most anxiously investigated. All we can venture to offer in this place is a very general summary of the numerous researches undertaken in connection with this subject.

Age.—Phthisis is not a disease that is common in early infancy or in advanced age. It is more frequent during childhood and youth, although cases may be seen in many persons of middle age, as well as among young children. From the returns of the Brompton Hospital for Consumption, it would appear to be most frequent between the ages of twenty and thirty. Age unquestionably greatly influences the progress of Phthisis, the acute being most common in young, and chronic in elderly persons. We should not forget, however, that Phthisis in advanced life is frequently the termination of a prolonged case, which commenced many years previously.

Sex.—It is generally supposed that Phthisis is more common in females than in males, but this does not appear to be an invariable rule. It is certainly not the case in the Royal Infirmary of Edinburgh, Dr. Home having pointed out that in the years 1833, '34, and '35, 185 cases were males, and only 112 females. The same excess of males laboring under the disease has prevailed in that institution ever since, as in the years 1843 to 1846 inclusive there were—males 356, females 217; and in the latest reports for the year 1865 the numbers are—males 126, females 64.

Hereditary tendency.—Instances are not uncommon in which members of the same family are observed to become affected one after another with Phthisis, on arriving at a certain age. This, however, may depend not so much upon weakness inherited from parents, as it does upon a vicious method of rearing the infants and children of certain families. We have seen the children of many families become phthisical, in whom no hereditary taint could be traced, and have frequently pointed out, in the clinical wards of the Royal Infirmary, that, among the six or eight cases of Phthisis then present, not one could be traced to hereditary causes. Although, therefore, there can be no doubt that weakness in parents is a cause of weakness in the offspring, we are of opinion it is by no means so general or influential a source of Phthisis as is usually supposed.

Vitiated atmosphere.—This has been concluded to be a powerful cause of Phthisis by numerous authors, and there can be no

doubt that the habitual breathing of deoxidized or impure air must greatly impede nutrition. Among the poor there can be little difficulty in attributing its effects to close or overcrowded rooms, in which they work and sleep. Among the higher classes this is not so obvious a cause, although Baudelocque, in support of this his favorite theory of the origin of tubercles, accused them of lying in bed too long, and said that the children slept with their heads under the bedclothes.

Climate.—It is an undoubted fact that Phthisis is more frequent in temperate climates than in very cold or very warm ones. It is by no means common in Russia and Canada, notwithstanding the long continued cold, nor does it prevail among the nations of the tropics. These last, on the other hand, are peculiarly liable to Phthisis on coming to Europe. Some favored spots are stated to be free from Phthisis; among these, it has been recently pointed out by Drs. Macrae and M'Coll, are the islands of Lewis and Mull, among the western Isles of Scotland. Dr. Hjaltelin has informed me that Iceland enjoys a like immunity.

Contagion and Infection.—Several of the older writers were of opinion that Phthisis was contagious and infectious, an opinion still widely disseminated in certain countries, more especially Spain and Italy. We have too frequently seen the death of a phthisical patient in Italian hotels give rise to the most extortionate demands for the pretended destruction of bedding and furniture, all of which should be firmly resisted. It has occasionally been observed that Phthisis in a wife or husband has been followed by the appearance of the disease in the husband or wife. The frequency also with which young women become phthisical after pregnancy has given rise to the idea that they may have been infected by the opposite sex through the uterus. These ideas have received no support from the profession. In 1865, however, it was announced by M. Villemin[1] that the cause of tubercle was a virus, and that he had succeeded in inoculating it in healthy rabbits, by inserting gray granular tubercle below incisions in their skins. These experiments appear to have been carefully performed. They have been successfully repeated by Lebert, and also by others, with varying results. The experiments of Drs. Andrew Clark,[2] Wilson Fox,[3] and Burdon Sanderson[4] have further shown that not only tubercle but a variety of other morbid products, and even local irritation of the tissues, may produce deposits in the glands, lungs, and various organs in rabbits, and especially in guinea-pigs. Thus the introduction of a seton produced them in one case, and pieces of putrid muscle in no less than four out of five inoculations.[1] These facts show that the lesions described as tubercle are analogous to the secondary deposits occurring in pyæmia, and which are known to result from the poisoning of the blood by absorption and injection into it of putrid fluids, but they in no way support the hypothesis that Phthisis Pulmonalis is contagious or infectious. But we shall again allude to this matter under the head of Theory of the Production of Phthisis.

[The controversy in regard to the communicability of Phthisis has not yet been set at rest. Amongst the most careful experiments upon the subject appear to have been those of Tappeiner, of Meran, in the Tyrol.[2] He caused dogs to breathe for several hours daily the air of a chamber which had been impregnated, by means of an atomizer, with a mixture of phthisical sputa with water. After a period varying from twenty-five to forty-five days, all but one of eleven animals so treated were found, upon being killed, to have miliary tuberculization of both lungs; most of them having the same deposit also in the kidneys, and some in the liver and spleen. Microscopical examination accorded with the naked-eye appearances.

Dr. Max Schottelius, becoming acquainted with these experiments, repeated them, with important variations. In a number of instances he impregnated the air to be breathed not with tubercular sputa, but with those of simple bronchitis; in other like experiments, with brain, cheese, and cinnabar. Bronchitic sputa produced tuberculosis in the animals so exposed as often as did the sputa of phthisis. Cheese had a less frequent effect; pulverized brain still less; and cinnabar the least of all, but still producing some tubercles in the lungs. These investigations only confirm the conclusion above stated by Dr. Bennett, that the causation of tuberculosis by an introduction of material into the system from without is *not specific;* since other matters besides tubercle can produce the same effects.

Still, this does not finally dispose of the subject. The question whether there is or is not a specific *contagium* of tubercle, as there is of syphilis or smallpox, has much of pathological interest. But the paramount inquiry for the physician is,

[1] For a good summary of M. Villemin's views and experiments, see Edinburgh Medical Journal for February, 1867, p. 756.

[2] Medical Times and Gazette, 1867.

[3] On the Artificial Production of Tubercle in the Lower Animals. 4to. London, 1868.

[4] Tenth Report of the Medical Officer of the Privy Council. London, 1868.

[1] Wilson Fox, op. cit. p. 5.

[2] Lancet, Nov. 23, 1878.]

can Phthisis, whether specific or not, ever be communicated?

Drs. W. H. Webb[1] and E. Holden,[2] in papers upon this topic, give references showing an affirmative opinion in regard to this question to have been expressed by Galen, Cullen, Heberden, Morgagni, Laennec, Andral, Bright, Addison, Copland, Drake, Dickson, Budd, Walshe, Beale, Bowditch, Flint, Stillé, Da Costa, and others.

Dr. Holden obtained, in answer to circulars of inquiry, two hundred and fifty replies from leading physicians in various parts of the United States. Of these, one hundred and twenty-six affirmed their belief in the communicability of consumption. Seventy-four gave a negative answer; and fifty were in doubt upon the subject.

The evidence which has produced this affirmative conviction in so many minds is of a simple character. A man or woman, previously in excellent health, and without inherited predisposition to consumption, nurses a wife, husband, sister, or friend, through a fatal attack of Phthisis; and then, after a few weeks or months, sickens and dies of the same disease. Coincidence is, of course, apart from communication, a possibility; and the effect of long, anxious watching, probably with much confinement in a close atmosphere, must not be ignored.

Some cases, however, have a more striking appearance. Take, for example, the following:—[3]

"The only two midwives practising at Neuenberg, a healthy little town of 1300 inhabitants in 1875, were R. and S. Of these, the woman S. was undoubtedly the subject of Phthisis, with abundant puriform expectoration. In the first case described, Dr. Reich extracted the child by turning. While his attention was engaged with the mother, he noticed that, owing to some difficulty in the child's breathing, the nurse S. sucked the mucus from the infant's mouth, and also endeavored to promote respiration by blowing into its mouth. For the first three weeks the child progressed well, but then its health failed, and within three months of its birth it died of well-marked tubercular meningitis, initiated by symptoms of bronchial catarrh. In May and June following two more children died of the same disease. These three cases had been attended by the nurse S. Dr. Reich's attention being thus attracted, he found, on investigation, that between the 4th April, 1875, and the 10th May, 1876, seven children, in addition to the above three, had died (all within the first year) of tubercular meningitis, although in no case was there any history of hereditary tuberculosis; that all these cases had been attended by the woman S., while of all the cases attended by the other midwife, R., not one had died of this disease, nor had any manifested in any way indications of any tubercular form of disease. The duration of the illness varied from eight days to three weeks; whereas of the ninety-two children who died in their first year during the nine years from 1866 to 1874, only two died of tubercular meningitis; and similarly, among the twelve infants who died in 1877, there was only one such case, and its parents were tuberculous. The midwife S. herself died of Phthisis in July, 1876. It was ascertained that S. had been frequently in the habit of sucking mucus from the mouth of infants, and also of caressing and kissing them."

On the other hand, as Dr. A. Stillé remarks, "if Pulmonary Phthisis were often conveyed by contagion, the cases ought to be of daily occurrence, since the disease is the most frequent of all mortal diseases." Dr. Cotton,[1] of the Brompton Hospital for Consumptives, and Dr. MacCormac,[2] of Dublin, have argued strongly against the idea of communicability. From the statistics of the Brompton Hospital, collected by Drs. Cotton and Edwards, it has been shown that of the many nurses and others engaged in that institution during twenty-one years, but one nurse and one servant died of Phthisis. Especial care seems to have been taken, in the Brompton Hospital, in regard to ventilation and other hygienic conditions. Dr. Cotton's expression is, that "a residence in the consumptive hospital, and long-continued working in its wards, is a very good way, indeed, *not to catch the disease.*" In the most cogent instances cited in favor of contagion, the person appearing to contract Phthisis from another has been one who, for weeks or months together, slept in the same room, often in the same bed; besides being in the same apartment also for a great part of every day. Thus not only the injurious effect of "rebreathed air" (MacCormac) was felt, intensified usually by shutting up windows, &c., to keep out the cold, but the inhalation of air exhaled from diseased lungs was almost constant. "Continuous molecular change" (Snow) may be easily supposed in this way to occur; through the introduction into healthy lungs of minute particles of disintegrated

[1 Amer. Journal of Med. Sciences, April, 1878, p. 426.]

[2 Amer. Journal of Med. Sciences, July, 1878, p. 145.]

[3 Reich, in Berliner Klinische Wochenschrift, Sept. 18, 1878.]

[1 Brit. Med. Journal, 1872, vol. ii. p. 239.]

[2 On Consumption; London, 1865.]

lung tissue, given out in the breath of a phthisical patient, and not removed by ventilation.

From the foregoing considerations, the following conclusions may be derived:—

1. Tubercle is not a *specific* morbid product, and therefore in no strict sense can Phthisis be called a contagious disease.

2. Exposure to the atmosphere breathed by consumptives is not attended by danger, so long as good ventilation is maintained.

3. Inhaling the breath of patients far advanced in Phthisis, in close rooms, and for long periods together, has been, in some instances, followed by the development of the disease in persons previously healthy.

4. Therefore, we should always advise that no healthy person shall sleep in the same bed with a consumptive; nor, if avoidable, in the same room, unless ample ventilation is maintained.—H.]

Occupation.—Phthisis is unusually common among the workers in certain trades, more especially stonemasons, grinders and polishers of steel, dressers of flax and feathers, cotton carders, china scourers and potters, tailors, sempstresses, straw-plaiters, lace-makers, silk-workers, and iron and coal miners. On the other hand, cooks, butchers, tanners, tallow-chandlers, and soap-boilers, enjoy to a great degree an immunity from the disease. In the first class of cases the inhalation of foreign particles into the lungs excites local irritation, which proves injurious to the respiration, and deteriorates the constitution; or the result is occasioned by the combined operations of sedentary employments, impure atmosphere, exhaustive work, and bad food. In the second class of cases there are good wages, and, as a concomitant, good food, while a constant contact with oil is supposed to offer an additional explanation of the fact.

Humidity has been supposed to exercise a considerable influence in the production of Phthisis. Magendie thought he had produced tubercle in rabbits by confining them in damp cellars. Baudelocque points to numerous localities, such as morasses, houses surrounded by ditches, and so on, where the disease is rife. It is also common in Holland, and other countries liable to damp fogs and an atmosphere saturated with moisture. Phthisis has been shown to prevail in the damp soils of the United States by the careful investigations of Dr. Bowditch, of Boston, U. S., and of England by those of Dr. Buchanan.[1] On the other hand, in elevated dry regions it is said to be comparatively rare. In the Seventh Annual Report of the Registrar-General for Scotland, it is pointed out that for every 100,000 inhabitants there died annually from consumption 206 persons in Leith, 298 in Edinburgh, 310 in Perth, 332 in Aberdeen, 340 in Dundee, 383 in Paisley, 399 in Glasgow, and 400 in Greenock. In these towns, therefore, the death-rate is diminished in proportion to the dryness of the site.

Diet.—Of all the causes producing Phthisis and tubercular diseases generally, a low diet, or imperfect assimilation of food, is the most obvious and unequivocal. Among the lower orders we observe this to be the case in all large cities, among the ill-fed and half-starved poor, in orphan and foundling institutions, and whenever from any cause the food of the people is rendered scarce or dear. In the higher classes we observe it following the system of nourishing infants by hired nurses, or bringing them up by hand, and in early childhood from a pampered indulgence in indigestible or non-nutritious substances. Not unfrequently it results from allowing weak children to reject the fatty constituents of food. Most of the other causes to which we have referred will be found on examination to have influenced the economy, by diminishing appetite, and impeding digestion and assimilation of food.

Other diseases.—It has frequently been observed that Phthisis follows attacks of previous diseases, which by either affecting the lungs, or strongly depressing the system, and not unfrequently by both, appear to have caused the disease. Thus it has followed pneumonia, bronchitis, measles, and hooping-cough in persons previously healthy. Want of appetite and dyspepsia in the young are fertile sources of Phthisis. Indeed, all disorders which permanently lower the strength in the young, and interfere with the nutrition so necessary at that period of life for developing the growth of the body, may be regarded as a cause of tubercle. The weakness resulting from parturition and prolonged lactation in feeble women is a striking example. For the same reason it occurs in some rheumatic and gouty persons.

Predisposition.—Seeing that none of the causes mentioned invariably produce the disease, and that striking exceptions may be cited of persons who exposed to one or all of them have yet escaped the malady, the difficulty has been attempted to be got rid of by recourse to predisposition. In the same manner that many persons exposed to fever or smallpox are not affected, or that certain plants only grow on particular soils or patches of ground, so it is said there must be a something superadded to other causes in tubercular cases, which is called predisposition. It is unnecessary to enter upon the subtle argument which has thus been raised, and

[1] Tenth Report of the Medical Officer of the Privy Council, 1868.

which appears to us, in the present state of science, as reasonable as is the calculation of chances concerning the probability of escape to any particular soldier who exposes himself to the fire of an enemy. In neither case is it predisposition nor chance, but rather the operation of fixed laws, which it is not given to us as yet to recognize, or regarding which we cannot so calculate as to avoid their operation.

It may be observed, especially among the lower classes, that vitiated air, humidity, want of cleanliness, bad diet, drunken habits, and a variety of debilitating causes, all concur apparently to produce the effects, so that it becomes very difficult to attribute the disease to any one especially. In the higher classes two causes more especially are found, viz. an hereditary taint, and improper nutrition. On looking at the whole train of causation, it seems to me certain that they may all converge in mal-assimilation or deficiency of food. As far as the strength of the economy and constitution of the blood are concerned, it matters little whether deficient vitality be caused by the food being deficient, or, if abundant, its not being digested; or again, if digested, its being deteriorated in the lungs by noxious gases, by inoculation of morbid matters, or by constant congestion, the result of tissue irritation. As a general conclusion we hold to the belief that the great cause of tubercle is weakness of constitution, or diminished vital power, however produced; a theory which has the merit of teaching mankind to avoid all causes which may exhaust the frame, and to establish as remedies everything that can communicate to it strength and vigor.

Natural Progress of Phthisis.—The commencement of Phthisis may be said to be established as soon as it is distinctly shown that tubercles exist in the lung. This period, however, is generally preceded by more or less deterioration in the general health, indications of debility, and impoverishment of nutrition. It is true there are many individuals in whom the deteriorating process is so gradual, that this change has not been observed either by themselves or their friends, but it is seldom that such will escape the observation of the experienced physician. At other times the impaired health is caused by some exhausting malady of a general character, or of one especially affecting the chest. It sometimes happens that the first obvious departure from health is a hemorrhage coming from the lungs. It is under these or other exhausting circumstances that a matter is exuded in a fluid state from the capillaries of the lungs, which collects and coagulates in such portions of the pulmonary texture as offer least resistance. Although a small portion may insinuate itself between the elementary textures of the organ, it will principally pass into the air-vessels, so as to obstruct the entrance of air. A miliary tubercle may in this way block up from three to twenty of these air-vesicles. The amount of isolated tubercles so formed in the lung, their aggregation and union together giving to the morbid product the appearance of infiltration, somewhat impedes respiration and the functions of the pulmonary organs, according to the extent of the morbid product. Their presence, also, by irritating the pulmonary nerves, gives rise to the frequent dry cough so common in the early stage of the disease. The tubercular matter having coagulated, constitutes a foreign solid body, which can only be removed by being again broken down and so rendered capable of being either absorbed or excreted. Thus the miliary or infiltrated forms, whether gray or yellow, after a time soften—a process which may commence at any part of the mass, and gradually affect the whole. This softening is a disintegration or slow death of the tubercular exudation, constituting true ulceration, which is more or less extensive, according to the amount of the morbid deposit. When recent, the pulmonary tissue in the immediate neighborhood is more or less congested, but when chronic it is thickened and indurated, often forming a capsule, which surrounds the hardened tubercle, or a membrane lining an excavation. The other neighboring tissues are also necessarily involved. The pleuræ are thickened, the bronchi sometimes loaded with tubercle, at others obliterated by pressure, the bloodvessels are congested, ruptured, and ultimately impervious, and the nerves compressed and irritated. As the ulcerative process extends, the elementary structures of the lung are more and more destroyed, the excavations become larger, more numerous, and unite with each other, until at length the pulmonary organs can no longer perform their functions. In most cases, however, before this is arrived at, tubercle appears in other parts of the body, producing complications, under the united effects of which the strength is exhausted.

It is only in rapid or acute cases of Phthisis that the ulcerative tendency of the tubercular exudations pursues an uniformly destructive progress. In chronic cases this is frequently checked, and for a time slumbers, the symptoms improving and the patient exhibiting temporary signs of recovery. These arrestments of the disease may be of greater or less duration; and there can be no doubt that they are permanent in a far greater number of persons than is generally supposed. Indeed, while the more extended cultiva-

tion of morbid anatomy in recent times has demonstrated the frequency of cretaceous and calcareous concretions at the apices of the lungs, as well as of pulmonary cicatrices, physical diagnosis and more careful observation have shown in the living, that corresponding with the disappearance of symptoms and physical signs the health has improved, and ultimately been permanently restored. We are satisfied that there is no period in the history of the disease in which permanent arrestment may not take place, although, of course, it is far more common when it is limited in extent, and confined to one lung. The facts we have seen and recorded on this subject, however, show[1] that individuals with extensive cavities and disease on both sides may, under favorable circumstances and with appropriate management, ultimately recover.

THEORY OF THE PRODUCTION OF PHTHISIS.—It is not our intention to enter into an account, descriptive and critical, of the numerous views which have been held in past times as to the essential nature of Phthisis. It will be sufficient to speak of the two theories which are now being discussed, and of the reasons which induce us to adopt the one and to reject the other. The first theory supposes an altered condition of blood, originating in a perversion of nutrition. This perversion, as we have seen, has been considered by some to be owing to vitiated air, by others to imperfect assimilation of food, and by others to an hereditary taint. It has also been shown experimentally, that it may be caused in the lower animals by inoculation of various morbid matters. All these, and indeed other causes, may originate or co-operate in diminishing the vital power of the individual, and directly or indirectly produce weakness, feeble digestion, and an impoverished blood. It is when in this condition that any accidental irritation of the lungs, often inappreciable and undetectable, causes a limited congestion here and there in the pulmonary organs, which terminates in more or less exudation of the liquor sanguinis. This exudation coagulating causes the miliary and infiltrated forms of tubercle previously described, which partaking of the diminished vital power of the organism, instead of being transformed into the pus characteristic of a similar exudation in a healthy person, produces the small, irregular, and imperfect bodies called tubercle corpuscles. Instead of cells, which are rapidly produced, broken down, and absorbed as in pneumonia, we have numerous molecules and bodies resembling ill-formed nuclei. In short, we have a chronic exudation, in which the vitality is so lowered that it tends to disintegration and to produce the lowest kind of organic forms,—*i. e.*, molecules, granules, and nuclei.

The second theory is one which, instead of ascribing tubercle to an exudation from the blood, of low vital power, regards it as the result of increased cell development and multiplication of the included nuclei. According to this view tubercular matter is a new growth, which when we consider that it sometimes reaches the size of an apple, as in the brain, would demand for its production increased rather than diminished nutrition. Notwithstanding the desire of those who support an exclusive cell theory to trace tubercle as well as every morbid product to some cell transformation, the most careful and repeated investigations of histologists have failed to do so. According to Virchow, however, upon isolating the constituents of a tubercular mass "either very small cells provided with one nucleus are obtained, and these are often so small that the membrane closely invests the nucleus, or larger cells with a manifold division of the nuclei, so that from twelve to twenty-four or thirty are contained in one cell; in which case, however, the nuclei are always small and have a homogeneous and somewhat shining appearance."[1] This description of small nuclei in the interior of cells, and the appearances figured as constituting the structure of tubercle, have, so far as we are aware, never been confirmed by any experienced histologist. Tubercle is so common a morbid product that if such indeed were its constitution, it ought to be seen at once; but our most anxious and repeated efforts have failed to discover it, nor does there exist a single preparation anywhere capable of demonstrating it. Cells containing many nuclei are very rare, associated with tubercle, and when they do occur are evidently dependent on the occasional irritation of texture which is produced around the morbid products—they are a result and not a cause. As a matter of fact, therefore, not to speak of the theoretical improbability of a disease originating in weakness commencing with increased power of vital development in the pre-existing tissues of the organism, this theory must be rejected.

In support of this last theory it is further maintained by Virchow and his followers, that the term tubercle should be limited to the minute, indurated granulations which, as Lebert originally pointed out, are the result of increased nuclear growth in the fibrous tissues—what he denominated fibro-plastic corpuscles. The larger so-called tubercular infiltra-

[1] See my work on Pulmonary Consumption, Cases 1, 2, 21, 22, &c.

[1] Virchow, by Chance, p. 476, and fig. 140.

tions of morbid anatomists and practical physicians they regard as chronic or, as they call them, cheesy exudations. Dr. Burdon Sanderson proposes that tubercle should be called an "adenoid growth,"[1] and it may be granted that a mass of molecules and tubercle corpuscles, such as we have described, in a fibrous tissue, may present a vague resemblance to one of Peyer's glands. But a slight consideration must show that these distinctions are more verbal than real. It is not the occasional, scattered, and rare indurated granulation with which we are so much concerned as the extensive, chronic morbid deposit. Transferring or limiting the term tubercle to the accidental granule, and calling the general and essential morbid product chronic inflammation, or adenoid growth, constitutes no real advance in pathology. What we have from the first maintained is that we have to do with a *tubercular exudation*, which differs from an inflammatory and cancerous exudation in its low vital energy and diminished power of transformation into cell forms; and that this is the essential element of Phthisis Pulmonalis. Two recent French admirers of Virchow's doctrines have proposed to separate ordinary Phthisis from granular tubercle of the lungs, under the name of Tubercular Pneumonia,[2] and Niemeyer suggests for the term Phthisis, Chronic Pneumonia.[3] These propositions, while they indicate an essential agreement with the doctrines contended for in this article, offer no real advantage. It is not the name we attach to a morbid state, but a clear comprehension of the morbid state itself, which is of real importance. It is now many years ago that we pointed out the existence of a true vesicular pneumonia, which to the naked eye resembled scattered grains of yellow tubercle, but which under the microscope was composed of desquamated epithelial scales and pus-cells, mingled with fine molecular matter.[4] That a pneumonia may be vesicular, lobular, or lobar, is now agreed upon by every pathologist, and the same forms dependent on the extent and seat of the exudation may be observed in tubercular deposits.

Satisfied then that tubercle is essentially a coagulated exudation, we have next to ask, why such exudation is not rapidly transformed into pus-cells, as occurs in an acute pneumonia? The reply is, in consequence of the deficient strength and want of vital formative power in the organism. If it be further asked, on what that deficient energy, in its turn, is dependent? the answer is, that in consequence of impeded nutrition, or other causes, the blood is rendered so abnormal, that its fluid constituents when exuded are incapable of supporting cell formation. But it must not be forgotten that as the blood is continually undergoing changes, now receiving and then giving off new matters, it never remains the same for many hours together. An exudation at one period may abound in elements which do not exist in it at another. Hence why we find all kinds of intermediate formations in the textures in tubercular cases, and why the exuded matters associated with the lowest form of morbid formation may be occasionally mingled with the higher. A cancerous growth, however, is very rarely met with in conjunction with tubercle.

When we next come to inquire what is the nature and essential cause of that altered nutrition which so modifies the blood, that when its fluid portion is exuded it should constitute tubercle, we must inquire in what manner the digestive processes are primarily impaired. And here we must remember that all food essentially consists of albuminous, fatty, and mineral constituents, which are reduced in the alimentary canal to a fluid condition by the mechanical triturating action of the teeth, jaws, and stomach, as well as by the chemical solvent action of alkaline and acid juices. An observation of the peculiar dyspepsia which so frequently accompanies tubercular disease will satisfy the observer that it depends upon excess of acidity in the alimentary canal, which favors the solution of the albuminous and mineral matters, but is opposed to the emulsionizing of fat. It has consequently been attributed by Dr. Dobell to diminished secretion from the pancreas. In youth the indisposition to eat fatty substances is well marked, and among the ill-fed poor it is fat which is the most costly ingredient of food.[1] In either case it is the non-assimilation of the fatty elements of food and their diminution in the blood, while the albuminous elements are comparatively in excess, that gradually interferes with nutrition; the molecular basis of the chyle is impoverished, the elementary molecules so necessary for the formation of healthy blood corpuscles are diminished, the liquor sanguinis consequently is poor in fat and rich in albumen, the entire growth of the constitution, as a result, is affected, and its powers rendered weak; lastly, when exudations do occur, more especially in the lung, they are of an albuminous char-

[1] See Edinburgh Medical Journal, November, 1869, p. 386; and Eleventh Report of the Medical Officer of the Privy Council, plate 5, fig. 3.

[2] Herard et Cornil sur la Phthisie, 1867.

[3] On Pulmonary Consumption; Sydenham Society's Translation.

[4] Clinical Lectures, 5th edit. p. 689.

[1] See Report by Dr. Edward Smith.

acter, exhibit slight power of transformation into cells, and only produce that slow abortive nuclear material which is called tubercle. Such is the theory of Phthisis we consider most consistent with all the recognized facts connected with the origin and progress of the disease, the correctness of which is still further supported by what is now known, 1st, of the chemical constitution of the food, and the transformations it undergoes in the body; 2dly, of the relations which exist between digestion and the working powers of the individual; and 3dly, as we shall subsequently see, by what experience has taught us of its successful treatment.

II. Symptoms of Phthisis.

From what has been previously said under the head of Morbid Anatomy of Phthisis it must be apparent that the symptoms which it presents will not only have reference to alterations in the functions of the lungs, but to those which may arise from disease in other organs. We must further consider that its onset may be insidious and scarcely perceptible, or on the other hand startling from its violence or acute character; that its progress may be rapid, slow, or irregular, and its termination ushered in by various phenomena not unfrequently of a very complex character. Notwithstanding, to the pathologist who has carefully studied the morbid anatomy, natural progress, and theory of the disease, the symptoms and physical signs of Phthisis will enable him to determine the morbid condition present in the great majority of cases with an exactitude and certainty of which the modern cultivators of medicine may well be proud.

Premonitory Symptoms.—Before any one can positively state that tubercle exists in the lung, there generally occur symptoms indicative of diminished general health, and of deteriorated constitutional vigor. In many cases it is observable in young persons that they are not good eaters, dislike fatty substances, are capricious with regard to food, become thin, pale, weak, and liable to dyspepsia, complain of indigestion and irregularity of the alvine discharges, and to the observant eye are at once recognized as individuals ill nourished and liable to tubercular disease. This condition, however, is often not noticed by the parents or friends, who regard it as only natural to youth, or to the circumstance that they eat so little. On other occasions it creates apprehension and alarm, the physician is consulted, who, however, can detect no pulmonary disease or pulmonary symptom of any kind. If, in addition to the above phenomena, the individual complains of chills, cold feet, occasional perspiration, quick pulse, rendered more frequent at night, the general condition is one highly favorable to the occurrence of Phthisis.

In adult persons the premonitory symptoms are most commonly lassitude, incapacity for following the usual employment, diminution of appetite, with or without indigestion, and a sensible falling off in flesh. Various diseases may manifest themselves, such as gouty or rheumatic attacks, influenza, bronchitis, fever, dysentery, and others, which leave the individual in a debilitated state. There may now come on considerable hæmoptysis, although an examination of the lungs reveals no sign of tubercle; or an attack of pneumonia may appear, which if treated by lowering remedies may usher in the disease. Occasionally the skin of the face becomes gray, and a haggard and worn expression is communicated to the countenance. Pregnancy and lactation in weak females frequently introduce Phthisis, as, indeed, may everything that calls too strongly for exertion of the vital powers in weak and predisposed persons, or that causes vitiation of the blood. It is in this respect that the recent experiments of Clark, Fox, and Sanderson, previously referred to, indicate how Phthisis may follow suppuration or irritating diseases of texture, and how if occasioned in one organ it may spread to others.

It is when the constitution is thus enfeebled that Phthisis appears in its acute or chronic forms.

Acute Phthisis.—This form of the disease, commonly called "galloping consumption," is generally distinguished not only by its rapid progress, but by the febrile symptoms which accompany it. There are frequent chills, followed by great heat and sweating, red tongue, nausea, loathing of food, vomiting, and diarrhœa. There is a rapid pulse, at first of good strength, but soon becoming feeble, dyspnœa on slight exertion, cough, profuse expectoration, sometimes tinged with rusty-colored blood. Occasionally the expectoration is trifling. There is great exhaustion, rapid emaciation, restlessness, and, before death, wandering of the mind and delirium. On percussion one or both lungs exhibit unusual dulness, which rapidly extends and becomes more intensified. It is sometimes most marked at the base. On auscultation there are at first dry, bronchial sounds, and prolonged expiration, which soon pass into moist rattles, loudest with inspiration. The crepitations are now transformed into mucous râles more or less coarse, frequently accompanied with dry, bronchial murmurs and pleuritic frictions. The extent of these signs in-

dicates the area of lung-tissue involved, while the amount of increased vocal resonance points out the density of tubercular and pneumonic exudation infiltrating the lungs, or the anfractuous softening and excavations produced.

These acute symptoms occur occasionally in most cases of Phthisis, and indicate the period when exudation is being rapidly deposited in the lungs, or on the pleuræ. In many cases they constitute attacks supposed to be the result of having "caught cold." Then they decline, and are absent for varying periods. The greater the number of these attacks, the more rapid is the progress of the disease; and when they are continuous, it produces that form of it denominated acute Phthisis. Such cases may prove fatal in a period varying from two or three weeks to a few months.

Chronic Phthisis.—In the vast majority of cases the progress of Phthisis is slow, often coming on imperceptibly, and too frequently exciting little attention until it is far advanced. I have known the only daughter even of a medical man slowly pass through all the stages of the disease, the cough and expectoration failing to attract special notice in the family until three weeks before death, when on examination by a physician large cavities were detected. At other times it is ushered in by well-marked disease, such as pneumonia or bronchitis, and in some instances the first symptom observed is hemorrhage. These different modes of onset in the disease we regard as sufficiently important to merit a separate description.

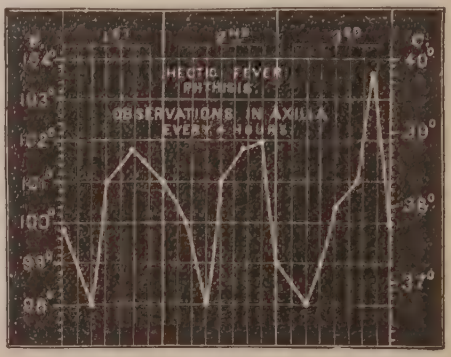
Gradually-developed Phthisis.—The first symptom which appears is cough; at first, however, so slight as scarcely to attract attention, and attributed to transient exposure to cold, or tickling in the throat. It may be observed, however, to be persistent, and of a dry, hacking character. Sometimes the cough is accompanied with pains in the shoulders, tightness in the chest, slight dyspnœa on exertion, together with all the other symptoms described as premonitory. On percussing the chest no dulness can be detected at this early period; but on auscultation there may frequently be detected feeble respiration under one clavicle, and, during forced inspiration, harshness of the breath murmur, with prolongation of the expiration. After a variable time expectoration follows the cough; at first consisting of transparent, frothy mucus in small quantity, but soon becoming opaque and purulent, and often streaked with a little blood. The cough and expectoration now become gradually increased, and all the other symptoms which have preceded or accompanied them are intensified; the failing appetite is more marked, the quickened pulse and feverish excitement more evident, and the general weakness, falling off in flesh, pallor, and languor make progress. A period, sooner or later, arrives when on careful percussion a sensible dulness may be detected under one clavicle. On auscultation over this dulness, either there is increased harshness of the breath-sound on taking a deep inspiration with prolonged expiration, or a slight crepitation may be discovered during some parts of the inspiratory act. Increased vocal resonance, also, is present over the dull portion of lung. The various symptoms and signs enumerated characterize what many authors regard as the first stage of the disease.

The physical signs now assume marked importance in the history of the case, indicating, in the majority of instances, with great exactitude, the extent of the tubercular deposit, and the changes which it undergoes. The area over which dulness can be detected by percussion gradually extends from the apex downwards, until it occupies one-third, one-half, or even a greater portion of the lung. Dulness may appear at the summit of the other lung, and all the signs observed on the one side may follow on the opposite one. The crepitation on inspiration also extends, and, at first very fine, gradually becomes larger and coarser, until a loud, mucous rattle is established. The vocal resonance, which at first is only slightly increased, becomes louder and louder, until at length decided bronchophony is produced. During the occurrence of these changes in the physical signs, the cough becomes more frequent and prolonged, especially early in the morning, the expectoration is more abundant, and at length consists of dense, purulent masses, some of which sink in water. These also may, from time to time, be streaked with blood, or even slight hemorrhage from the lungs may occur. There is now generally visible emaciation of the body, considerable debility, indisposition to take exercise, dyspnœa on exertion, and especially on going up an ascent. The tongue is red, often glazed, and occasionally anæmic. There is anorexia and nausea, or the appetite is much diminished, and very capricious. The night sweats are often distressing; there is thirst, quick pulse, and not unfrequently marked fever at night. Sometimes diarrhœa may supervene, which invariably accelerates the progress of the disease. At others there may be various complications, such as attacks of laryngitis, pharyngitis, bronchitis, pleuritis, pneumonia, all of which produce increased weakness, and aggravate the sufferings of the patient. These occurrences characterize what have been termed the second stage of the disease.

The further progress of Phthisis is now characterized by the formation of excavations in the lungs, which are distinguished by loud, moist rattles, passing into gurgling or splashing sounds, if the cavities be large and contain fluid, or by loud, bronchial blowing, and rarely amphoric breathing, if they be dry. Percussion with the mouth open sometimes elicits a clear tone over such cavities; at others a peculiar chinking or cracked-pot sound. On speaking there is a shrill vocal resonance, called imperfect pectoriloquy; and occasionally the words uttered seem to come out of the chest, and strongly strike the ear through the stethoscope, a sign termed perfect pectoriloquy. Together with the signs of a dried cavity are frequently coarse creaking sounds, indicating the existence of chronic adhesions. At the same time dulness, and the other signs audible in the second stage of the disease, are more or less extended over one or both lungs. The cough is now very harassing and prolonged, and often so violent as to occasion vomiting, and it disturbs sleep at night. There is more or less dyspnœa, and occasionally, if the lung be extensively diseased, orthopnœa. The expectoration is greatly increased, consisting of nummular masses of dense, purulent matter, often containing portions of infiltrated lung, which rapidly sink in water. Sometimes it is greenish, ichorous, and of offensive odor. In very chronic cases, on the other hand, with dry cavities, the expectoration is trifling, and brought up with considerable difficulty. Hæmoptysis is now a more common symptom, and may vary in amount from a few teaspoonfuls to twenty ounces, or even more. Such attacks invariably cause great alarm, and produce exhaustion in proportion to the amount of blood lost. The patient frequently complains of pain in the thorax, which in very chronic cases is often severe, ushering in, more or less, flattening of the chest, that may now occur to a greater or less extent. As the disease extends, and the cavities enlarge, the strength of the patient declines, the appetite is lost, and it becomes difficult to eat anything. Hectic fever appears, there is a pink blush on the cheeks, rapid pulse, occasional rigors, profuse sweating at night, and extreme emaciation. Sometimes the vital powers slowly decline, and at length become extinct; at others, a colliquative diarrhœa appears, which more rapidly closes the scene. These symptoms constitute the third and last stage of the disease.

In the majority of chronic cases the progress of the disease is not uniform, but subject to numerous interruptions, and even long pauses in which there is decided amendment, with great amelioration and even absence of symptoms. But the physical signs, though they become modified, still indicate the existence of organic lesion. Not unfrequently, however, such pauses and ameliorations are continued for a long period, and in many cases may

[Fig. 23.



Diurnal range of the Temperature in Hectic Fever.—(Finlayson.)]

usher in a permanent arrestment of the disease. In such cases the expectoration gradually ceases, and the cough becomes dry. This, in its turn, becomes less frequent, and at length disappears. Auscultation indicates that the moist rattles are converted into dry blowing or bronchial murmurs. Coarse friction sounds appear, and indicate adhesions and cicatrizations. Dulness on percussion and increased vocal resonance remain, and, although seldom altogether got rid of, become more and more circumscribed, leaving sometimes only a trace behind to indicate the presence of disease. In severe cases the subclavicular regions of the chest are retracted; dense pleuritic adhesions are formed, which circumscribe the movements of the thoracic walls; but healthy respiration is heard in such portions of the lungs as were unaffected. Under such circumstances, although full vigor of body is not restored, life is continued, and enjoyed for an indefinite period, and death ultimately caused by circumstances altogether independent of the pulmonary lesion.

Hemorrhagic Phthisis.—The peculiarity of this form of Phthisis is that it commences with hæmoptysis more or less violent. I have now seen several cases in which individuals who imagined themselves to be in very good health, and in whom, on the most careful inquiry, nothing but some slight dyspepsia or falling off in appetite could be discovered, were suddenly seized with hemorrhage from the lungs. From that moment their general health began to give way, and Phthisis was developed, of which they died. I remember the case of an extensive sheep-farmer in the south of Scot-

land, who walking home one afternoon—as he thought in the possession of perfect health—was seized in the road with bleeding from the lungs. I saw him a few days afterwards; and failed to detect, either from his external appearance, general symptoms, or physical signs, the slightest evidence of pulmonary disease. Nevertheless in a few weeks he became pale and languid, cough appeared, and on his again visiting me a peculiar roughness, or what some call a dry crackling, was distinctly audible at the apex of one lung. He spent the following winter in the south of France; but, notwithstanding every care that could be exercised, he died of Phthisis at the end of three years.

So many cases of this kind have come under my notice, that I have no hesitation in regarding it as a peculiar form of the disease, in which tubercle is deposited in such a manner as in the first instance to induce degeneration and rupture of a considerable-sized vessel in the lung. The loss of blood so occasioned from one or more attacks assists in developing the disease, which subsequently progresses in the usual way. Occasionally such hemorrhages may occur several times before tubercle deposit has spread so as to be recognizable by physical signs. Not long ago I saw an Australian who for upwards of two years had several such attacks, and who only on reaching this country, in the month of November, when I examined him, had cough developed, with the incipient harshness of respiration.

This form is most common in adults, and is generally fatal, although I have seen a few instances in which, after a time, it was permanently arrested. It is allied to that class of cases in which at any period of the disease hemorrhage makes its appearance, and is recurrent.

Bronchitic Phthisis. — This form of Phthisis is more common in the young than in adults, and manifests itself in bronchitis, which attacks the apex of one or both lungs. It is a common sequence of severe attacks of influenza, hooping-cough, measles, or other diseases in which the bronchi are affected, in weak persons. They do not readily throw off the pulmonary affection, are very liable to colds; dyspnœa is readily excited by unusual exertion; they complain of a sense of tightness or constriction about the chest, which, on being examined physically, is quite resonant on percussion; but there is harshness of the inspiratory murmurs on taking a forced breath, with prolongation of the expiration, without increase of vocal resonance. In short, there is slight bronchitis at the apex, which, however, is permanent, or if it disappear for a time shows a great tendency to return. Occasionally there is wheezing, more or less sibilation, and great dyspnœa on exertion, with cough, expectoration, and slight hæmoptysis. For a long time the general health exhibits no further evidence of disease; but at length frequent cough and expectoration appear, weakness, failing appetite, emaciation, and the usual symptoms of Phthisis. In some cases the ordinary physical signs are also manifested, but in others I have known death occasioned without the production of dulness on percussion, increased vocal resonance, or other distinct signs of tubercular consolidation. In such cases, from first to last, bronchitis appears to be the only lesion while the patient wastes away and dies, although on inspection of the lungs afterwards they will be found to contain more or less tubercle. In 1845 I was consulted in the case of a young lady eleven years of age, who, after a violent and prolonged attack of hooping-cough, complained of dypsnœa on exertion, and cough. There was no dulness on percussion, and on auscultation there was harshness of inspiration, and slight prolongation of expiration at the apices of both lungs, especially on the right side. Under this affection she labored for eight years, in all other respects enjoying tolerable health, when the appetite began to fail, purulent expectoration became continuous, and all the symptoms of Phthisis were manifest. She died early in 1855, never having exhibited any of the physical signs of Phthisis, the disease apparently being structurally one of bronchitis and emphysema. On examination after death, however, I found circular patches of miliary tubercle, about three-quarters of an inch in diameter, irregularly scattered through the pulmonary tissue on both sides, together with emphysema.[1] I have since seen several similar cases, and am satisfied that bronchitis developed in weak young persons, especially when it appears at the apex of the lungs, is a frequent prelude and accompaniment of Phthisis, communicating to it a peculiar character, which has frequently led to much error in determining the nature of the disease. This form of Phthisis is allied to all those cases in which bronchitis, in its various phases, constitutes a leading feature of the disease.

Laryngeal Phthisis. — This distressing form of Phthisis is from an early period accompanied by a tickling in the larynx, which seems to be the origin of the cough. The voice becomes weak and hoarse, and not unfrequently there is more or less pain on deglutition. On inspection of the fauces and throat, follicular disease or great dryness of the mucous membrane is common. Sometimes the laryngeal disease completely masks the pulmonary

[1] See the author on Pulmonary Consumption, 2d edit. Case xvi p. 70.

lesion, causing a hoarse rough murmur on inspiration, which renders the physical signs at the apex of the lung inaudible, so that unless marked dulness is distinguished by percussion, it may be overlooked. Ultimately the voice is lost from destruction of the vocal cords by tubercular ulceration. Deglutition becomes difficult, and vomiting readily excited by reflex actions through irritation of the laryngeal, pharyngeal, and glosso-pharyngeal branches of the eighth pair of nerves. Under these circumstances emaciation makes rapid progress, all the symptoms of ulcerative laryngitis being added to those of Phthisis. (See Laryngitis.)

Pneumonitic Phthisis. — I have now watched a considerable number of cases in which unquestionable Phthisis has originated in an acute pneumonia at the apex of the lungs, which, instead of disappearing in the usual way, has become chronic. Under such circumstances the dulness on percussion and bronchophony remain, the summit of the lung is consolidated, the general health, instead of rallying, remains weak, cough and expectoration become troublesome, while loud mucous and gurgling rattles are gradually formed in the lung, indicating the existence of cavities. Sometimes the consolidated lung remains latent for a considerable time, the patient in vain endeavoring to restore his original strength. Then an attack of hæmoptysis has occurred, which induces him to visit a physician, and it is discovered that the lung is consolidated, and all the signs of Phthisis are more or less apparent. Discussion has taken place as to whether such cases should be denominated chronic pneumonia or Phthisis. In my opinion there is no difference between them. The exudation of the pneumonia degenerating, and not being absorbed, is transformed into tubercle, causing softening, ulceration, and destruction of the lung, in exactly the same way as if Phthisis had been developed from tubercle at the commencement. I have also seen survivors from this form of the disease with flattening of the chest, as in ordinary chronic Phthisis. It must not be overlooked either that intercurrent attacks of pneumonia are very frequent during the progress of Phthisis, and that at all times the two diseases exhibit a marked tendency to run into one another. This circumstance confirms the truth of the pathology previously given, and unequivocally proves that tubercle is only a low type of exudation from the blood. In healthy persons such exudation is transformed into pus, and rapidly disappears, whereas in individuals who are weak, and whose vital power is low, this process is more or less interfered with, is prolonged, and in extreme cases terminates in Phthisis. This view has recently been adopted by Niemeyer, who is one of those who purpose to call Phthisis Pulmonalis a chronic pneumonia, in the propriety of which, as applied to all its forms, I cannot concur.

Complications. — Tubercular disease of the lungs is necessarily associated with every lesion occasioned by inflammation and tubercular exudation of the textures of the organ. Indeed it may be said to be made up of exudation disorders, acute or chronic, affecting the air-vesicles, bronchi, fibrous tissues, and serous coverings of the lungs. Hence the various symptoms of laryngitis, bronchitis, emphysema, hemorrhage, pneumonia, and pleurisy are more or less mingled together, may supervene on each other, and occasionally, as we have seen, be so predominant and permanent as to give peculiar characters or forms to the disease. Occasionally pleuritis gives such a character to Phthisis, occasioning local acute or stitching pains. Tubercular cavities in the majority of cases induce thickenings and dense adhesions between the pleuræ, but sometimes they may burst or ulcerate through the pleuræ, where there is no adhesion, causing pneumo-thorax, associated or not with more or less empyema.

In addition, however, to these lesions of the chest, Phthisis may be associated with tubercular deposits occurring in other organs, in which case a train of symptoms will arise dependent upon the local lesion, wherever that may be. Of these the most common, and the most to be dreaded, is tubercular ulceration of the intestines, inducing colliquative diarrhœa, and perhaps perforation of the gut, with fatal peritonitis. In the young, also, we may find the disease associated with various tubercular or scrofulous diseases of the osseous texture, and sometimes of the brain or its membranes. It would exceed our limits to enter upon the innumerable complications which in this manner may arise; all that is necessary to say is, that there is no tubercular disease of any organ or tissue which may not be found sometimes associated with Phthisis, and which, contributing its own special symptoms to the pulmonary ones, increases the general disease and downward progress of the patient.

Besides this class of affections, there are others of importance. It is by no means uncommon during the progress of Phthisis to find persons complaining of puffiness of the feet, or face, and on examination of the urine it will be found to contain albumen. In short, one of the forms of Bright's disease may develop itself, and usually that now recognized as the waxy form. The liver also may enlarge, and add to the distress of the patient by its pressure and bulk. Such increased growth of the hepatic organ will also generally be found to be dependent on a

waxy transformation of its cells and vessels. The spleen may undergo a like alteration, although its enlargement is more rare. Pericarditis and other inflammatory diseases may occur—occasionally gout or rheumatism. Cancerous disease, it is now known, may be associated with Phthisis, but it is an occurrence of extreme rarity. In chronic cases the practitioner must be prepared to meet with a variety of other complications, which, though they may bear no essential or constant relation to Phthisis, render the disease more distressing and fatal should they occur.

III. Diagnosis of Phthisis Pulmonalis.

It has been previously pointed out that Phthisis is preceded by premonitory symptoms, which indicate diminished health, weakness, or imperfect nutrition of the individual. This condition has been spoken of by some writers as constituting a pretubercular stage of disease. All that can be said, in a diagnostic point of view, of this state of health, is that in young and delicate persons it should occasion much anxiety, as it may, or may not, terminate in Phthisis, and that it should demand great watchfulness and frequent careful examination, in order that the first positive signs of the disease may be detected.

Acute Phthisis.—The diagnosis of this form of the disease is exceedingly difficult, as all the symptoms and signs are identical with those of an acute inflammation of the lungs. It is only by careful observation of the premonitory symptoms, the existence of a marked hereditary taint, the amount of emaciation as compared with the extent of local disease, the continuity of the fever, and the rapid formation of cavities, that we are at length able to pronounce with confidence as to the presence of acute Phthisis. In all its essential features the attack is similar to acute pneumonia of the apex, from which in its earliest stages it cannot be separated. As the disease progresses, however, the excessive exhaustion and breaking down of the lungs establish the nature of the affection, while its rapid progress and the continued fever too certainly indicate its acute nature. In the present day the extreme difficulty of diagnosis is fortunately not of so much importance as it used to be, when such symptoms led to bleeding, and an antiphlogistic treatment. In the course of chronic Phthisis similar symptoms may arise, either from fresh exudation of tubercular matter, or from intercurrent attacks of pneumonia or pleurisy, communicating to the disease for a time an acute character.

Chronic Phthisis.—In this, by far the most common form of the disease, it is of the greatest consequence to determine its commencement by the conjoined methods now in vogue. Its progress is capable of being recognized with considerable certainty, and the means at our disposal for doing this may be considered under the heads of Pulmonary Symptoms, Pulmonary Percussion, Pulmonary Auscultation, Microscopical Examination of the Sputum, and Altered Changes in the Form and Movements of the Chest.

Pulmonary Symptoms.—The earliest symptom is cough, which, at first short and dry, resembles the ordinary effort at clearing the throat. Sometimes it is attributed to the chest, but more commonly is thought to arise from dryness or tickling in the throat. Such a cough too frequently excites little attention, although its persistency and defiance of ordinary remedies communicate to it a grave character. After a time the cough is followed by expectoration, at first of a thin mucous fluid, which, however, soon becomes thick and opaque, or is slightly streaked with blood. There is now occasionally felt a tightness or constraint, on taking a deep breath, under one clavicle, which, as the disease progresses, becomes painful, especially on coughing. This cough and expectoration, more particularly when they follow the premonitory symptoms, and are developed in the manner described, are highly characteristic of Phthisis. In the subsequent stage of the disease, the cough becomes more frequent, harassing, and long-continued. The tickling in the throat may excite vomiting. The expectoration is more abundant and prevalent, frequently tinged with blood, and forms distinct masses (nummular sputa) generally indicative of excavations, and may be so heavy that, instead of floating, it sinks in water. Lastly, it may contain masses of indurated matter, composed of portions of tubercular lung, or, in very chronic cases, fragments of cretaceous or calcareous matter. Early hæmoptysis, as we have seen, is highly diagnostic of Phthisis, and should always excite grave attention. Should it be soon followed by or mixed up with the other symptoms, the diagnosis is considered more certain.

Pulmonary Percussion.—When miliary or infiltrated tubercle occupies a certain number of the air vesicles, careful percussion above or under one clavicle elicits slight dulness of the pulmonary note, especially well marked when compared with the clear note on the opposite side. As it is seldom that the disease com-

mences at the apices of both lungs at once, this sign is one of great value, and indicates very positively, not only the existence, but very frequently the extent of the disease. The greater the dulness or flatness of tone, the more solid is the portion of lung struck; and the further over the chest, anteriorly and posteriorly, the dulness can be produced, the greater is the amount of pulmonary tissue involved. It should not be overlooked, however, that occasionally the disease exists equally on both sides, when diagnosis by means of percussion is always difficult. In the earlier stages, indeed, it is then impossible, and in the later stages, even with large cavities on both sides, I have known the percussion note so equal and clear as to mislead the careless observer. Sometimes also, though the lung be greatly condensed, an amount of emphysema anteriorly communicates clearness on percussion: hence the lung should always be examined posteriorly as well as anteriorly, in order to avoid error.

On percussing the chest in cases of Phthisis with the mouth open, there is sometimes elicited a peculiar noise, called by Laennec the *bruit de pot fêlé*, or cracked-pot sound, which he thought was diagnostic of a cavity. But I have found this noise could also be produced in cases of pneumonia, in pleurisy with effusion, and even in several healthy chests. Moreover it is often absent when pulmonary cavities are unquestionably present, and cannot therefore be considered as diagnostic of their presence, unless it be coexistent with other symptoms and signs of Phthisis. When present, it seems to indicate either healthy lungs, with very elastic thoracic walls, or else increased density mingled with confined or compressed air in the thorax. In either case, on striking the chest smartly, the air beneath is forcibly ejected through the bronchi and trachea, producing vibrations which occasion the peculiar sound.[1]

Pulmonary Auscultation. — The first sounds audible with the stethoscope are prolongation of the expiratory murmur and slight harshness, or a wavy interrupted character communicated to the inspiratory murmur. These signs, if clearly marked under one clavicle, following the premonitory symptoms, and accompanying persistent hacking cough, can leave no doubt that tubercle is actually present, and the disease pronounced. It frequently happens, however, that these signs are so indefinite that, although we may suspect, we hesitate to speak confidently. In all chronic organic diseases there must be a period so nicely balanced between health and disease—in which the altered texture is so slightly altered—that our senses are incapable of appreciating any alteration that may be produced. It is in such cases that everything which enables us to determine such delicate signs with greater exactitude becomes valuable, and I have no hesitation in stating that the differential stethoscope of Dr. Scott Alison has here afforded me the greatest assistance. In several delicate young persons, in whom when every precaution and care has been employed we fail to discover any alteration in the pulmonary sounds, an increased intensity in the sound of the carotid artery below the clavicle has afforded valuable indications. It is at this early and uncertain period of the disease that the greatest skill in auscultation and diagnostic powers are required in the physician.

As the disease advances, the prolonged expirations and harsh inspirations become more marked, and at length a decided increase in the vocal resonance of the affected side is audible. This indicates considerable condensation of the apex of the lung. If the disease progresses, slight crepitation is audible, at first at the termination of a forced inspiration, and gradually it occupies the whole of that act. This is diagnostic of tubercular softening. The fine moist rattle now becomes evident, and the increased vocal resonance louder, until it amounts to bronchophony. The auscultatory signs also extend in area over the chest, preceding the dulness on percussion, and generally appearing in the order in which they were noticed over the apex. At length the crepitation passes into mucous râle. This in its turn becomes coarser and coarser, indicating the existence of greater softening and even of cavities. As these enlarge, gurgling and splashing sounds are heard, especially on coughing, and the increased vocal resonance becomes pealing, and imperfect or perfect pectoriloquy is present. These latter sounds are diagnostic of a cavity or cavities. The sounds heard over these vary according to their size, contents, and the condition of the walls. If large, with rigid walls, and partly filled with fluid and partly with air, tinkling or metallic sounds may be heard on coughing or speaking. If altogether dry, amphoric or blowing noises may be distinguished. These last, if persistent, indicate that the secretion of pus is arrested, the softened tubercle got rid of, and contraction and cicatrization possible.

When in chronic cases of Phthisis dry blowing, combined with friction sounds, can be determined at the apex, it points out that adhesion and contractions of the tuberculated pulmonary tissues are taking place. If absence of respiratory murmur

[1] See the author on Pulmonary Consumption, &c., Diagnostic Value of the Cracked-pot Sound, p. 108.

exist, it may depend on pleuritic effusion, when dulness on percussion and increased vocal resonance, or ægophony, will determine the nature of the lesion. But it may be accompanied by resonance on percussion, with a brazen, hollow, or metallic sound on coughing or a forced inspiration; in which case there is pneumo-thorax, and the tubercular cavity has formed a communication with the pleura.

In retrograde Phthisis, the auscultatory signs disappear in the inverse order to that in which they appear. The moist sounds become dry, and these last diminish in intensity and extent. Friction noises and dry bronchial murmurs are heard, with prolonged expiration, wheezing, and sonorous rhonchi indicative of rigid bronchial tubes, conjoined with more or less emphysema. The area of dulness gradually diminishes, but a condensed mass in the lung generally remains for years at one or both apices, giving rise to harsh respiratory murmurs and increased vocal resonance, constituting strong evidence to the judicious observer of the diseased changes through which the lung has passed.

Microscopical Examination of the Sputum.—The sputum of phthisical patients, in the great majority of cases, may be found to contain, under the microscope, fragments of the areolar and elastic tissues, derived from disintegration of the lungs. They not infrequently present circles and half-circles, indicative of the form of the air-vesicles, and, when present, offer the most positive proof of pulmonary ulceration. Van der Kolk, of Utrecht, was the first to point out that

[Fig. 24.

Lung tissue obtained from sputa after digestion in caustic soda. (Drawn by Dr. John Wilson.)]

such fragments might be seen with the microscope, at the commencement of the disease, long before percussion or auscultation gave any positive signs of its existence. Although such examples are rare, I am satisfied that they do occur, and that the microscopical examination of the sputa under such circumstances enables us to arrive at a clear diagnosis when otherwise there would be great doubt. Drs. Andrew Clark and Fenwick have confirmed this fact by their researches into the structure of phthisical sputum. The latter physician has pointed out that the examination is much facilitated by first liquefying the sputa with a solution of caustic soda, when the fragments of lung tissue are precipitated, and their amounts as well as character readily estimated.

Altered Changes in the Form and Movements of the Chest.—As Phthisis advances, a distinct flattening and sinking in of the thoracic walls below the clavicle may be observed, generally coincident with the formation of cavities and loss of lung substance, of which it is diagnostic. An alteration in the movements of the affected side may be seen even earlier, and may be roughly ascertained by spreading the fingers of both hands like a fan over the two sides of the chest, and bringing the thumbs together at the middle of the sternum. On a forced inspiration, it may thus easily be seen that the thumb corresponding with the affected side moves less. The amount of this movement can be ascertained with great exactitude by means of the stethometer, and compared with that on the opposite side.

In addition to the symptoms and signs referable to the chest, there must not be overlooked a variety of circumstances which in conjunction with these will materially assist the diagnosis. Among these are the preceding premonitory symptoms; the continued impaired appetite and disordered digestion; the augmenting languor and debility; the hectic night-sweats; lustrous eyes; the hopefulness and imaginative intellect; and even the alternations of the disease from better to worse, all of which are more or less characteristic.

Much has been written concerning what is called the differential diagnosis of

Phthisis, and the means of distinguishing it from other diseases of the chest. But the truth is that a Phthisis necessarily implies the existence of almost every lesion of the lung, the tubercular exudation giving rise to or being accompanied by congestions and inflammations of the pleuræ, bronchi, and pulmonary parenchyma, with all their local signs and general symptoms. Pulmonary hemorrhage and abscess are common. Emphysema, though seldom present in its advanced stage, so as to alter the form of the chest, is common in limited portions of the lung near chronic and retrograde tubercular deposits. Any lesion whatever, occurring at the apex of the lung in a young person laboring under the premonitory symptoms we have described, must be regarded with suspicion. In adults, an acute pneumonia at the apex may go through its natural progress, and leave no trace behind. But if it becomes chronic, a Phthisis may be the result. Indeed there are many cases in which a chronic pneumonia of the apex and Phthisis Pulmonalis may be said to constitute the same disease. Cancer of the lung is a disease of advanced age; the dulness on percussion is more marked, the tubular respiration and bronchophony are much greater, and moist rattles are scarce or absent. Expectoration is trifling, and, when present, unlike that of Phthisis; sometimes it resembles currant jelly. The emaciation, night-sweats, and general aspects afford little assistance. A dilated bronchus, independent of Phthisis, is rare, but when present is often associated with bronchitis and asthmatic symptoms, while the physical signs of the cavity are generally best marked at the posterior and middle regions of the chest, rather than at the apex. In advanced cases a pleurisy with effusion or a pneumothorax may occur, when the physical signs distinctive of each will readily establish the diagnosis.

The great difficulty is to detect Phthisis at its first appearance, and hence every circumstance that can throw light on its history at this period is important. According to Dr. Ringer, the heightened temperature of the body, as determined by the thermometer, indicates the deposition of tubercle for several weeks before physical signs are developed. It is true that a similar increase of temperature occurs in a few other diseases, such as typhoid fever or rheumatism, but their symptoms are readily separable from those of Phthisis. This new method of recognizing the disease at an early stage requires more extended observation before it can be generally adopted. The subsequent progress of Phthisis admits of being followed by the physician cognizant of its morbid anatomy, and well skilled in auscultation, not only with certainty, but in the majority of cases with a degree of exactitude that must be regarded as highly honorable to the progress of medicine in modern times.

IV. Prognosis of Phthisis Pulmonalis.

Phthisis Pulmonalis, up to a comparatively recent date, was not only regarded as a very dangerous disease, but as one which was uniformly fatal. This idea was supported by the circumstance that before the general introduction of physical diagnosis it was not clearly detectable until it was far advanced, while the merely palliative treatment then in vogue was anything but favorable to recovery. If, notwithstanding, a case here and there did ultimately get well, medical men were more disposed to accuse themselves of an error in diagnosis than doubt the correctness of so general a dogma as the incurability of consumption. Even when at length morbid anatomy unequivocally demonstrated the possibility of tubercular cavities cicatrizing, and of individuals afterwards attaining an advanced age, such an event was regarded as one of extreme rarity, and as occurring altogether independently of treatment. "No fact," says Andral, "demonstrates that Phthisis has ever been cured, for it is not art which operates in the cicatrization of caverns; it can only favor this, at most, by not opposing the operations of nature. For ages remedies have been sought to combat the disposition to tubercles, or to destroy them when formed; and thus innumerable specifics have been employed and abandoned in turn, and chosen from every class of medicaments." Even Louis, in his admirable work, while admitting that a cure might rarely take place, points out that in such cases the disease must be limited and the result fortuitous. Hence the admitted occasional recoveries in no way interfered with the general view entertained of the unfavorable prognosis of this malady, or stimulated medical men to replace a palliative by a curative treatment.

At present, so far from Phthisis being considered to be uniformly or even generally fatal, it is admitted that treatment can in a great majority of cases prolong life, whilst in many, the number of which is annually increasing, a complete and permanent cure may be effected. This revolution in our prognosis of the disease is owing—1st, to the facts arrived at by morbid anatomy; 2d, to a more perfect theory or pathology of the disease; and 3d, to the discovery of cod-liver oil as a remedy.

1. The careful post-mortem examina-

tions now made with such regularity in our large hospitals have demonstrated the frequent occurrence of old condensations, cicatrices, and calcareous concretions at the apices of the lungs in persons of advanced age who have died of other diseases. In 1845, I pointed out that in the Royal Infirmary of Edinburgh they occurred in the proportion of from one-fourth to one-third of all the individuals who died after the age of forty. Roger and Boudet had previously shown that at the Salpêtrière and Bicêtre hospitals in Paris, amongst individuals above the age of seventy, they occurred in one-half and in four-fifths of the cases respectively. There can be no doubt that these cicatrices and concretions indicate the healing and drying up of cavities and softened tubercular matter at some previous period in the life of the individual, and the consequent spontaneous cure of the disease in a considerable number of persons.

2. The careful examination of tubercle by means of the microscope demonstrates that it neither originates in nor gives rise to cell formations, but that it consists of an exudation of the blood rendered feeble in vital power by impaired nutrition, and especially by deficiency of primary molecules of fat in the blood.[1] Hence the encouragement given to our efforts in stimulating the nutritive functions, and especially assisting in the increased assimilation of an easily digestible oil, whereby, while the tissues generally are supplied with formative material, the tubercular matter has time to degenerate and be absorbed; so that any cavities which have been produced may cicatrize. Attempts at cure in this direction have been so eminently successful as to influence our prognosis in a marked manner.

3. It is very much to be doubted, however, whether this pathology would ever have been arrived at, or if it had, whether a successful treatment could ever have been established, unless the therapeutical properties of cod-liver oil had been recognized. This animal substance is easily assimilated, is not purgative, and meets all the indications required, while experience has demonstrated that it restores to the emaciated body the nutritive elements it so much requires, and enables it to triumph over the disease. It can no longer, therefore, with truth be considered that Phthisis Pulmonalis is that *opprobrium medicinæ* it was formerly considered. Nor should certain charitable institutions any longer refuse to admit such cases on the ground of their incurability.

In my work on Pulmonary Consumption[1] will be found full details of the arrest of the disease in its most advanced stage, the individuals not only being still alive, but having enjoyed excellent health since their recovery, for periods varying from ten to twenty-five years. To the list of cases therein given I could now add many more. Twelve similar cases were recorded by Dr. Quain in 1852,[2] and many others may be found scattered in the works of different authors, and in the practice of individual medical men. There can be little doubt that could they be collected it were easy to prove that such examples, instead of being few and far between, are much more numerous than is generally supposed. It is very difficult, however, to watch for many years in succession the progress and termination of chronic Phthisis; and in hospitals this difficulty is increased, as the patients on getting better go out long before the disease is even permanently arrested. All attempts to induce medical men to unite and record their experience on this or any other great question involving the prognosis or treatment of disease have hitherto failed. We are, therefore, limited to the conscientious efforts of individuals in our attempt to elucidate this question, which cannot be expected in a matter of such magnitude and importance to be, at present, of any great avail. Among these, however, I have great pleasure in referring to the accurate method in which Dr. Pollock has recorded his ten years' experience at the Brompton Consumption Hospital.[3] Were such method and care more uniformly practised by hospital physicians, and extended over more lengthened periods, many of the unsolved problems connected with this subject might be elucidated. I confidently look to the future as affording means for demonstrating the ratio and conditions under which the prognosis of Phthisis may be determined. In the mean time, I can only express my conviction that its permanent arrestment and cure are, by judicious treatment and hygienic management, becoming every day more frequent and more widely extended.

In reference to the prognosis of individual forms or cases of Phthisis, we must regard acute Phthisis as generally fatal. The difficulty here lies in the diagnosis. Once recognized, however, the persistency of intense fever, with rapid emaciation and formation of cavities, give us little hope of a favorable termination.

[1] In making this statement I am fully aware of the observations and arguments of Virchow and his followers, but which, for the reasons previously given, I regard as not only inconsistent with histological and pathological research, but as especially opposed to all we know of clinical facts in modern times.

[1] Pp. 152 *et seq.*

[2] Lancet, pp. 487 *et seq.*

[3] The Elements of Prognosis in Consumption. London, 1865.

In the earliest periods of Phthisis, the prognosis should be very guarded, but on the whole encouraging. As a general rule, the more slowly it advances, the less fever and emaciation, and the better the appetite, the more probability exists of an arrestment.

In the second stage, the favorable symptoms are limitation of the disease to one lung, dulness not extensive, and not increasing rapidly; no persistency of moist rattle; expectoration moderate; fever trifling; emaciation not great; capability of taking nourishment and a certain amount of exercise. The unfavorable symptoms are continuous fever, quick pulse, hæmoptysis repeated, profuse expectoration, rapid softening of the tubercle, and its deposition in both lungs; bad appetite and impaired digestion; increasing emaciation; profuse diaphoresis and the existence of unfavorable complications.

In the third stage, the favorable signs are the existence of a cavity in one lung; gurgling or other moist rattles occasionally disappearing, and the excavation becoming dry, with blowing sounds; gradual flattening of the subclavicular space, while the other parts of the chest move freely. Further, the disease in the opposite lung absent, or if present slight, without a tendency to extend; coarse friction sounds over the cavity; and a general tendency to concentration, density, and fixity of the lesion. The favorable symptoms accompanying these local changes are, a tranquil pulse, no fever or sweating, emaciation checked, tolerable appetite, and capability of digesting nutriment, diminished cough and expectoration, power of taking more exercise and gaining flesh, and absence of complications.

On the other hand, the unfavorable symptoms are the converse of these, especially cavities on both sides, loud, moist, and gurgling rattles, increasing dyspnœa, profuse expectoration, especially of greenish or ichorous matter, extreme emaciation, anorexia, nausea, vomiting and incapability of retaining or digesting nutriment, profuse diaphoresis and quick pulse, fever and restlessness at night. If now any serious complication arises, more particularly continued diarrhœa, albuminous urine with œdema of the feet or ankles, laryngitis, or pneumo-thorax, &c., &c., death is not very distant. It is very rarely that hæmoptysis proves fatal, but should it occur profusely when weakness is extreme, death may be immediate.

V. Treatment of Phthisis Pulmonalis.

The treatment of Phthisis Pulmonalis, up to a recent period, has been too much governed by a desire to relieve symptoms —in other words, has been more palliative than curative. Unfortunately the remedies useful for the former purpose are altogether incompatible for the latter, and ultimately even fail to relieve the functional derangements to which they are directed. A study of the pathology of the disease has led us to the conclusion that Phthisis is dependent,—firstly, on impoverishment of the blood; secondly, on exudations into the lung, which assume a tubercular character; and thirdly, on destruction of the lung, owing to the successive depositions and softening of these. It follows that, instead of endeavoring to relieve cough or favor expectoration, our chief attention should be directed to improve the faulty nutrition, to cause absorption of the tubercular exudation, to arrest the ulcerative process, and, lastly, prevent a recurrence of the disease. The special treatment required in individual cases should be made subordinate to these great ends—at all events should not be opposed to them. We shall therefore consider the treatment as general and special: the first directed to favor the removal of the pulmonary lesion; the second to check occasional symptoms and complications.

General Treatment of Phthsis Pulmonalis.—The great indication in the treatment of Phthisis Pulmonalis should be to improve the nutrition of the economy. This does not merely consist in increasing the quantity and improving the quality of the food, but in employing all those means which shall secure—1st, an appropriate diet; 2d, causing its assimilation, and the formation of good blood; 3d, securing the proper purification of this by the atmosphere; 4th, seeing that a proper demand for the addition of new matters to the tissues is created by sufficient exercise; and 5th, that the effete matters be properly excreted from the economy by the emunctories. All these processes are comprehended in the function of nutrition. We shall most concisely convey our ideas as to the best means of increasing nutrition in phthisical cases, under the distinct heads of Diet, Cod-liver Oil, Pure Atmosphere, Climate, Exercise, and Bathing.

Diet.—One of the leading symptoms in cases of Phthisis is the diminished, capricious, or disordered appetite, and power of taking food. It is true that many cases persistently assure you that they eat heartily, but on careful inquiry they will admit their appetite is easily satisfied, or that they are small eaters. Even the friends sometimes assert that the patients eat as usual, that they have observed no change and so on, the fact being that the nutritive matter actually taken into the economy is far less than it ought to be. So little observation and attention

do those affected exhibit concerning their own cases, and so anxious do they appear to represent every circumstance in the most flattering point of view, that it is far from uncommon for them to declare themselves as constantly getting better, up to the moment of their death. I have frequently pointed out to my clinical pupils that, in the reports of these cases taken down by the clerk at the bed-side, in answer to questions, it has been recorded day after day that the appetite is better and better, while the patients are visibly getting more emaciated, more weak, and at length die. Among the poor and half-starving population, it frequently happens that it is not the appetite or desire for food that fails, so much as the food itself. The result here, however, is the same, viz. that the body is not sufficiently nourished, that the tissues disintegrate more rapidly than they can be supplied with new substance, and that the blood is deficient in what is so necessary for supporting health.

In all cases of Phthisis Pulmonalis the diet should be generous, consisting of boiled milk, cream, eggs, butter, toasted bread, and all kinds of animal food, and farinaceous puddings. Acid substances and drinks should be as a rule avoided, the tendency to dyspepsia from too much acidity being generally present. After dinner a glass or two of generous wine (sherry) or two or three glasses of sound claret (not acid) may be indulged in. As much variety as possible should be secured, and every pains taken by good cooking and superior quality of the viands to tempt the weak and capricious appetite. As to quantity, I have never seen any necessity for limiting it. The only difficulty is to take enough; the which once accomplished, amelioration in all the symptoms may be confidently predicted. It should be remembered, however, that mere eating and loading the stomach, without a proper digestion and assimilation, can be of little benefit. In this respect individuals differ, some doing best with two or three meals a day, whilst others find that eating more frequently, but less at a time, answers better. In nothing is the constant attendance of a judicious medical man more serviceable than in watching the effects of diet, and observing from its influence on each individual case how it should be regulated.

When fever runs high, the pulse is quick and the tongue furred, there will naturally be no disposition to take solid food. Under these circumstances we should take care that nutritious drinks are regularly administered, especially beef-tea and milk, and seize the earliest opportunity of returning to a more substantial diet. Many may think that in most acute cases, or during an intercurrent attack of pneumonia, these rules should be departed from. Formerly, indeed, antiphlogistics and the local application of leeches were employed; but it has now been satisfactorily demonstrated that even in acute cases of pneumonia itself, in vigorous constitutions, such practice is injurious:[1] how much more, then, would it be so in cases of Phthisis? Inflammations are now recognized to be diseases of weakness, and we feed them as we do fevers, with the most marked success. When, therefore, attacks of either supervene during the progress of Phthisis, so far from doing anything to diminish the strength of the economy, the most anxious care will be required on the part of the practitioner to counteract, by all the support he can administer, the future exhaustion of his patient.

An increase in the quantity and improvement in the quality of the food may frequently be observed to benefit cases of Phthisis, especially among the half-starved poor; the more so if associated with change of scene, active exercise, or varied employment. The treatment practised at the commencement of this century by Dr. Stewart, of Erskine, near Glasgow, which consisted in freely administering beefsteaks and porter, and causing exercise to be taken in the open air, excited considerable attention in its day by the success it occasioned. I have been informed that in America the consumptive patient, by eating the bone marrow of the buffalo on the prairies, is at length enabled to hunt down the animal. I have known several young men on large sheep farms in Australia cure their tubercular lungs by eating fat mutton and galloping about on horseback. Whenever food rich in fat can be tolerated by the stomach, it will produce like effects, and hence the occasional value of bacon, pork chops, caviare, suet, yolks of eggs, and the produce of the dairy, such as milk, cream, and butter.

Cod-liver Oil.—All good food must consist of a proper mixture of albuminous, fatty, and mineral principles. The two former, holding the third in solution, after being prepared by the digestive fluids form a molecular fluid—the chyle—out of which the blood is formed. In Phthisis, however, the process of chylification is impaired; the fatty constituents of the food are not separated from it and assimilated, or they are deficient, as very commonly results from a dislike to fatty substances. In either case, the blood abounds in the albuminous elements, and when exuded into the lungs, as we have seen, forms tubercle. To induce health, it is necessary to restore the nutritive

[1] See the author's Treatise on the Restorative Treatment of Pneumonia, 3d edit. 1866.

elements which are diminished, and this is done directly by adding a pure animal oil to the food. By so doing, we form richer chyle and better blood; we restore the balance of nutrition, which has been disturbed; respiration is again active in excreting more carbonic acid gas; the tissues once more attract from the blood the elementary molecules so necessary for their maintenance; the entire economy is renovated, so that while the histogenetic processes are revived, the histolytic changes in the tubercle itself also are stimulated, and the whole disappears. We have previously seen that food rich in fat will occasionally produce these effects, but then the powers of the stomach and alimentary canal must not have undergone any great diminution. In most cases, however, the patient is unable to tolerate such kind of food, which is not digested. Under these circumstances, cod-liver oil is directly indicated, by giving which we save the digestive apparatus, as it were, the trouble of separating fluid fats from the food. By giving the oil directly in quantity, a large proportion of it enters the system, unites with the albumen, and thereby forms the molecular basis so essential for the chyle. Since the days of Liebig, chemists have generally supposed that albumen forms the basis of the tissues, and is a flesh-former, while fat is necessary for respiration, and by its decomposition furnishes heat. An unacquaintance with histology is the cause of this error, fat being demonstrably necessary for the development and support of muscle and of every tissue. This has recently been further shown by the investigations into the diet of laborers by E. Smith, the feeding of animals by Lawers and Gilbert, and the experiments of Haughton, Frankland, Fick, and Wislicenus. Hence the universal craving and necessity for fat by the vigorous and working man, whilst a dislike to it is a strong symptom of inherent weakness, and an incapability of assimilating it the chief cause of tubercular disease.

It was in the years 1840 and '41 that I found cod-liver oil used very generally in the German hospitals in all scrofulous and phthisical cases. In England it at one period had been employed in Manchester, at the beginning of the century, by Drs. Kay and Bardsley, in rheumatism, but had fallen into neglect. In the hospitals of Heidelberg and Berlin, I watched with great care the effects of the oil in several cases of Consumption, and satisfied myself of its remarkable powers as a nutrient, under circumstances which in British hospitals would have been attended with little hope. In the autumn of 1841, therefore, I published a monograph containing an account of what was then known of this substance, and recommended it especially to my countrymen, both from theoretical and practical grounds, as a valuable remedy in Phthisis.[1] The first physician who tried it in the Royal Infirmary of Edinburgh was Dr. Spittal; but so little were druggists acquainted with the oil, that I found on visiting his wards that all the patients were taking linseed oil. The same mistake had previously occurred to Rush, in Berlin. I was therefore obliged to get it made expressly, which, after a time, was done by the Messrs. Parker and Co., oil merchants, Leith-walk, who for many years made the purest cod-liver oil in Great Britain, which they sent over the country at the moderate rate of 16s. a gallon. When in the course of time it was asked for in London, Mr. Jacob Bell, the eminent druggist in Oxford-street, caused a very pure oil to be made from the livers of the cod, which, however, was so expensive, that he dispensed it at the rate of half-a-crown an ounce. I was consequently written to by numerous persons in London and elsewhere, and was thus the means of causing hundreds of gallons to be distributed by the Messrs. Parker to all parts of the country. Gradually, its value was generally appreciated throughout Scotland, and was extending in England, when it was tried in the Brompton Consumption Hospital of London. In 1849, Dr. Williams published a paper, in which, from extensive trial of the remedy, its value and mode of action were confirmed, a result still further supported by the Report of the Brompton Consumption Hospital, published in 1851. Since then the employment of cod-liver oil in Phthisis has been almost universal, and has contributed in no small degree to remove that hopelessness and despair with which the treatment of the disease had been previously accompanied. In 1841, it was unknown in our druggists' shops, except here and there, where it was kept in small quantities for the use of tanners, who, curiously enough, had discovered that it possessed far superior power to all other fatty substances in penetrating and softening leather. At that time the eminent Edinburgh druggists, Duncan and Flockhart, did not dispense one gallon in the year, whereas at present they dispense between six and seven hundred gallons annually.

A most extensive experience has now amply confirmed the opinion I published regarding it thirty years ago—viz. "That no remedy has so rapidly restored the exhausted powers of the patient, improved the nutritive functions generally, stopped or diminished the emaciation, checked the

[1] See the author's treatise on the Oleum Jecoris Aselli, &c., 1841; also with Appendix, 1847.

perspiration, quieted the cough and expectoration, and produced a most favorable influence on the local disease. Many individuals presenting the emaciation, profuse sweats, constant cough and expectoration, as most prominent symptoms, with a degree of weakness that prevented their standing alone, after a few weeks' use of it, are enabled to get up with ease and walk about, with a visible improvement in their general health, and an increased amount of flesh." Thus it must be regarded as an analeptic (ἀναλαμβάνω, to repair) or general restorative, being digestible where no other kind of animal food can be taken in sufficient quantity to furnish the tissues with a proper amount of fatty material. It is not by a chemical so much as by a histological process that the result is produced.

By some, however, it is supposed that the superiority of cod-liver oil over other fatty substances is owing to the iodine, bromine, resin, and other medicaments it contains. But the quantity of these drugs in cod-liver oil is very minute, and it has been abundantly proved that no combination of them given internally has any effect on the progress of Phthisis. Hence the idea of giving a watery extract of cod-liver oil, when the oil cannot be taken, appears to us to be erroneous in theory, and unlikely to succeed in practice. On the other hand, there are so few persons who cannot take the oil when it is absolutely necessary, that such preparations need be very seldom employed. I have known many individuals who prefer the brown and apparently nauseous to the light and comparatively pure oil. In all cases, that kind of oil is best that is most readily tolerated by the stomach. Those who at first express dislike to the remedy, by a little perseverance may be made to take it readily; if not, they should try whether it be retained best immediately before, immediately after, or in the intervals of meals. The crunching a biscuit, or a lump of sugar on which there has been placed a drop of some essential oil, sometimes removes the difficulty. At others, a little coffee, orange wine, or a bitter, and occasionally slightly warming the oil, so as to render it more fluid, answer well. By these or similar methods, it is rare indeed that the oil cannot be taken. [The addition of ether to cod-liver oil has lately been recommended.—H.]

Numerous substitutes have been proposed for cod-liver oil, such as shark, dugong, and skate oils, cocoa-nut oil, neat's-foot oil, &c. Any of these substances, including cream and butter, so long as they can be assimilated, and do not prove purgative, are beneficial in Phthisis. It will be found, however, that, of all oleaginous matters known, cod-liver oil is the most generally useful and the best. Dr. Baur, of Tubingen, recommended that it should be used externally, but extensive trial has demonstrated what physiology teaches, viz. that the skin, being only slightly pervious to substances from without, cannot be made the vehicle for introducing nutritive matter. Dr. Buist, of Aberdeen, recommended injections into the rectum, but objections to the use of constant enemata in this country are insurmountable, and although useful as a temporary measure, cannot be made available to a sufficient degree for the cure of a disease like Phthisis. What then is really required is not oil added directly to the blood, but oil digested and emulsionized by the pancreatic and other intestinal fluids; a truth which has induced Dr. Dobell to recommend that before administration it should be mixed with pancreatic juice.

In most cases where there is fever, rapid pulse, and furred tongue, cod-liver oil is no more tolerable than food. Under such circumstances it should not be insisted on. It will also be judicious, when taken for any length of time, to intermit its use now and then for a few weeks, and give in its stead a vegetable bitter. By attention to this circumstance, the medical practitioner will easily satisfy himself that in this substance he possesses a most valuable means of prolonging life, and sometimes even of causing permanent cure in Phthisis Pulmonalis, especially when the benefits it confers are conjoined with the other methods of general treatment to be noticed.

An additional benefit has followed the obvious good effects of cod-liver oil in Phthisis, as stated by Dr. E. Smith, who says: "A prime reason of the good which has resulted from the use of the cod oil is the regular supply of fat to persons who otherwise would not have taken it in due quantity; and a great merit in the introduction of it to general use is in having led inquirers to prove the very important part which fat plays in the animal system, and the real necessity for it which exists in all persons and particularly in the young."[1] This observation evidently results from the histology, pathology, and treatment of Phthisis which for so many years we have endeavored to impress upon the profession.

Pure Atmosphere.—If it be essential for the purpose of nutrition to supply the blood with those materials which are necessary for building up the tissues and compensating the waste they undergo during their action, it is equally so that such materials should be properly prepared and fitted for the purposes to which they are to be applied. Of the various

[1] On Consumption, p. 348.

processes necessary to this end there can be little doubt that that of respiration is the chief, the object of which is constantly to introduce into the blood from the atmospheric air a certain amount of oxygen, and constantly to give off from the blood to the air a corresponding amount of carbonic acid gas. If the lungs be feeble or diseased, their action is of course diminished, a circumstance which only renders it the more necessary that no difficulty to oxygenation of the blood should be allowed to originate from a deteriorated constitution of the air itself. But this truth is one which it is exceedingly difficult to impress upon patients, the irritability of whose chests and whose susceptibility to cold induce them to close the doors and windows, and thus prevent fresh air from entering their rooms. Now, while the giving off carbonic acid gas by the lungs makes no impression upon the mass of the atmosphere at large, it soon sensibly deteriorates the amount of air inclosed in a moderate-sized room, the breathing of which is most destructive to the phthisical invalid. Instead of inhaling only oxygen and nitrogen, and expiring carbonic acid gas and nitrogen, they take in a sensible amount of carbonic acid at each inspiration, which poisons the arterial blood, renders it less fit for nutrition, and irritates and burdens the lungs, occasions languor, bad appetite, pallor of countenance, and indeed every evil which it should be the aim of the physician to remove. Moreover, good diet and cod-liver oil must be useless unless a vigorous respiration exists at the same time, as they tend to increase the carbonaceous elements in the frame, which are mostly excreted by the lungs. A proper ventilation of the rooms occupied by the patient is therefore absolutely essential, and this rule especially applies to the sleeping room. The majority of mankind spend one-third of their life in sleep, while the invalid often remains in the bed or bedroom much longer. How important then is it to secure a pure breathing air during this period!

It is now twenty-five years since I became convinced of the injury of shutting up patients in their rooms during winter, and regulating the temperature, as was formerly the custom. A young man, with cavities in his lungs, who had borne confinement in this way tolerably well for a winter, found it so irksome on a second trial, that on one occasion he went out and walked to the top of Arthur's Seat. Instead of being worse, he that day ate his dinner with appetite, all his symptoms were moderated, and under the combined influence of pure air and exercise he not only was better, but ultimately worked out a perfect cure, and is now alive in good health. Since then I have had abundant opportunities of satisfying myself of the great advantages to be derived from securing free ventilation and pure air to consumptives.

These points are dwelt on as forcibly as possible, because it must be admitted that, partly as the result of custom or prejudice and partly in consequence of the severity and changeableness of the climate, a good ventilation of the house and sleeping room is, in this country, a matter of extreme difficulty. In all cases, however, it merits the especial attention of the physician. Hence he should regard the position of the house, the nature of the prevailing winds, the windows of the sitting-room, and the place in it occupied by the patient, how the bed is placed in reference to the door and windows, &c. The great end he should aim at is to surround his patients with *as much pure air as possible, consistent with warmth and absence of draughts*, a problem often very difficult to work out. There should be no curtains round the bed, an open fire should burn in the room during winter, in itself an excellent ventilator, the bed should be placed in a position free from the direct draught between the fire and the door or window, and only a moderate temperature permitted, as when in bed the patient ought not to feel cold. In summer good ventilation should be secured by letting down the windows an inch or so at the top — an excellent method, first strongly insisted upon by Dr. McCormack, of Belfast, and one which, indeed, is at all times available in this country; unfortunately, abroad, the construction of the windows does not admit of it. The necessity of constantly breathing pure air should prevent the phthisical patient from attending crowded assemblies, *tables d'hôte*, theatres, concerts, or any amusements where the atmosphere must necessarily be deteriorated, and which, being breathed for hours, almost invariably exacerbates the symptoms and increases the malady. It is in consequence of the facility of breathing a purer air all day, and the necessary avoidance of crowded and closed rooms at night, that I am persuaded the upper classes of society experience much of the good effects of residing in certain places famed for their climate—the next point we must consider.

Climate. — It was formerly supposed warm climates were beneficial for consumptive patients, and artificially heated temperatures, cow-houses, and other contrivances were had resort to, to compass this end. But it will be invariably observed that unaccustomed warmth, the excessive heat of summer and autumn, or the climate of India and other tropical countries, is most injurious. Continuous frost and cold are in themselves beneficial,

but by preventing the individual taking exercise in the open air they are not on that account to be recommended. What is really required is a cool temperate climate, free from great alternations of temperature, which should range from 55° to 66° Fahr. during the day, and from 45° to 55° at night. The air should be dry, or with only slight moisture, and a clear bright sun. Such an exhilarating climate, in which exercise can be taken almost daily in the open air during the winter and spring months, is the best for the consumptive patient. It exists to the greatest perfection on the north shore of the Mediterranean, between Cannes and Savona in the western, and between Spezzia and Pisa in the eastern Riviera. It may also be found in various places on the southeast coast of Spain, especially at Malaga ; on the north African shore, such as Algeria and Egypt, and many other places. In the western hemisphere, suitable places may be found, especially in the islands of the West Indies, and in Australia the southern shores of Victoria. The native of the British Isles who visits the sheltered nooks of the south European shore between Cannes and Pisa will be struck with the bright sun, clear atmosphere, genial yet bracing air, steady temperature, verdure, and brilliant vegetation which surround him from January to March[1]—months which at home are characterized by frost, snow, rain, fog, gloom, bleak winds, and a barren vegetation. After this period, however, the picture is reversed. Then a hot and sultry atmosphere, a scorching sun, an intolerable glare, innumerable mosquitoes, a brown and burnt-up vegetation exist, while at home there prevail a genial atmosphere, cool breezes, moderate sunbeams, a varying sky, an emerald foliage, and a charming variety of mountain and lake which gives all that can be desired. I do not know a better winter residence for the invalid than some sheltered bay in the western Riviera where, in consequence of the sea being immediately in front of his house, and innumerable little valleys of the Alps close behind it, he can at all times protect himself from wind, from whatever quarter it may blow. Many observations have satisfied me that the still, warm, and moist relaxing atmosphere, though of the greatest service in cases of asthma, is injurious to the phthisical invalid. Great care should be taken to avoid sharp winds, and especially east winds. This at Mentone is readily done by walking out of the back door of your house directly into some protected Alpine valley. In this country, however far we go west, it is escaped with difficulty, and as a general rule North Britain more especially should be avoided from January till the end of May. In summer and autumn, on the other hand, I am satisfied that the cool atmosphere of Scotland cannot be surpassed in benefit, especially as we find it on the shores of a Highland lake, admitting of every variety of exercise, active and passive, in the open air. Indeed, whatever advantages may result from a well-chosen winter residence, carelessness in fixing on a proper habitation during summer will more than counterbalance the good previously obtained. It is by perseverance in well-doing that the great end of cure is to be arrived at.

For winter, the best climate for the consumptive invalid in this country is the south coast, extending from Hastings on the east to Penzance on the west side, including the several stations of Bournemouth, Ventnor, Sidmouth and Torquay. In Scotland, Rothsay, and in Ireland, Cork, are the best stations. To the large mass of persons who cannot avail themselves of even these advantages, every opportunity should be seized on of going out when the weather admits of it. It is not so much to a foreign climate itself as to the facility it affords for enjoying exercise, and free atmosphere, without the risks that prevail in Great Britain, that the benefit is to be attributed. With proper care, however, much may be done at home, and many cases have been permanently cured in this country by means of hygienic treatment, conducted on the principles we are now advocating.

For summer, the west coast of Scotland; and especially the beautiful bays on the shore of Loch Lomond, near Tarbet, offer the best residence for the consumptive. Here the immediate neighborhood of Loch Long furnishes the visitor with all the advantages of a marine as well as of a fresh-water lake, both which are so situated that the most perfect protection from wind, combined with shade, is close at hand. It cannot be too strongly impressed upon the patient that carelessness in summer too often more than counterbalances the good results that have been obtained in winter.

Exercise.—The best stimulant for nutrition is appropriate exercise, which by accelerating the circulation and respiration, and causing natural wasting of the tissues excites the demand for substance to repair it. It will generally be found useless to give nutriment, even when combined with pure air and good climate, unless by means of exercise, air be forced

[1] On this subject I cannot too strongly recommend the perusal of Dr. Henry Bennet's work, "Winter in the South of Europe," although the views expressed in the text are derived from personal experience, and careful examination of the great advantages referred to.

into the lungs in somewhat increased quantity, and circulated by means of the blood throughout the system. And here it is that favored localities are of so much value, by tempting the invalid out of the house, and permitting him to remain there, without encountering cold, wind, rain, or other risks to which he is exposed in this country. All exercise, however, should stop short of considerable fatigue. I say considerable, because some patients are always indisposed to move, and plead weakness and fatigue as incapacitating them from any exercise whatever. Walking, or riding on horseback, are the best kinds of exercise when weakness is not great. Slowly climbing a hill brings all the muscles into action, and is a good stimulant to the respiratory and circulatory systems. All violent, sudden and unequal exertions should be avoided. Reading or speaking aloud, singing or practising upon some wind instrument, may be permitted in moderation, when the disease is not active, but should never be long continued. As a general rule they are injurious. It is often better to take a little exercise at a time, but frequently in the course of the day, and to continue it regularly and methodically, gradually increasing its amount and varying its character as the strength improves.

For those who are weak and feel soon exhausted, passive exercise is best, such as in a carriage or in a boat, of course well wrapped up and protected from the wind. In summer, sitting or lying, well supported, in a boat pulled on a Highland lake, while, for occupation, reading, mixed with a little fishing, and the conversation of a pleasant companion—the various tints and outlines of the landscape also serving occasionally to occupy the attention—is perhaps the most salubrious kind of exercise for the not over-weakened invalid. For the same reason long voyages at sea are beneficial. I can speak with confidence of the three months' voyage to Australia by the Cape of Good Hope, commencing about the end of October. The climate is all that could be wished for, the trade-winds assist the vessel forward, the sea breeze is invigorating, and the life on deck all that could be desired. I have known many persons, very ill on leaving, lose all their symptoms before landing at Sydney or Melbourne. Sultry heat on shore must then be carefully avoided, and the visiting neighboring mountains or Tasmania becomes necessary in summer, in order to avoid the enervating effect of extreme heat. The return voyage should be carefully considered, and the winter at Cape Horn especially avoided.

When none of these methods are available, sitting out in the open air should always be insisted on, in a garden, on a balcony, or even at an open window, anything being better than remaining shut up in a room from morning to night.

In this, as in all other matters concerning hygiene, the patient requires to be cautioned and carefully watched. For if some feel disposed to do too little, others imagine they cannot do too much. Under the idea that riding was beneficial, I have known a man hire a horse, and gallop about until he was so exhausted that he did not recover for a fortnight. Others in foreign hotels have taken rooms at the top of the house to obtain pure air, without considering the excessive toil imposed upon them by having to climb the lofty stairs. Others take villas in the neighborhood of towns, and are thus led into a daily fatiguing walk greater than their strength will sustain. Again, free exposure to the air must be conjoined with avoidance of draughts and cold winds. The rapid motion of a carriage through a dry bracing atmosphere is too much for the invalid, who should proceed slowly. Carelessness and often an unacquaintance with these dangers are constantly producing mischief, so that the watching and regulating these matters will require all the vigilance of the practitioner.

Bathing.—There is no doubt that the relation between the skin and lungs is very intimate, a fact better observed perhaps in Phthisis than in any other disease. When the lung can no longer exhale the large amount of watery vapor which is required, it is separated by the skin as insensible or sensible perspiration. Any sudden cold or chill affecting the skin is at once communicated to the lungs by reflex action, and excites irritation and cough. Now this susceptibility of the skin, so far from being prevented, is fostered and increased by constantly living in warm rooms, wrapping up too closely in shawls or furs, warm bathing, ointments, &c. &c. What is required is, that the skin should be kept constantly clean, and the epidermis and sebaceous matter that obstructs the orifices of the ducts daily removed by cold bathing whereby the organ is gradually accustomed to the application of a lower temperature, and rendered less liable to be affected by changes in the atmosphere or wind. In the majority of cases also a momentary plunge into the cold bath produces a glow of heat and pleasant feeling of reaction, exciting the capillary circulation of the surface, and relieving congestion in the lungs. When, however, in consequence of weakness, such reaction is not experienced, but in its stead, shivering, headache, and continued cold, then either a tepid bath should be employed, or the sitz bath, and sponging rapidly only the chest and throat should be practised. There is no better protection against catching frequent colds than daily sponging the chest

with cold water. The neck and chest, however, should always be covered, the growth of beard and moustache in men encouraged, whilst women should avoid low dresses, and always be prepared with an extra shawl to throw round the shoulders, even in going from one room to another through an exposed lobby. Respirators are not useful in conveying warm air into the lungs, nature having carefully provided for this, but by acting as extra pieces of clothing, and protecting the skin of the face. An ordinary comforter, and a small shawl held in the hand to be applied to the face on encountering a sudden gust of wind, is a better contrivance.

From what has been now stated with regard to the general or hygienic treatment of Phthisis Pulmonalis it will, we trust, be apparent that all the means spoken of unite to produce one result, and that no one of them alone can be depended upon. It will be of little use giving good diet or cod-liver oil, unless a pure atmosphere enter the lungs so that chylification may produce good sanguification, while these in their turn are directly stimulated by exercise and judicious bathing. All these operations work together for good, the object being to stimulate the whole nutritive functions, augment appetite, gradually increase the strength, arrest the onward progress of the disease, and initiate in it that retrograde process formerly described, which shall terminate in health. To arrive at this end, however, a special treatment will be required for each individual case, which we must next proceed to describe.

Special Treatment of Phthisis Pulmonalis.—It is to the undue importance so frequently given to the special as distinguished from the general treatment of Phthisis that the former want of success may be attributed. The management of individual symptoms and the administration of drugs, so far from being the chief, should invariably be the subordinate part of our object, and this for the obvious reason that, if nutriment succeed in checking the disease, the symptoms will disappear of themselves. At the same time it must necessarily happen in the course of every case that various symptoms and complications will press themselves upon our notice, and their palliation or removal, while still continuing our general efforts at cure, is always a matter of great importance. It is only by studying individual examples of the disease, observing the numerous and varied combinations and indications that each presents, that the difficulties the practitioner has to combat in this way can possibly be understood. I have too frequently seen patients lying in bed, enervated, without appetite, sweating at night, and apparently sinking, with a mass of bottles and boxes at the bedside bewildering to contemplate—each of these it is imagined has some special symptom or purpose to fulfil—such as lozenges, drops, and mixtures, to relieve coughs; opiates and sedatives, to cause sleep and diminish irritability; catechu, gallic acid, tannin, and acetate of lead, to check diarrhœa or arrest hæmoptysis; sulphuric acid, to relieve sweating; chalk and antacids, to combat acidity and dyspepsia; quinine, iron, or bitters, as tonics; wine, to support strength; cod-liver oil, &c. &c. All these I have seen administered at intervals about the same time, so that the stomach, drenched with drugs, is utterly prevented from performing its healthy functions. Under such circumstances suspending all such supposed remedies, or preventing the patient from having recourse to them at will, is often the best introduction to an improvement, which the cold or tepid bath, insisting on their getting up and going into the open air, has, much to their surprise, tended to increase. It follows that, in all our attempts to relieve symptoms, the utmost care should be taken not to interfere with the far more important object of arresting and ultimately curing the disease by general treatment. The various phenomena that present themselves, therefore, should be managed as follows.

Loss of Appetite and Dyspepsia.—These are the most constant and important symptoms of Phthisis, inasmuch as they interfere more than any other with the nutritive processes. If food, or its substitute, cod-liver oil, cannot be taken and digested, it is in vain to hope for amelioration. Here we should avoid a mistake into which the inexperienced are very liable to fall. Nothing is more common than for phthisical patients to tell their medical attendants that their appetite is good, and that they eat plentifully, when more careful inquiry proves that the consumption of food is altogether inadequate, and that they loathe every kind of animal diet. We should never be satisfied with general statements, but determine the kind and amount of food taken, when sufficient proof will be discovered, in the vast majority of cases, of the derangement, formerly alluded to, of the appetite and digestive powers. Very commonly also there will be acid and other unpleasant tastes in the mouth, loathing of food, and other dyspeptic symptoms. In all such cases, especially if too much medicine has been already given, the stomach should be allowed to repose itself before anything be administered, even cod-liver oil. Sweet milk, with toasted bread, and small portions of meat nicely cooked, so as to tempt the capricious appetite, should be tried. Then ten drops of the sp. ammon. aromat., given every four hours in a wine-glassful

of some bitter infusion, such as that of calumba or gentian, with a little tinct. aurantii, tinct. cardamomi, or other carminative. In this way the stomach often regains its tone, food is taken better, and then cod-liver oil may be tried, first in teaspoonful doses, cautiously increased; or other forms of fat, such as pork fat, bacon, suet, or butter, may be tried. Should this plan succeed, amelioration in the symptoms will be almost certainly observed.

Nausea and Vomiting.—Not unfrequently the stomach is still more deranged; there is a feeling of nausea and even vomiting on taking food. In the later stage of Phthisis, vomiting is also sometimes occasioned by violence of the cough, and the propagation of reflex actions, by means of the par vagum, to the stomach. In the former case, the sickness is to be alleviated by carefully avoiding all those substances which are likely to occasion a nauseating effect, by not overloading the stomach, but allowing it to have repose. Here also, in cases where too much medicine has been administered, a suspension of all medicaments for a few days will frequently enable the practitioner to introduce nourishment cautiously with the best effect. I have found the following mixture very effectual in checking the vomiting in Phthisis: ℞. Naphthæ medicinalis ʒj; tinct. cardamomi comp. ʒj; mist. camphoræ ʒvij. M. ft. mist., of which a sixth part may be taken every four hours. When it depends on the cough, those remedies advised for that symptom should be given. I have tried emetics for the relief of nausea and vomiting, but with no good result.

Cough and Expectoration.—At first the cough in Phthisis is dry and hacking. When tubercle softens or bronchitis is present, it becomes moist and more prolonged. When excavations exist, it is hollow and reverberating. In every case cough is a spasmodic action, occasioned by exciting the branches of the pneumogastric nerves, and causing simultaneous reflex movements in the bronchial tubes and muscles of the chest. The expectoration following dry cough is at first scanty and muco-purulent, and afterwards copious and purulent. When it assumes the nummular form,—that is, occurs in viscid rounded masses, swimming in a clear fluid mucus,—it is generally brought up from pulmonary excavations. The accumulation of the sputum in the bronchial tubes is an exciter of cough; and hence the latter symptom is often best combated by those means which diminish the amount of sputum. When, on the other hand, the cough is dry, those remedies should be used which diminish the sensibility of the nerves. In the first case, the amount of mucus and pus formed will materially depend on the weakness of the body and the onward progress of the tubercle. Hence good nourishment and attention to the digestive functions are the best means of checking both the cough and the expectoration; whereas, giving nauseating mixtures of ipecacuanha and squills is perhaps the worst treatment that can be employed. There is no point which experience has rendered me more certain of than that, however these symptoms may be palliated by cough and anodyne remedies, the stomach is thereby rendered intolerant of food, and the curative tendency of the disease is impeded. On the other hand, nothing is more remarkable than the spontaneous cessation of the cough and expectoration on the restoration of the digestive functions and improvement in nutrition. When the cough is dry, as may occur in the first stage, with crude tubercle, and in the last stage, with dry cavities, slight counter-irritation is the best remedy, employed in various forms. Opium may relieve, but it never cures. The occasional use of the sponge saturated in a solution of nitrate of silver is frequently of the greatest service, especially when from irritation of the fauces or larynx vomiting is occasioned.

There is a period in the history of chronic Phthisis when the cavities become dry and the sputum inspissated, tough, and difficult to expectorate. The practitioner is then frequently asked for some medicine to loosen the phlegm, relieve the feeling of tightness or compression in the chest, and dyspnœa. Under these circumstances, in no case should he resort to expectorants and opiates. The patient should be instructed that these are favorable symptoms, and indicate healing and cicatrization going on in his chest. Instead of relaxing, now is the time to persevere in avoiding palliatives which nauseate and depress the system. A few drops of sulphuric ether in camphor julep, diminishing alarm, and a little quietude, constitute all the treatment required. [An excellent combination for such cases, which does not nauseate, is of ammonium carbonate, 2 or 3 grains at a dose, with syrup of wild cherry bark, in teaspoonful doses.—H.]

Pain.—It is very surprising to what an extent tubercular disease of the lung may occasionally proceed, without causing inconvenience in the chest. Frequently there are sensations of constriction or oppression, which, however, scarcely excite attention; or from their fugitive character are attributed to any cause but the right one. Occasionally there is a fixed pain in the affected side, which is increased on coughing. This more especially occurs when there is chronic pneumonia or pleurisy. The best method of relief is to keep

the parts at rest as much as possible, and apply warm fomentations or a hot poultice. Slight counter-irritation with tincture of iodine may also be tried.

On the other hand, leeches and cupping, though they may give relief, are opposed to the general principle of supporting the strength, and should be avoided. The same may be said of blisters, croton oil, tartar emetic ointment, and the moxa. I have long satisfied myself that severe counter-irritation is of no real benefit, whilst it produces an amount of suffering that irritates, and frequently does harm. Opiates are also injurious, by destroying the appetite and increasing the perspirations. At the same time, if pain be very distressing and long-continued, and especially if it destroy sleep, some anodyne must sooner or later be had recourse to. Under these circumstances I have found chlorodyne derange the appetite, tongue, and stomach less than any other remedy of this class. Recently, chloral in fifteen or twenty-grain doses has seemed to me to act as a pure hypnotic and cause less disturbance to the economy than other remedies. Again, when all curative efforts are obviously useless, and death is approaching, palliatives need no longer be withheld. Then, all hopes of course being abandoned, relief of pain, if it exists, becomes our chief duty. But even then it should be effected with caution and discretion, otherwise the discomfort and increase of other symptoms in the patient will more than counterbalance the temporary benefit obtained.

[There is reason for attaching some importance to the suggestion of Dr. E. Smith, that small or moderate doses of opium or morphia, given (in advanced cases) through the day as well as at night, may lessen the waste of substance and of energy, the palpable occurrence of which has given rise to the name, *consumption*. —H.]

Diarrhœa. — This is a very common symptom throughout the whole progress of Phthisis, at first depending on the excess of acidity in the alimentary canal, to which we have alluded, but in advanced cases connected with tubercular deposit and ulceration in the intestinal canal. The best method of checking this troublesome symptom is by improving the quality and amount of the food. The moment the digestive processes are renovated, this, with the other functional derangements of the alimentary canal, will disappear. Hence at an early period we should avoid large doses of opium, gallic acid, tannin, and other powerful astringents, and depend upon the mildest remedies of this class, such as chalk with aromatic confection, or an antacid, such as a few grains of carbonate of potash. When, on the other hand, in advanced Phthisis, continued diarrhœa appears, and is obstinate under such treatment, then it may be presumed that tubercular disease of the intestine is present, and the stronger astringents with opium may be given as palliatives.

Hæmoptysis.—This symptom sometimes appears suddenly, as we have seen, in individuals in whom there has been no previous suspicion of Phthisis, and in whom, on careful examination, no physical signs of the disease can be detected. On other occasions, the sputum may be more or less streaked with blood; and lastly, it may occur in the advanced stage of the disease, apparently from ulceration of a tolerably large vessel which may be dilated or aneurismal. In all these cases the best remedy is perfect quietude, and avoidance of every kind of excitement, bodily and mental. Astringents have been recommended, especially tannin, gallic acid, acetate of lead, and opium; but how these remedies can operate, I am at a loss to understand; and I have never seen a case in which their administration was unequivocally useful. Can it be supposed that either of these substances can be absorbed into the blood in such quantity as to render that fluid more capable of coagulating in the lung where the vessel is ruptured? I have now met with several cases where supposed pulmonary hemorrhage really originated in follicular disease of the pharynx or larynx, and, with the supposed phthisical symptoms, was removed by the use of the probang and nitrate of silver solution.

Sweating I regard as a symptom of weakness, and therefore as a common, though by no means a special one in Phthisis. Here, again, the truly curative treatment will consist in renovating the nutritive processes, and adding strength to the economy. It will always be observed that, if cod-liver oil and good diet produce their beneficial effect, the sweating, together with the cough and expectoration, ceases. On the other hand, giving acid drops to relieve these symptoms, as is the common practice, by adding to the already acid state of the alimentary canal, is directly opposed to the digestion of the fatty principles, which require assimilation.

It should not be forgotten that consumptive patients, and all those suffering from pulmonary diseases, are especially sensitive to cold. The impeded transpiration from the lungs in such cases is counterbalanced by increased action of the skin, which becomes unusually liable to the influence of diminished temperature. Again, cold applied to the surface immediately produces, by reflex action, spasmodic cough and excitation of the lungs. Every observant person must have noticed how cough is induced by crossing a lobby, going out into the open air, a draught of

wind entering the room, getting into a cold bed, &c. &c. The mere exposure of the face to the air on a cold day takes away the breath, introduces cough, and obliges the patient instinctively to muffle up the mouth. The numerous precautions, therefore that ought to be taken by the phthisical individual, should be pointed out, especially the necessity of warm clothing, to which large additions should be made on going out into the air. Thus, covering the lower part of the face is important as a means of extra clothing, and not as a means of breathing warm air, as the favorers of respirators imagine. The patient should always sit with his back to the horses or to a steam-engine, and if by accident his shoes or clothes become wet, they should be changed as soon as possible. In the house ladies should have a shawl near them, to put on in going from one room to another, in descending a stair to dinner, &c. By attention to these minutiæ, much suffering and cough may be avoided.

Febrile Symptoms.—The quick pulse, general excitement, loss of appetite, and thirst, which are so common in the progress of phthisical cases, are dependent on the same causes as those which induce symptomatic fever in general. Vascular distension, resulting in exudation and its absorption, is proceeding with greater or less intensity in the lungs, and frequently in other organs. This leads to nervous irritation and increase of fibrin in the blood, accompanied by febrile phenomena. The intensity of these is always in proportion to the activity of local disease, or to the amount of secondary absorption going on from the tissues, or from morbid deposits. Nothing is more common than attacks of so-called local inflammations in Phthisis, and the careful physician may often determine by physical signs the supervention of pleurisy, pneumonia, or bronchitis on the previously observed lesion, and not unfrequently laryngitis, enteritis, or other disorders. In such cases, nature herself dictates that the analeptic treatment, otherwise appropriate, is no longer applicable—food disgusts, and fluids are eagerly demanded. Under these circumstances, it has been common to apply leeches to the inflamed part, and extract blood by cupping, measures which undoubtedly cause temporary relief, but which are wholly opposed to the plan of general treatment formerly recommended, and to what we know of the pathology of the disease. Every attack of febrile excitement is followed by a corresponding collapse, and it should never be forgotten that, in a disease which is essentially one of weakness, the patient's strength should be husbanded as much as possible. Hence the treatment I depend on in such circumstances consists of at first the internal administration of the neutral salts, combined with diuretics, in order to favor crisis by the urine. Subsequently quinine is undoubtedly advantageous. I have satisfied myself that such attacks are not to be cut short by leeches or cupping, and although in many cases, as previously stated, temporary relief is produced, the exposure of the person, and unpleasant character of the applications, the trickling of blood, and wet sponges, as often irritate, and give rise to unnecessary risk. Still there are cases where topical blood-letting, if it cannot be shown to have advanced the cure, cannot be proved to have done harm; but these cases, as far as my observation goes, are very few in number. In the rapidly febrile cases, or the so-called instances of acute Phthisis, mercury has been recommended, but has never produced the slightest benefit.

Debility.—This is a very common symptom of Phthisis from the first, and frequently leads the patient into indolence both of mind and body, a condition very unfavorable for the nutritive functions, upon the successful accomplishment of which its removal depends. It is to remove the weakness that tonics have been administered, but I have never seen quinine, bitter infusions, or even chalybeates, of much service alone, while the continual use of nauseous medicine disgusts the patient, and interferes with the functions of the stomach. Neither have I ever been able to satisfy myself that the hypophosphites of soda or of lime, or the syrup of those phosphates and iron, have ever been of service. In all cases, the removal of debility is to be accomplished by counteracting the dyspeptic symptoms, giving cod-liver oil, an animal diet, and improving the appetite by gentle exercise and change of scene. Should the practitioner succeed in renovating the nutritive functions, it is often surprising how the strength increases, in itself a sufficient proof as to what ought to be the method of removing the debility. I have frequently seen patients who have been so weak that they could not sit up in bed without assistance so strengthened by the analeptic treatment, that they have subsequently walked about and taken horse exercise without fatigue, and this after all the vegetable, mineral, and acid tonics have been tried in vain.

Despondency and Anxiety.—It is impossible for the careful practitioner to avoid noticing the injurious influence of depressing mental emotions on the progress of Phthisis. Indeed the worst cases are those of individuals with mild, placid, and unimpassioned characters, who give way to the feelings of languor and debility which oppress them. Such persons are most amiable patients—they give no trou-

ble—anything will do for them—they resign themselves to circumstances, and state that they are eating well and getting better up to the last. These are cases of bad augury, for it is exceedingly difficult to inspire them with sufficient energy to take exercise, or to carry out those regulations which are absolutely essential to renovate the appetite and the nutritive functions. Such persons are benefited by slow travelling, cheerful society, and everything that can elevate the spirits, and, insensibly to themselves, communicate a stimulant to the mental and bodily powers. Anxiety, on the other hand, though it may sometimes depress and interfere with the digestive functions, is often a most useful adjunct to the physician. Those who experience it are most careful of their health, sometimes indeed too much so; but, if once satisfied of the benefit of any particular line of treatment, they pursue it with energy. These are cases of good augury, and most of the permanent cures I have witnessed have been in such persons—medical men, and others acquainted with the nature of their disease, who have exhibited resolution and a noble fortitude, who have bravely struggled against local pain, general debility, and nervous fear, and literally fought the battle of life with the greatest success.

When the disease has been arrested, all the symptoms have disappeared, and even some degree of *embonpoint* returned, the patient must still be careful, still consider himself an invalid, and continue to pursue the hygienic regulations which have proved so beneficial. These, however, will not materially interfere with his enjoyment of life, or even the pursuit of active business or professional life. Amongst the poorer classes, it will be more difficult to obtain such handiwork or occupation as may not be injurious. In order to live, however, they must exchange their unhealthy for more healthy modes of life. As a general rule, the dwellers in towns should seek the country, and the inhabitants of rural districts change the scene of their labors—always remembering that it is not mere place that can benefit, but the opportunities it may offer for carrying out that improvement in the nutritive functions we have endeavored to show is so necessary.

Local Treatment.—It has not failed to suggest itself to medical practitioners that remedies might be useful if applied directly to the lungs. To this end condensed air, an oxygenated atmosphere, carbonic acid, sulphurous and tar fumes, and all kinds of substances in a gaseous form have been inhaled. Solutions in a state of vapor, or divided into spray, have also been tried. Astringent and other fluids have been injected down the larynx and bronchi. Pulmonary cavities have even been opened from without, and variously treated with a view of causing cicatrization. The result of all these efforts has been—what an intelligent consideration of the pathology of the disease might have anticipated—a uniform failure.

STATISTICS.—It is a matter of extreme difficulty to determine with exactitude how the change in the treatment of Phthisis which commenced in 1841, and became pretty general in 1850, has influenced the mortality of Phthisis Pulmonalis. In 1852, Dr. Wood, of Philadelphia, remarks of it, that in that city, during the ten years from 1840 to 1849 inclusive, the average proportion of mortality from Phthisis was 1 in about 6·76 from all causes, or 14·8 per cent., and the same average existed in previous years. Cod-liver oil was then generally used in its treatment, and the mortality sank in this disease during 1850–1 to 1 in 8·33, or about 12 per cent., and in 1851 it was only 11·86 per cent.

In 1862, Dr. C. J. B. Williams, in one of the Lumleian lectures delivered to the London College of Physicians, observes that the experience of Louis and Laennec gave an average duration of two years' life in Phthisis after it was decidedly developed, but that, since cod-liver oil was introduced, he infers from 7000 cases that the average duration of life has been four years.

The registration of deaths in Scotland only commenced in 1855, and offers therefore no means of comparison, as regards Phthisis Pulmonalis, between the mortality occurring before and after that period. But the English registration of deaths commenced in 1837, and, with the exception of a few years, has continued up to the present time. The following is the result :—

Years.	Average annual population.	Average of total number of deaths.	Average of deaths from phthisis.	Percentage of deaths from phthisis to total deaths.
37–41	15,720,385	347,070	55,718	16·0
50–54	18,174,011	359,681	50,515	14·0
55–59	19,257,184	425,703	50,187	11·3
60–64	20,196,787	495,531	51,595	10·4

It would appear from the above table that, taking a five years' average previous to 1841, before cod-liver oil and an analeptic treatment were introduced, the proportion of deaths from Phthisis was 16 per cent.; whereas, in the years 1850 to 1854 inclusive, the deaths were 14; in 1855 to 1859, 11·3; and in 1860 to 1864, only 10·4 per cent. of the deaths from all causes. It must be observed, however, that a certain number of cases annually are vaguely returned as "lung diseases," and that whilst deaths from Phthisis have diminished, those from pneumonia and bronchitis have greatly increased. Doubtless exactitude in diagnosis has very much extended among medical practitioners during the last twenty years, whilst it is a matter of common observation that the winter and spring seasons have increased in severity and duration, circumstances which to a certain extent might account for the numerous returns of pneumonia and bronchitis. Without attaching, therefore, too much importance to the exactitude of the results obtained by the Registrar-General, all that can be said is, that as far as they can be relied on, they exhibit during the last twenty-five years a marked diminution in the mortality of Phthisis Pulmonalis, as compared with the period before cod-liver oil and a restorative treatment were employed.

CANCER OF THE LUNGS.

By Hermann Beigel, M.D., M.R.C.P. Lond.

Literature.—*Hollerius*, Op. omnia, De Morb. intern. 1674; *Heister*, De Asthm. schirr. 1749; *De Haen*, Ratio medend. Parts v., vi. 1765; *Morgagni*, Epist. i. xxii. art. 22; *Ib.* Epist. xx. art. 39, 1780; *Van Swieten*, Comment. ad Aptor. Part ii. p. 797; *Bayle*, Recherches sur la Phthisie pulmon. Paris. 1810; *Langstaff* and *Lawrence*, Med. Chir. Transact. viii. p. 272; *Langstaff*, Med. Chirurg. Transact. ix. p. 297; *Andral*, Clinique Médicale, 1830; *Cailliot*, Sur l'Encéphaloïde, 1833; *Williams*, Diagnosis of Diseases of the Chest, 1835; *Durand-Fardel*, Journal Hebdomad. 1836; *Laennec*, Traité de l'Auscultation, 1839; *Stokes*, Diseases of the Chest, 1837; *Strave*, De Fungo pulmonal. 1839; *Kleffaus*, De Cancr. pulmon. Gröning, 1841; *Marshall-Hughes*, Guy's Hosp. Rep. 1841; *Watson*, Lond. Med. Gazette, 1841; *John Simon*, General Pathology, 1850; *Lebert*, Traité des Maladies cancéreuses, 1861; and his Anat. Pathol. 1855–1862; *Ebermann*, De Cancro pulmon., Petropolis, 1857; *Bright*, Guy's Hosp. Rep. v. p. 377; *Harrison*, Dub. Journ. xvii. p. 326; *Green*, Dub. Journ. xxiv. p. 282; *Tiniswood*, Monthly Journal, July, 1844; *Burrows*, Med. Chir. Trans. xxvii.; *Maclachlan*, Lond. Med. Gaz., 1843; *King*, Ibid.; *Köhler*, Krebskrankheiten, 1857; *Pemberton*, On Melanosis, Midland Quarterly Journ. of Med. Science, May, 1857, p. 129; *Bright*, Diseases of the Heart, Lungs, &c., 1860; *Aviolet*, Du Cancer du Poumon, Paris, 1861; *Begbie*, Archives of Medicine, 1861; *Rokitansky*, Pathol. Anat. 1861, vol. iii.; *Walshe*, Diseases of the Lungs—on Cancer, 1863; *Skrzeczka* in Virchow's Archiv, vol. xi. p. 179; *Virchow's* Geschwülste; *Cockle*, On Intrathoracic Cancer, 1865; *Andrew*, Primary Cancer of the Lungs, Transact. Path. Soc. 1865, p. 51; *Charles Moor*, Report on Cases of Cancer, Brit. Med. Journ. 1866, vol. ii.; *Rindfleisch's* Pathologische Gewebelehre, Dritte Lieferung, 1868.

Cancer of the Lungs is by no means a frequent occurrence. Bayle observed only three cases at the post-mortem examination of 150 individuals who died of phthisis. Begin, at 200 dissections, has only twice observed the disease. Herrich and Popp found malignant growths in 68 out of 1171 corpses; but amongst these 68 there were only six cases of Cancer in the Lungs. Recent observations by Dr. James Russell, Dr. Andrew, and others, have, however, confirmed the opinion held by excellent observers, that the lung may not only be the only affected organ, but in secondary cancer be really a place of predilection.

Walshe considers "Cancer in the lungs to be particularly common as the secondary development, where the testicle has been the primary seat of the disease;" whilst Dr. Day, of Stafford, appears strongly inclined to consider it more frequently a sequence of cancer of bones than of any other primary cancerous development.[1]

[1] Med. Times, 1866, vol. ii. p. 230.

The truth is that cancerous affection of the lungs is comparatively common after primary development, both in the testicles and bones, but that other organs may also—though not with equal frequency—be the nidus for primary deposits, which then may be followed by secondary Cancer in the Lungs. But it must be borne in mind, that the place of primary deposits sometimes is revealed only at the post-mortem examination, which fact leads us to believe that many cases, recorded as primary Cancer in the Lungs, have been in fact secondary affections, and that the organ in which primary deposits have been formed was overlooked.

Concerning the *age* which seems most liable to be attacked, we learn from Ebermann that in 72 cases the following relations are recorded :—

From 1	to 9	years,	1	individual.
" 9	" 19	"	1	"
" 19	" 69	"	66	"
" 69	" 79	"	3	"
" 79	" 89	"	1	"

It appears, then, from this table, that the disease is rare before the age of 20, when it becomes frequent during a long period. It may be mentioned that of 78 cases in which the *sex* had been noted, 51 occurred in men ; so that the ratio, therefore, was eight to three.

Concerning the forms in which Cancer of the Lungs may be observed, *colloid* is extremely rare, *scirrhous* very rare, but *encephaloid* comparatively common. In fact, some first-rate observers—Bayle, Laennec, and others—consider encephaloid the only species of cancer to be found in the lungs. This form, likewise called *medullary carcinoma*, which has received its name from the striking resemblance to brain, being thus the prevalent form of Cancer in the Lungs, to which the whole clinical interest is attached, it seems but right that, in a work like this on practical medicine, our remarks on Cancer of the Lungs should principally be confined to that form.

PATHOLOGICAL ANATOMY.—Encephaloid, as already mentioned, so much resembles the medullary substance of the brain, that, for the unaided eye, it would sometimes be difficult to say whether it be brain or pathological growth. Its consistence is generally soft, pulpy, and depends upon the amount of stroma present, the meshes of which contain the creamy fluid, generally known as *cancer juice*. The vessels traversing the fungus have but thin walls, which sometimes rupture, and, admixing blood and clot with the medullary matter, give rise to the modification of encephaloid, which has been called *Fungus hæmatodes*.

In the early stages of development it is not the extravasation of blood which tinges the growth, but the abundance of very minute vessels traversing the growth, and detectable only by the aid of the microscope. Their walls are very thin and transparent, and easily liable to break. The extravasation extends through the cancerous mass in the same way as it does through the tissue in apoplectic effusions, and the pleural cavity sometimes also contains a clot of pure blood.

If, on the other hand, the cancer-cells contain black coloring matter—probably a modification of the coloring matter of the blood—the growth, of course, assumes a dark appearance, and is then called "Cancer, or Fungus Melanodes." According to Rokitansky, this species is observed only in cases of general cancerous cachexy, or, in other words, as a secondary form ; but Dr. Rogers mentions that it appears also as a primary affection.

Of 60 cases of melanosis collected and published by Pemberton,[1] the post-mortem appearances were recorded in only 35. Of these 35 cases, 17 exhibited deposits in the lungs ; but there is no practical difference between encephaloid, fungus hæmatodes, and cancer melanodes.

Secondary Cancer of the Lungs is rarely limited to these organs, but generally involves the adjoining parts, as costal pleura, pericardium, heart, diaphragm, bronchi, vessels, and nerves ; or the Cancer may on the contrary, take its rise in one of these organs, and during its progress involve the lungs.

The bronchi may become compressed or filled with cancerous matter and their walls corroded. The arteries, but not the veins, enjoy a certain immunity when traversing a cancerous growth. The glands generally participate in the infiltration and transformation of structure ; the mediastinal glands particularly may grow into an enormous and highly vascular, cerebriform mass of several (seven) pounds weight,[2] traversed by the aorta and pulmonary artery, which may become compressed, and even converted into a very thin, soft, yellow elastic band.

I have met with an extremely rare case; the patient was a woman, aged 59 years. She was several times operated on for Cancer in the right breast, but the growth always recurred. Ultimately the lungs become involved and the patient died. At the post-mortem examination, large encephaloid masses were found at the root of the left lung, and both lungs were infiltrated with medullary cancer. But the

[1] Midland Quarterly Journal, May, 1857, p. 145.

[2] A case under the care of Dr. Rees : Lancet, 27th August, 1864. See also Dr. Fr. Braun's "Das Vorkommen des Williamsche Tracheal Tones ;" Erlangen, 1861.

mediastinal and a very great number of bronchial glands had been changed into large dark-colored lumps of Cancer melanodes.

Of the nerves, by their anatomical relations, the vagus and recurrentes are particularly liable to become involved in the process, and to be materially altered. Amongst the cases contained in Dr. Cockle's most elaborate and able work on Intrathoracic Tumors, the one simulating laryngeal phthisis is of particular interest, in which "the cervical portion of the left par vagum was manifestly enlarged."[1] The shape in which the heteroplastic growth under our consideration may be found, varies very much from numberless miliary dots to cancerous tumors of twelve or fourteen pounds weight. In other instances, the lung may preserve its shape, but its normal tissue be entirely destroyed, or rather replaced by cancerous matter. In other instances again, cancerous patches may be observed with intermediate healthy tissue, or, which is the most common, the different forms coexist—miliary deposits in one spot, nodules or nodes and larger growths in another, while a third part may be infiltrated.

As an extremely rare occurrence, which has been observed only a few times, is the form which Rokitansky has called *cancerous pneumonia*, and in which the tissue of the lung may be compressed but otherwise normal, whilst the air cells are filled with detritus, fat globules, and principally with cancer-cells. Such a case has recently been published by Dr. Shrzeczka.[2]

The diseased lung is generally adherent to the inner surface of the sternum and ribs, or it may be compressed or retracted, entirely uncovering the heart, and most closely agglutinated to every part and organ contiguous to it. In cases of compression of one lung, the other generally becomes dilated, in order to compensate for the diminished size of the diseased one.

In some instances, the cancerous formations are limited to the costal or pulmonary pleura; and often assuming a shape which has been compared to "wax-drops,"—Cruveilhier's "Plaques squirrheuses"—do not penetrate into the lung-tissue or air-cells, but remain superficial. In other instances, nodular deposits are formed in the very substance of the lung, growing in a centrifugal direction, and breaking through the pleura.

If cancerous derangements of other organs than the lungs have proved the immediate cause of death, but few—four or five—cancerous spots, of the size of a pea only, may be found in the lungs.[3]

[1] Dr. Cockle, On Intrathoracic Tumors, vol. ii. p. 109.

[2] Virchow's Archiv, vol. xi. p. 179.

[3] Garrod, in the Lancet of 1867, vol. i.

The tumors, of course, undergo the same changes as cancer generally does. The softening begins in the centre, and, advancing towards the periphery, gives rise either to cancerous ulcers or, which is a rarer occurrence, to a cavern filled with puriform, bloody, and putrid juice; the walls of such a cavity are generally thick, infiltrated with its contents, and are likewise in a state of disintegration.

On microscopic examination, the encephaloid is seen to consist of two distinct formations, the one being the *stroma*, forming differently shaped and sized meshes, which consist of fibrous bundles, partly or totally converted into an agglomeration of fatty molecules.

The consistence of the encephaloid depends upon the density of the stroma.

From these meshes, the other formation, viz. the so-called *cancer-juice*, can easily be squeezed, and appears as a creamy semi-liquid fluid. The microscope reveals its color, as depending on an abundant amount of spindle-shaped and other cells, which contain one or more large nuclei and blastema.

The cells are generally in a state of retrograde formation, or fatty degeneration, which causes their contours to appear more distinct. In a still more advanced stage, the cells become completely transformed into an agglomeration of fatty molecules.

Symptoms.—Not unfrequently the patient exhibits but slight symptoms, if any, even when the disease is already far advanced. This is particularly the case with secondary, less frequently in primary Cancer, and depends on the nodular formation of the disease; for these nodules being surrounded by normal lung-tissue, permeable to the air, render auscultation and percussion useless. Dr. Stokes relates a remarkable case, illustrating not only the comparative slightness of symptoms, but also the rapidity of growth. The patient was under the care of Dr. Little, in Sligo Infirmary.[1] A young man was brought in, simply dying from a diseased leg which had been neglected. Dr. Little conceived that the only possible means to save life was amputation above the knee, which he did with the happiest result. Hectic fever disappeared, and in four or five weeks the patient had increased a stone and a half in weight; but he came back shortly, complaining of pulmonary irritation, and died in a fortnight after re-admission, when it was found that both lungs were converted completely into cancerous masses. The rapidity of growth in isolated cancerous masses was very singular. Yet, in the majority of cases, there exist symptoms

[1] Medical Times, Sept. 1, 1866.

enough for the formation of a strict diagnosis. The symptoms generally met with may be arranged in the following manner:—

1. *General appearance of the patient.*—Cases which run through *all stages* without apparent alteration of the patient's general health, are exceptional. Generally, the health is impaired in one or another way; and if there exists anything in disease which may be called "the habit" of that disease, I should be inclined to speak of a "cancerous habit." It may, perhaps, be difficult, nay impossible, to describe appropriately this habit, but a practitioner's eye trained to observe diseases and to notice even slight alterations in the countenance of his patients will surely discover it.

There is something inexpressibly painful and anxious in the lineaments of patients laboring under cancerous affections, which is not met with in any other disease. Nor is the characteristic tint of the patient's skin often absent. Rapidly progressing emaciation is another concomitant of Cancer; and fever of hectic nature, a rapid, small, irregular pulse, which throbs 100 to 130 times in a minute, generally are present to the last moment of the patient's life.

The literature of the disease under consideration furnishes us with numerous cases, the course of which has been precisely similar to that of phthisis; colliquative night-sweats, diarrhœa, exacerbating fever, copious expectoration. In such cases, errors in diagnosis are not only excusable but unavoidable, and such errors have been committed. The appetite is likewise mostly deficient; the natural functions in disorder; and sleep, either by pain, dyspnœa, or other causes, interrupted or entirely disturbed, and languor and debility take possession of the poor patient.

2. *Shape of the thorax.*—The thorax may become altered in two directions, being either increased or diminished in bulk. In both instances the alteration may extend over the whole diseased side, or be partial.

Enlargement of the thorax will be observed, when by heteroplastic growth or effusion into the pleural cavity, pressure is exercised from within upon the chest-walls; whilst diminution of the volume of the thorax will ensue from decrease of the organs situated within the chest, thus allowing the atmospheric pressure and certain muscles to act from without upon the walls of the thorax, in such a manner as to cause loss of its curved shape, and to produce flattening and depression at certain points.

The same effect may be brought about by adhesion of the pulmonary to the costal pleura. The alteration may sometimes occasion a difference between the one side of the thorax and the other, amounting to six or eight inches.

In other instances, the alteration is but slight and discernible rather by inspection than by measurement.

The movement of the thorax during respiration, depending in a very great measure upon the permeability of the lungs to air, will alter under the same conditions as if the lungs had undergone infiltration by other diseases, or had been compressed by fluid or air into the pleural cavity.

3. *Auscultation and Percussion.*—It need scarcely be mentioned that the physical signs will correspond with, and depend on, the state of the organs contained in the chest. We are aware from the principles of physical examination, that separate cancerous nodules, though they may exist in a very great number, do not exercise any influence upon the normal respiratory sound, nor do they materially alter the sound on percussion. The tissues surrounding the cancerous nodules lose their contractility, and would give a tympanitic sound, if their tympanic character were not injured by the solid nature of the newly-formed nodules.

When the nodules become confluent, and the deposits are large, they of course interfere with normal respiration; and, according to their nature and extension, the normal sounds of auscultation and percussion will be altered.

4. *Cough.*—Cough may exist and continue in a slight degree, so as to deceive in respect to the real nature of the disease, both the patient and the physician. But the cough may increase, and become so violent as to resemble hooping-cough, and to torment the sufferer day and night. If the disease be confined to one lung, or if one pleural cavity become filled by effused fluid, cough and shortness of breath set in from very evident causes, as soon as the patient tries to lie on the healthy side. Implication of the one or both vagi in the cancerous process will, of necessity, also be followed by frequent distressing cough of a laryngeal character.[1]

5. *Expectoration.*—It is in some cases entirely absent, but in others very copious, muco-purulent, separating into two or more layers when allowed to stand undisturbed in a glass or any other appropriate vessel. The lowest layers frequently containing so-called cancer-cells, or masses of Cancer, afford conclusive assistance in forming a diagnosis.

When a communication exists between a broken bronchus and cavity, and disintegration is going on, the expectorated matter is sometimes unbearably fetid, and contains elastic fibres and detritus of lung-

[1] Cockle, loc. cit. vol. ii. p. 106.

tissue. In case of corrosion of a vessel, hæmoptysis sets in, and may possibly immediately endanger life. Admixture of small quantities of blood with the sputa is neither a rare occurrence, nor of great importance.

In the above-mentioned case of communication between a bronchus and a cavern, large cancerous masses, with an admixture of blood, may be expectorated, as has been observed by Andral, Bayle, Hartman, Langstaff, or the sputa consist only of blood, and the expectorated masses are of a dark brownish color, as described by Stokes, Burrows, and others.

6. *Pain.*—The lancinating pain, which forms a most distressing symptom of cancer in other parts of the body, is happily a comparatively rare occurrence in Cancer of the Lungs. When present, it is by no means restricted to the diseased organs, but extends to parts distant from the original place of affection. This is easily explicable by the anatomical distribution of the nerves, on which pressure may be exercised, or by the compression, embolism, or thrombosis of large blood-vessels, which may prevent proper circulation in distant parts, and even cause gangrene.

I have observed a very interesting case in a female fifty-two years of age. She had been operated on for Cancer in the left breast. Three years after operation she was suddenly seized with violent pains in the chest, lasting for some hours, disappearing then, and reappearing several days. The pain was so excruciating, that the patient in one of the paroxysms attempted suicide, but was prevented from committing it. When she was free from pain, she had neither cough nor any other sign of chest-disease. Her previous history, together with her present state, confirmed my opinion on the case as being one of intra-thoracic cancer. About a fortnight before her death, which occurred six months after I had first examined her, she began to cough and to waste away with remarkable rapidity; and three days before death the left lower extremity exhibited symptoms which left no doubt that circulation had ceased in it. At the post-mortem examination, both lungs were found studded with small cancerous tumors, the largest of the size of a pea, leaving between them healthy tissue. The root of the right lung was involved in a large cancerous soft mass; the liver likewise contained a considerable number of cancer-nodules, and the left iliac artery was entirely closed by a firm thrombus.

7. *Dyspnœa and Palpitation of the Heart.*—Dyspnœa may exist in a very troublesome degree even when the physical signs are still insignificant; such will particularly be the case when the lungs are filled with miliary deposit. But the same may take place, the lung being but little or not at all affected, when pressure is exercised on those vagus-fibres which are inserted into the lungs. Physiology teaches that such pressure will cause acceleration of the respiratory movements, whilst irritation of those branches of the vagi, which reach the upper part of the larynx, retard these movements. In both instances dyspnœa may be the result, and this again may become the cause of palpitations. These, however, are generally the consequence of the implication of the heart or pericardium in the disease, be it indirectly by pressure, displacement, &c., or by direct participation in the cancerous depositions.

Displacement of the heart by tumors or fluids will, of necessity, alter the action of the heart, which, according to Louis, is smaller in persons dying of Cancer than of any other disease. In such cases it seems to waste in common with the other tissues of the body, and becomes still more contracted from the quantity of the circulating fluid being so much diminished.

It needs no explanation to prove that degeneration of, or infiltration into, the lungs, compression or closure of the larger bronchi, their being filled with cancerous matter, or the effusion of fluid into the pleural cavity, will likewise be followed by dyspnœa, or—particularly at more advanced stages of the disease—by orthopnœa.

8. *Dysphagia* is oftener connected with intrathoracic tumors of considerable size than with Cancer of the Lungs. It is always the result of pressure on the œsophagus, or of swelling of that organ in consequence of pressure. In very rare cases dysphagia may exist as a reflex action, but then it will exhibit a remittent character, whilst it will remain stationary when dependent on pressure; in some cases the symptoms will appear as soon as the patient assumes a certain position, wherein the tumor is allowed to exercise pressure upon the œsophagus. Dr. Cockle's work contains cases illustrating both kinds of dysphagia.[1] This symptom may exist in so high a degree, and the compression of the œsophagus may be so complete, as not even to allow fluids to pass, and it may become necessary to feed the patient by nutrient injections.

9. *The Voice* of a patient suffering from Cancer of the Lung is liable to many alterations. A deep bass may become altered into a high treble, or into hoarseness, according to the different causes, viz. pressure on the recurrent nerves, compression of the trachea or direct affection of the larynx by the disease. In more advanced stages of Cancer of the Lung, as well as of tuberculosis, there is

[1] Cockle, loc. cit. vol. ii. pp. 107, 144.

scarcely a case in which the voice would not be altered in some way. According to Dr. Cockle, extinction of voice may exist without any sign of obstruction in the larynx, and without either stridor or dyspnœa, being dependent solely on paralysis of the laryngeal muscles, consequent on pressure upon the nerves by the cancerous mass within the chest. By means of the laryngoscope, such an affection in our days will be recognized during the patient's life. In a case of complete aphonia, it was observed by Andral at the post-mortem examination, that a cancerous mass had been exercising pressure on the inferior laryngeal nerves.

10. *Contraction of one or both pupils* as a symptom of intrathoracic tumor, and as due to interference with the sympathetic nerve, was first pointed out by Dr. Gairdner. Though this symptom is not pathognomonic, viz. characteristic either of Cancer in the Lungs, or of intrathoracic tumor, yet its presence may, in some instances, form a valuable link in the chain of symptomatic evidence.

11. *Effusion into one or both pleural cavities* is another symptom which is comparatively more often met with in cases of intrathoracic cancer than Cancer of the Lungs. If present, the lung is often adherent to the vertebra, drowned as it were in the fluid, and compressed sometimes to the size of a fist, but may otherwise remain healthy in structure. If the lung-tissue, under these circumstances, is in an infiltrated state, we have a remarkable instance of an organ being infiltrated with a new formation and, at the same time, diminished in size.

The effused fluid has generally a limpid, yellow appearance, and contains albumen. The effusion generally takes place with great rapidity, and when paracentesis has been performed it is replaced in the same manner.

A case published by Dr. Begbie, in the "Archives of Medicine," in 1861, is of great interest in respect to the symptoms under consideration. The patient was a quarryman, 50 years of age, who came to the Edinburgh Royal Infirmary, desirous of obtaining advice for what he thought a slight affection of the chest. The symptoms had become troublesome only ten days before Dr. Begbie saw the patient, who, on being obliged to leave off work, had consulted a medical man in his neighborhood. This gentleman ordered some cough-mixture, and applied a mustard-plaster over the chest; but the symptoms became worse. When Dr. Begbie saw the patient, he diagnosed intrathoracic cancer, and, from the 24th of September to the 16th of October, 550 ounces of fluid were drawn from the enlarged chest. The patient eventually died, and primary mediastinal and pulmonary cancer was found at the post-mortem examination.

It must be borne in mind that cancerous infiltration into the lungs may progress so rapidly as to be mistaken for effusion into the pleural cavity. Mr. Middleton brought such a case under the notice of the Pathological Society of London, at the meeting on the 14th of November, 1850. During life, several medical men concurred in the opinion that the phenomena which the patient exhibited could only be due to effusion into the right pleural cavity. But at the post-mortem examination it was found that very rapid infiltration, and enlargement of the right lung, had taken place. Such cases we must bear in mind, in order to examine thoroughly and very carefully before we decide on performing the operation of paracentesis.

12. *Fever* is generally moderate, of hectic type; the pulse but little accelerated; the aid of the thermometer is, however, of great importance, for though the temperature may be normal, or but little raised, the daily exacerbation will not escape attentive observation. The pulse increases likewise towards evening, and each exacerbation is followed by perspiration, which in many cases is, indeed, very profuse and quite as violent as that which occurs in phthisis, and exhausts the patient in an extreme degree.

Diagnosis. — Primary Cancer of the Lungs, in the majority of cases, admits of no diagnosis. Physical examination tells us whether or not alteration of the lung-tissues has taken place, whether or not the pleural cavity be filled with fluid or solid; but we remain ignorant of the nature of that alteration. In rare cases only, a suspicion will arise; but, unfortunately, the post-mortem examination will finally show whether our opinion has been justified, or based on wrong conclusions. Microscopical examination of the sputa should never be neglected, it being one of the principal means by which the real nature of the disease may sometimes be revealed. "I have seen many instances," says Dr. Williams,[1] "and others are on record, of ulcerous cavities formed in melanose and encephaloid solidifications of the lungs, and the expectoration in one case of a black and red, and in the other of a streaky, whitish, sanguinolent, and puriliginous matter, led to a suspicion of the nature of the disease before death."

The diagnosis of secondary Cancer generally does not afford such insurmountable difficulties as many believe. Its appearance, after primary deposits have been

[1] Pathology and Diagnosis of Diseases of the Chest, p. 154; London, 1835.

made in, and eventually removed from, other organs, will very often serve as a guide for our conclusion. In fact, if after the removal of a malignant growth, pulmonary or bronchial symptoms of any kind appear, it is but wise to suspect them as the beginning of the occurrence of Cancer; at all events let us be on our guard, and not treat these symptoms as if they would occur in persons in whom no signs of cancerous diathesis have ever made their appearance.

It is in these cases in which Hutchinson's much-neglected instrument, the spirometer, will afford good services. Individuals from whom Cancer of any organ has been removed, should, after operation, from time to time be measured in respect to the capacity of their lungs. If the amount of air evidently becomes diminished, gradually or suddenly, then we shall seldom be wrong in assuming that cancerous deposits have been made, and respectively are still progressing.

But, notwithstanding our greatest care and attention, we shall meet—and that not seldom—with cases in which a strict diagnosis will either prove impossible, or be made only after repeated examination and closely watching the case for a longer period. The diseases which are particularly liable to be confounded with Cancer of the Lungs are *chronic pleurisy with effusion into the pleural cavity*, *tubercular infiltration* and *aneurism*.

Differential Diagnosis.—1. *Chronic Pleurisy with effusion into the pleural cavity.*—Though the consistence of encephaloid may be of a semi-fluid nature, yet it will differ in many points from effusion in respect to the symptoms as revealed on physical examination. The area of dulness on percussion, in different positions of the patient, never so strictly follows the laws of gravity as in cases of effusion. Another point of importance is, that in chronic pleurisy the area of dulness sometimes diminishes, which is particularly the case after much perspiration, or after exhibition of diuretics, or similar medicine; but the Cancer, once formed, will under no circumstances decrease.

It is true that, as Dr. Cockle says, "In many cases, mere physical diagnosis is utterly incompetent to decide the question, inasmuch as chronic pleurisy constitutes in itself an integral part of the natural history of intrathorax cancer." But in this instance, viz. when during the cancerous process effusion into the pleural cavity has taken place, we have not any more to decide between Cancer and pleurisy with effusion; it is evident that physical examination has contributed its share towards the formation of the diagnosis, when it has taught us whether the pleural cavity be filled with fluid, solid, or semi-fluid matter; and in respect to this point, with proper care and attention, we shall always arrive at a satisfactory decision. According to Winterich,[1] the vocal fremitus in Cancer is oftener present than absent, whilst in effusion the reverse holds good.

But, if physical examination in some cases is at a loss to answer the questions proposed for diagnostic purposes, then the history of the case, the general appearance of the patient, the rapidity of development of the cancerous growth, the peculiar expression of the patient's face, the peculiar tint of his skin, and perhaps the coexistence of Cancer in other organs, will sufficiently make up for the deficiencies of physical signs, and place us in a position which will enable us to make the diagnosis certain.

2. *Tubercular Infiltration.*—The physician will only be called upon to decide between phthisis and Cancer, when the affection has assumed great proportions. In this case it must be remembered that the latter disease never spreads so extensively as the former does, in which the total absence of rhonchi may also be an important sign. Hæmoptysis is a comparatively rare occurrence in Cancer, but not so in phthisis. The absence of the phthisical habit, the fact that patients suffering from Cancer are not unfrequently in a comparatively good condition, even in advanced stages of the disease, the coexistence of tumors, or the former removal of such, together with—sometimes lancinating—pain in the chest, and the microscopical examination of the sputa, whereby the product of Cancer sometimes may be found, will afford diagnostical hints. Compression of the œsophagus, displacements of neighboring organs in an extremely high degree, the rare occurrence of caverns, symptoms of compression of the aorta or vena cava, the not unfrequent limitation of the disease to one side only, are signs frequently met with in Cancer.

Diagnosis will become still more difficult or entirely impossible, in cases of coexistence of tuberculosis and Cancer. It was due more particularly to Rokitansky that the opinion became general that tuberculosis and Cancer exclude each other, *i. e.* that they never do coexist in the same person. Rokitansky, however, afterwards altered his opinion, saying that the coexistence of both diseases is merely a very rare occurrence. Other authorities hold the same opinion. But many cases have been published, showing that Cancer by no means excludes tuberculosis. I refer the reader to Dr. Pollock's

[1] Winterich's Krankheiten der Respirations-Organe, in Virchow's Pathologie und Therap. Erlangen, 1854.

case,[1] published in the "Transactions of the Pathological Society," and to a highly interesting one, recently published by Professor Friedreich,[2] concerning a woman forty-nine years of age, who suffered from primary Cancer of the left lung, with metastatic depositions in the heart, kidneys, suprarenal capsules, right lung, and pancreas, and from cancerous pleurisy of the left side. At the same time obsolete and recent tubercular enterophthisis and œdema of the brain were found at the post-mortem examination.

3. *Aortic Aneurism.*—In the course of development of cancerous affections, particularly at the root of the lung, great bulging in the clavicular region may take place, accompanied by pulsation and other symptoms resembling aneurism of the aorta. Here the remark of Stokes is of great value, concerning the contrast between the area of dulness on percussion and the pulsation. But the pulsation itself is of a different character in the two diseases, viz. circumscribed in aneurism, but diffused, not culminating in a particular spot, in Cancer, in which affection the ordinary signs of aneurism, as murmur or pulsation over the dull part, murmur above the clavicle, or propagated to the vessels of the neck, are also absent. I am furthermore inclined to believe that, in some cases, the sphygmograph will render great service in arriving at a decision, whether a disease be intrathoracic Cancer or aneurism. Gordon,[3] Marlin Solon,[4] and others, have published very instructive cases, in which Cancer was mistaken for aneurism, and the treatment of Valsalva adopted. But, notwithstanding these authorities, I maintain that a careful examination and consideration of all symptoms, together with the history of the case, will seldom fail to result in a strict diagnosis, and to screen us from erroneous conclusions.

PROGNOSIS AND TREATMENT.—Cancer of the Lungs is a deadly disease, and, in spite of all medical efforts, leads finally to a fatal end.

The first symptoms, as a moderate pain in the chest, difficulty of breathing, a dry cough, &c., sometimes last for years without alarming the patient, till more severe and dangerous phenomena make their appearance, and with tremendous speed hurry the patient into the grave.

In the present state of our science we have neither means for extinguishing an existent cancerous cachexia, nor for causing deposits to be absorbed, which, once produced, seldom remain stationary for any long period, but go on increasing, destroying the affected tissues, and interfering with neighboring organs.

In the good olden times, when physicians fancied that even a disease like Cancer would fly before a long prescription, many formulas were in vogue in which arsenic was the principal drug. This remedy was considered a specific, and eminent practitioners speak of it in terms of high commendation.

Others again advocated the use of conium, bichloride of mercury, the preparations of iron, and a number of other medicaments. But it appears that the efficacy of all these "specifics" became weaker and weaker in the same proportion as diagnostic science became strict and exact, and that arsenic and the other drugs effected a cure in those cases only in which a closer examination demonstrated that the case for which it had been applied was not Cancer at all.

But, though medical science has not yet arrived at a point to furnish us with means of *curing* Cancer of the Lungs, we must not rest quiet and leave such patients to their fate. Our profession has other tasks to fulfil where cure is impossible, namely, to relieve pain and alleviate other bad or dangerous symptoms, and thus to prolong life. In this respect we can act sometimes with very great benefit towards the sufferer.

I had a patient under treatment who dreaded the approach of night, this being for him the signal of excruciating pain, restlessness, and torture, during which he incessantly offered prayers to Heaven for his death. Besides deposits on his lungs, there were likewise some in his liver, and the stomach was also affected, and rejected food and medicines as soon as they were taken. When he came under my care, I injected, every night, half a grain of morphia hypodermically, and from that time he enjoyed at least good rest at night.

Our attention will therefore entirely be directed towards troublesome symptoms, improvement of the patient's nutrition, and keeping up his strength. Hence it becomes evident that bleeding in any shape and to any extent should only be resorted to in cases of pressing emergency. Dry-cupping, however, will prove beneficial when dyspnœa becomes troublesome, in which cases other counter-irritants may also be applied to the skin with success. In one case, under my care, a hot bath of a minute's duration gave rest to the much-exhausted patient, while other remedies failed to diminish the dyspnœa.

For the relief of pain, connected with Cancer of the Lungs, I can strongly recommend the hypodermic injection of morphia, beginning with a quarter of a grain,

[1] Transactions of the Pathological Society, vol. iii. (1851–52) p. 254.
[2] Virchow's Archiv, xxxvi. 4, 1866.
[3] Med.-Chirurg. Transact. vol. xiii.
[4] Archiv. Gén. de Méd. tome xxiv. p. 142.

and increasing the dose according to the requirements of the case. In respect to the method of injection and mixing the solution, I refer the reader to my paper "On Hypodermic Injections," which has been published in the "Medical Mirror" of 1866.

Cough is another symptom which often resists all therapeutic endeavors. Where medicines can be taken, we should apply narcotics, opium, hyoscyamus, Indian hemp, and similar drugs. But, unfortunately in many cases the stomach, either by reflex action or by being also affected by the disease, rejects the drugs, and renders our efforts useless. In these cases I propose the application of atomized fluids, which, indeed, would be the only means by which to introduce medicaments into the system. I refer the readers who are not acquainted with this mode of treatment to my work "On Inhalation."[1]

The patient's strength will appropriately be kept up by nutrient, easily digestible food, and avoiding everything which could possibly produce a conflux of blood towards the internal organs. In those unfortunate cases in which pressure on the œsophagus prevents the patient from taking solid food, it must, of course, be given as a fluid, and, if necessary, by the aid of the stomach-pump.

In the patient's room, a moderate but equal temperature ought to be kept by day as well as by night; all the natural functions must be regulated as far as possible, and moderate exercise in the open air should be encouraged on fine days, and avoided only when it causes difficulty of breathing.

Some physicians advocate cod-liver oil. It may be tried in cases in which it does not at all interfere with the function of the stomach, but it ought to be given up at once if it causes loss of appetite or sickness.

Fetid breath, sometimes of unbearable intensity, disgusts not only everybody in the patient's room, but even the patient himself. This disagreeable quality of the breath can be destroyed in a short time, by inhalation of liquor chlori, perchloride of iron, or creosote.

Should one be called upon to give some prophylactic hints to persons descended from parents who died of Cancer, the first care to be taken will be strictly to regulate the diet of such persons. Let them take regular exercise and live in mild climates, in places situated as high as possible; advise them to undertake voyages, or to undergo a course of the so-called "grape-cure" of which many physicians speak in commending terms, and which produced very good effects in a case under my own care.

The coast of England is a very healthy abode during the summer months for delicate individuals. But for such persons as wish to go abroad, Marseilles, Spezzia, Nice, Livorno, Venice, Heligoland, Kiel, Swinemünde, and the very pleasant isle of Rugia, could be recommended.

Places where grapes are methodically used for medical purposes are Meran in Tyrol, Dürkheim and Bingen in Germany, Krems in Austria, and Presburg in Hungary. The best time at which to send patients there is during the vintage, which is generally in the months of September and October.

PNEUMONIA.

By Wilson Fox, M.D., F.R.C.P.

Synonyms. — Peripneumonia,[2] Peripneumonia Vera (as opposed to Peripneumonia Notha, or Capillary Bronchitis); Febris Pneumonica, Hoffmann; Fièvre Pneumonique, Fluxion du Poitrine (French authors); Pneumonites, auct. var.

Varieties and other Synonyms.— Croupous and Catarrhal Pneumonia (*Rokitansky and modern German authors*). Acute Sthenic Pneumonia—Bronchopneumonia (*English and foreign authors, signifying a similar distinction of origin and course*). Lobar Pneumonia—Lobular or Disseminated Pneumonia (*signifying anatomical differences in the extent and characters of the pulmonary affection*). Acute Pneumonia—Chronic or Interstitial Pneumonia (*signifying differences in course and duration, and also in anatomical characters*). Interlobular Pneumonia (*an affec-*

[1] On Inhalation as a means of Local Treatment of the Organs of Respiration by means of Atomized Fluids and Gases, by H. Beigel, M.D. London: Hardwicke, 1866.

[2] Grisolle considers that the prefix περί is merely expletive.

tion of the interlobular tissue). Primary Pneumonia—Secondary Pneumonia (*signifying differences in origin*). Other varieties have been termed, according to the origin or characters of the disease—Bilious, Gastric, Typhoid, Latent, Intermittent, Hypostatic, Tubercular, Scrofulous, Rheumatic, Gouty, Puerperal, Metastatic, and Pneumonia Potatorum (Huss).

ACUTE PNEUMONIA.

Definition.—A disease whose essential anatomical feature consists in the inflammation of the vesicular structure of the lungs, which is thereby rendered impervious to air through the accumulation in the interior of the alveoli of the products of such inflammation. Clinically it is characterized by pyrexia, which, in the majority of cases, when the disease is primary, commences with rigors; it is also commonly attended by pain in the side, by dyspnœa, cough, sanguinolent sputa, great physical prostration, and by the physical signs of pulmonary consolidation. Its course, when primary, is usually acute, and tends to terminate favorably by a crisis occurring from the third to the tenth day, but it may prove fatal from the first to the fourteenth day, or at later periods. When secondary to other diseases, the termination by crisis is uncommon, and its duration is also more protracted; and under all circumstances of its origin it may, in some instances, lapse into the chronic state. Its immediate cause is uncertain, and it appears in the majority of instances to depend either on an unknown but suddenly produced dyscrasia, or on an alteration in the composition of the blood induced by various diseases. In other cases it is produced through the extension to the pulmonary tissue of bronchial inflammation, or it may originate through local disturbances of the pulmonary circulation occasioned by congestion or collapse, or by obstruction through emboli of the pulmonary artery, or it may be caused by mechanical injury to the tissue of the lung.

Although the anatomical characteristics of Pneumonia can be defined with a certain approach to accuracy, the clinical features of the disease may nevertheless present a considerable diversity of aspect under the varied circumstances of its origin.

In some cases variations in the anatomical process may be observed corresponding with these different features of the disorder, but distinct lines of demarcation are in this respect very frequently wanting, and the author believes that the anatomical distinction between the "croupous"[1] and the "catarrhal" forms, on which especial stress has of late been laid, is by no means so sharply defined as some recent writers have maintained.

From a clinical point of view, however, the separation of the main types of these two forms of the disease into distinct species has a practical value, and it may therefore be stated that the principal classes to be distinguished are (1) Primary or Acute Sthenic Pneumonia; (2) Secondary Pneumonia, including most of the catarrhal forms; (3) Interlobular Pneumonia; (4) Chronic Pneumonia. Under the head of Etiology, the relations of the different forms of the acute disease will be treated collectively.

History.—In the earlier days of medicine, since the times of Hippocrates and Galen (by whom, however, both diseases were recognized), Pneumonia was confounded with Pleurisy to such an extent that the rusty sputa characterizing the former disease were described as an attribute of the latter; and pleurisy was said to be capable of producing cavities in the lung. Valsalva, Morgagni, Huxham, and Boerhaave gave accurate descriptions of Pneumonia, but still the distinction between it and pleurisy was not completely recognized until the writings of Bichat and Pinel, and the collapse of the lung attending pleuritic effusion was by most other writers mistaken for inflammation of its substance.[2] The accurate

[1] The term "Croupous," introduced by Rokitansky, and largely used in Germany, appears to the author to be in some respects best avoided. It was originally employed by Rokitansky to define a particular form of exudation, and in its application to Pneumonia he drew a parallel between this disease and croup of the larynx, attended by false membrane. The analogy appears to be an erroneous one in two aspects, for in the first place the Pneumonia attending laryngeal diseases when false membranes are present is seldom seen in the form recognized as characterizing acute sthenic Pneumonia, but is most commonly of the type termed Broncho-pneumonia; and, secondly, there is no boundary line of distinction between the forms of the disease characterized by a coagulable exudation in the vesicles, and those where cell-products are mingled with some fluid exudation. The extreme types are, it is true, distinct, but every shade of gradation may be observed between them.

[2] According to Pinel, "Nos. Philos." ii. 145–191 *et seq.*, the question of the distinction between these two diseases appears to have given rise to the most animated discussion among the writers of the 17th century. The history of the earlier views on Pneumonia will be found at length in Grisolle's work on Pneumonia; also in Wunderlich's "Path. Therap.," art. Pneumonie, and in Neumann, "Krankheiten des Menschen," 2e Ed. i. 151

clinical separation of the two diseases was finally fully evolved by Laennec. Since his time, the most important advance in the definition of the disease has been that made by Jörg, Bailly, and Legendre in the separation and distinction of the various forms of collapse, or defective expansion from true inflammatory action. The other features of interest in recent researches will be alluded to in their appropriate places.

Etiology.—On many points in the etiology of Pneumonia the only data at our disposal refer to the disease as a whole, irrespective of any of the special varieties before alluded to. The circumstances predisposing to particular forms will be, as far as these are known, described separately.

A. *Race and Climate.*[1]—Inflammation of the lungs appears, with but few exceptions, to be more commonly associated with climates presenting marked and rapid variations of temperature than with extreme degrees of either cold or heat. Thus in tropical climates it is uncommon during the hot season, and, on the other hand, in some of the expeditions to the North Pole the disease has been almost unknown. It is said also to be very rare in Iceland. Throughout the European continent, below 60° north latitude, it is a very prevalent disease, and the southern portions, including the shores of the Mediterranean,[2] are nearly as liable as the more northern countries. Thus in Copenhagen the mortality from Pneumonia is 6·3 per 100 of all deaths; and in Gibraltar 41 per 1000 soldiers suffer from the disease. In the more tropical climates, elevation above the sea-level increases the frequency of the disease, and it is very common in the high table-lands of Mexico.[3] The disease appears to be rare in Egypt, though bronchitis is common in the valley of the Nile; in India it is more common in Bengal than in Bombay. Though equability of temperature appears to confer a certain degree of immunity from the disease, yet there are some remarkable exceptions; for in Senegal, which possesses a variable climate, Pneumonia is rare, while in the Bermudas, where the temperature is remarkably uniform, it is by no means uncommon; and it is stated, on the authority of Dr. Farry,[1] that Pneumonia and affections of the lungs in general are less common, both in the Northern and Southern States of the Union, than in the central portions where the temperature is more uniform. Oregon and California appear to enjoy a singular immunity from the disease. In certain countries, as in Sierra Leone, the Cape, and the Mauritius, the negro races, at least when employed in military service, appear to suffer more than the whites; but it is considered possible that the preponderance of the affection among them is due to their being more exposed to vicissitudes of temperature than the European soldiers, with whom greater precautions are taken.

The disease is said to be more common among sailors on land than when at sea;[2] but it may be questioned whether this difference is not in part due to other influences, causing an increased relative frequency on land, such as greater irregularity of life and severer exertion.

In England, Pneumonia appears, from the returns of the Registrar-General for 1863–4, to rank next after the following main causes of mortality:—Phthisis, bronchitis, scarlatina, old age, and convulsions. The frequency, and also the mortality of the disease, however, vary considerably in different years, as is shown by the contrast of 26,052 deaths registered under this head in 1855 when compared with 21,118 occurring in 1867;[3] and the data of nearly all the large hospitals of the Continent furnish confirmatory evidence of the same kind.[4]

(quoted by Wunderlich). The confusion between Pneumonia and pleurisy was aided by the fact, that before the writings of Bichat the term pleura was limited to the parietal membrane, the visceral portion being confounded with the tissue of the lung.

[1] For a large number of the data under this head, the author is indebted to the writings of Grisolle, "Traité de la Pneumonie," and Hirsch, "Handb. der Hist. Geograph. Pathol.," 1864; and also to an elaborate statistical work on the Geographical Distribution of Pneumonia, by Ziemssen, "Monatsblatt für med. Statistik und offene Gesundheits-pflege," 1857, analyzed at considerable length in Canstatt's "Jahresb.," 1857, ii. 119. Many of the data on this subject refer, however, to pleurisy and Pneumonia collectively, and this is especially the case with those given in Hirsch's work.

[2] Clark on Climate, p. 121.

[3] Elevation in cold climates, in some situations, also appears remarkably to predispose to the disease. Thus of the French troops quartered on Mont Cénis from December to May, one-fourth of the whole number were attacked by pneumonia. (Chomel, Leç. Clin. Méd., Ed. Sestier, p. 451.)

[1] American Journ. Med. Science, 1841. (Grisolle.)

[2] Dr. Wilson's report to the Admiralty gives for 1000 sailors: Short voyages, 29 per 1000; home service, 35·1 per 1000; Mediterranean, 31·8. Sailors as a class suffer but little—175 per 24,000. (Le Roy de Méricourt.) These data are quoted from Grisolle.

[3] In the last-named year these proportional numbers are 995 deaths from Pneumonia, to 1,000,000 living; and 45,275 to 1,000,000 of deaths.

[4] This is especially evident from the statistics of Huss, "Behandlung der Lungen Entzündung:" for while the average number of

It would appear from Ziemssen's analysis that the mortality from Pneumonia is greater in large towns than in country districts; but in this respect there are considerable differences in degree between different cities, that of Cork being 0·5; London, 1·7; Paris, 2·3; Turin, 3·8; and Algiers, 4·3 per 1000. Ireland seems to suffer to a less degree than most of the European countries.

B. *Classes and Professions.*—There appears to be a general consent that Pneumonia is more common among the laboring than in the wealthier classes of society, and that, among the former, those whose occupation involves the severest exertion and the greatest amount of exposure are the most liable to suffer. In the English army the soldiers suffer more than the officers.[1] The disease is more common in the French army than among the civil population.[2]

C. *Seasons.*—It may be stated as a general truth, that in European countries Pneumonia is most common during periods of the year in which there are the greatest vicissitudes of temperature, while either a continuously low or high temperature has much less influence in its production. Thus, of 2616 cases collected by Huss[3] during a period of sixteen years in Stockholm, the spring months, March, April, May, and June, gave 49 per cent.; the winter months, November, December, January, and February, yielded 30 per cent.; and the summer months, July, August, September, and October, 21 per cent. Of the individual months, August and September are those in which the greatest immunity is observed; but this is nearly equalled by June and July, while April and May show the greatest frequency. Huss states that the relative frequency in individual months in different years corresponds closely to rapid changes of temperature observed in them. Barometric variations, independently of the influence of wind, appear to have little or no effect in the production of the disease. The converse, however, appears to hold true of cold winds, and particularly of those from the north and east; and though the effects of these in the production of Pneumonia have been more observed in the aged, and also, though to a less degree, in the young, than in persons of middle life, yet there is a strong probability that their agency is similarly exerted at all ages.[1] It was stated by Huxham[2] that dry cold air was most frequently associated with Pneumonia of an inflammatory type, and that "bastard peripneumonies" were most common in damp seasons. Dr. Jackson[3] has also shown that in Massachusetts, a damp climate, complications are more common than in drier atmospheres.

D. *Age* must be regarded as an important etiological element in the predisposition to Pneumonia, and it is also one of the conditions most materially influencing its mortality.

Some of the details given by writers antecedent to the researches of Legendre and Bailly are, however, unreliable, owing to the confusion then existing between Pneumonia and collapse of the lungs occurring in infancy. Thus Valleix and Vernois[4] stated that of 114 newly-born children 113 had hepatization of the lungs. In spite of these doubts, however, there is very little question that Pneumonia is a very frequent disease of early life. Of 186 cases of primary acute (croupous)

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cases during 16 years was 163·5, these in 1849, 1851, and 1853 amounted respectively to 243, 242, and 203 admitted to hospital; while in 1840, 1841, and 1844, the numbers were only 107, 102, and 97. It will be seen in the section devoted to the prognosis that the mortality of the disease in different years also presents considerable variations; and also that the relative mortality at different seasons by no means corresponds to the frequency of the disease at these periods.

[1] On the Mediterranean stations the soldiers suffer from Pneumonia in the proportion of 32 to 42 per 1000; the officers in the proportion of 14·1 per 1000. On the Canadian stations the proportion of soldiers affected is 43 per 1000, and that of the officers is 10·6 per 1000. (Quoted from Grisolle.)

[2] Deaths from Pneumonia in the civil population of France, 30 per 1000; in the army, 39 per 1000. (Lancereau, Ann. d'Hygiene, 1860, xiii. 269. Valleix.)

[3] The amount of statistical evidence on this head is large and conclusive, and the results obtained by all observers agree very closely with those of Huss. For other references see Chomel, "Lec. Clin. Méd. 'Pneumonie,'" p. 444; Grisolle, loc. cit., 139; Wunderlich, "Allg. Path. Therap.," Bd. iii., Abth. ii. B., p. 304; Bamberger, "Wien. Med. Woch.," 1857; Roth, "Würzb. Med. Zeitsch.," 1860; Hamernigk, "Die Cholera Epidem.," Prag, 1850. Ziemssen, "Die Pleuritis und Pneumonie im Kinderalter," p. 187, found in Grieswald a rather larger proportion during the summer months than has been noticed by other observers. He attributes this to the cold winds and rapid variations of temperature observed there during this season. Morehead, "Dis. of India," pp. 300–303, found Pneumonia in India to be most common in the cold season, and next in frequency in the wet season. During the latter period it is very liable to be complicated by intermittents.

[1] See for evidence on this subject Grisolle, p. 142.

[2] Essay on Fevers, 1757, p. 222.

[3] Dr. Sibson, Brit. and For. Rev. 1858, xxii. p. 23.

[4] Valleix, Clin. des Malad. des Enfans nouveaux-nés, 1838, p. 114.

Pneumonia in children, recorded by Ziemssen,[1] 117 occurred in the first six years of life, and only 69 in the succeeding ten years. Gunsburg,[2] for 5000 cases of Pneumonia, gives the following relative table of frequency at different ages :—

Years.		Years.		
Under	$1\frac{1}{2}$		11	per cent.
From	$1\frac{1}{2}$	to 14	13	"
"	14	" 20	6	"
"	20	" 30	17	"
"	30	" 40	16	"
"	40	" 50	10	"
"	50	" 60	9	"
"	60	" 70	7	"
"	70	" 80	11	"

Lombard has given, further, the following proportion of deaths from Pneumonia and deaths from other diseases at different ages :—

Deaths from all causes.	Age. Years.	Years.	Pneumonia.
274	under $1\frac{1}{2}$		$56=\frac{1}{5}$.
310	from $1\frac{1}{2}$	to 14	$70=\frac{1}{4}$.
112	" 15	" 19	$3=\frac{1}{37}$.
387	" 19	" 27	$39=\frac{1}{10}$.
766	" 27	" 75	$46=\frac{1}{16}$.

Grisolle's statement may therefore be regarded as embodying the truth on this question, viz. that Pneumonia (both primary and secondary, lobar and lobular collectively) is a disease very frequent in infancy, that it is less common from infancy to twenty years of age, that it is comparatively frequent from twenty to forty, less so from forty to sixty, and very frequent, and also very fatal, after sixty years of age. To this it may further be added, that the Pneumonia of old people and of children approximates more, but by no means exclusively, to the type of catarrhal, or broncho-pneumonia.

E. *Sex.*—In the Pneumonia of adult life, males are more commonly affected than females in proportions varying from two or three to one.[3] This difference between the sexes is not observable in the earlier periods of life ;[1] but it becomes apparent first at ages when the occupations of the sexes differ, and when males are more exposed to climatic influences than females. When, however, the conditions of life for both sexes are identical, this relative disproportion in great measure disappears.[2] Huss has adduced the fact that it is also much less marked in advanced age.[3]

Females, as it would appear from Grisolle's data, are somewhat more predisposed to the occurrence of the disease at the menstrual period. Neither pregnancy nor the puerperal condition seems, however, to create any special proclivity, except when the latter is complicated by septicæmia.

F. *Constitution.*—Opinions differ whether Primary Pneumonia most commonly attacks the vigorous or those in previously bad health. The Hippocratic doctrine was in favor of the former view, which is also supported by Grisolle. Huss, on the contrary, thinks that it is more common in weakly subjects. Dr. Hughes Bennett,[4] in 118 cases—84 males and 34 females—found that of the males 27, and of the females 22, were in bad health at the time of the seizure. Huss considers that the fact that robust males are frequently attacked depends in great measure on the greater degree of exposure to external influences to which they are subjected. Chlorotic females seldom suffer. Rickets, on the other hand, appears to produce a predisposition to the disease, for of twenty-four patients dying rickety, Grisolle found Pneumonia in one-half. It is possible that this may be caused by the greater severity of bronchitis and the increased tendency to collapse in these subjects, and also to the fact that collapse of the lung when complicating bronchitis induces a liability to further inflammatory changes.

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[1] Loc. cit. p. 155.

[2] Klinik der Kreislaufs und Athmungs-Organe (Breslau, 1856), quoted from Huss, loc. cit.

[3] The proportion of 2 males to 1 female is that given by Grisolle and generally accepted. Of the actual numbers treated by Huss, the proportion was 5 to 1, but it amounted to 3 to 1 when calculated on the total numbers of all cases of males and females admitted to hospital. The proportion in the general hospital at Vienna (quoted by Huss) is 1·98 males to 1 female. Huss thinks that the greater disproportion observed in the more northern climate between males and females may be due in part to the greater intensity of climatic conditions to which the former are there exposed.

[1] Ziemssen, in 91 cases of children under four years of age, found that the boys affected numbered 41, and the girls 35.

[2] Thus Tolmouche has observed that in prisons the number of individuals of the two sexes suffering from Pneumonia are, comparatively speaking, equal (Ann. d'Hygiène, xiv. pp. 252–7.) Ruef also (Heidelb. Med. Annalen, ii. 1836) has noticed a similar equality in the liability of the sexes to the disease when women are employed in outdoor labor.

[3] Of the cases between the ages of 16 and 50, the males formed 85·5 per cent., and the females 14·5 per cent.; but of the cases between 50 and 70, the males constituted only 55·19, and the females 44·81 per cent. Dinstl also (Oest. Zeitsch. für prakt. Heilkunde, viii. 1862) found in 1212 cases of Pneumonia, that after ætat. 50 the number of females affected was greater than that of the males.

[4] The Restorative Treatment of Pneumonia, 1866, p. 24.

It has been observed that some persons are liable to repeated attacks of the disease—a peculiarity which may either be due to some special but unknown constitutional predisposition, or to the fact that previous attacks induce a proclivity to its return. The latter hypothesis is to some degree favored by the fact that the lung first affected is the most liable to suffer in a subsequent attack.[1] In 175 cases analyzed by Grisolle, 54 had suffered from previous attacks, but of these only two were in females. The period between the attacks varied from one month to twenty-five years. Most usually the intervals varied from three to five years; but these tend to become shorter in proportion as the attacks become more frequent.[2]

The number of attacks from which individuals have suffered is also very remarkable. Thus Andral[3] records a case of a patient who had had fifteen attacks in eleven years, Chomel[4] has seen ten recurrences, J. P. Frank[5] eleven, and Rust has even recorded twenty-eight attacks in the same individual.[6] Intermittent fever also predisposes to recurrence. A patient of Ziemssen's thus affected had four attacks in five years, three of which were in the left lower lobe and one in the right upper lobe.[7]

Difficult dentition predisposes to Pneumonia in children,[8] and also makes the prognosis more unfavorable. Favorable hygienic influences confer a certain degree of comparative immunity from the disease. Drunkenness appears to act powerfully as a predisposing cause of Pneumonia, though its effect in immediately producing the disease may be regarded as somewhat doubtful.

G. *Direct Exciting Causes.*—The influence of these in the production of the acute primary disease has been very variously estimated by different observers. Some authorities, and particularly writers of the last century,[1] attribute its origin mainly to the influence of a chill—an antecedent which others have denied from statistical data. Grisolle asserts that a discoverable cause of this nature could only be affirmed in one-fourth of his cases. Chomel[2] and Andral[3] express very similar opinions. Ziemssen says that among children a discoverable cause only existed in one-tenth of his cases. In fifty-three cases analyzed by myself, a distinct cause, which when present was always of the nature of a chill, could only be affirmed in sixteen. It must, however, be admitted that this is the most common of the discoverable causes, and that the frequent absence of evidence of such an origin is common not only to Pneumonia, but also to many catarrhal affections and further to acute rheumatism, diseases which, to say the least, are very frequently due to this immediate agency. The most probable explanation of such cases would appear to lie in the existence of a more extreme constitutional susceptibility, in consequence of which causes so slight as to pass unnoticed at the time of exposure may produce effects which persons less predisposed to suffer from their influence would have escaped. I do not think, as far as my own observation has gone, that the cases excited by a chill can be separated from the rest and placed in the category of Broncho-pneumonia, for in most of the instances coming under my own cognizance these cases have run as typical a course of acute primary Pneumonia as those in which no such cause has been discoverable. The indirect evidence afforded by the seasons of the year at which Pneumonia is most prevalent, strongly bears out the opinion that vicissitudes of temperature are among the most important agencies in its production. They appear to act most strongly

[1] In 35 cases of recurrence collected by Grisolle, the return of the disease was noted 25 times in the lung first affected. In the remaining 10 the disease changed sides: Pneumonia of the left lung recurred more frequently than that of the right, in the proportion of 16 to 9. This is the more remarkable when it is remembered how much more frequently the right lung suffers from the primary disease.

[2] Dr. West of 78 cases in children, found that 31 had suffered from previous attacks. Of these, 21 had been affected once, 4 twice, and 2 four times, and 4 others were said to have had several attacks. 10 of these patients were under 2 years of age; 10 more between 2 and 3, and the remaining 11 were between 3 and 6. Ziemssen, in 201 cases of children, found 19 cases in which the attacks were repeated. Of these, 14 had Pneumonia twice, 3 three times, and 2 four times. In some instances the disease recurred at corresponding periods of consecutive years.

[3] Clin. Méd. iii. 371.

[4] Dict. de Méd. xviii. art. "Pneumonie."

[5] Interpretationes Clinicæ, Tubingæ, 1812, p. 96. (Grisolle.)

[6] Quoted by Dr. Williams, art. "Pneumonia," Cyc. Pract. Med. iii. 406.

[7] Ziemssen, loc. cit. 154.

[8] Of 201 cases of Pneumonia observed by Ziemssen, this condition was present in 37. Of these, 16 had Broncho-pneumonia after long-continued bronchitis, and 21 suffered from primary or "croupous" Pneumonia.

[1] Pinel (Nos. Phil. ii. 163) defines as the causes of primary Pneumonia: "Impression brusque d'un air froid après un violent exercice, comme la course, la lutte, le chant, les cris, une équitation rapide contre la direction du vent, une boisson froide lorsqu'on est échauffé."

[2] Leçons, p. 464.

[3] Clin. Méd. vol. iii.

at the two extremes of life. Cruveilhier[1] particularly noticed the injurious effects of cold on the aged in the Salpêtrière; and Hourmann and Dechambre,[2] out of 156 cases of Pneumonia in old people, observed 140 in the winter and early spring months, from November to May. Both these writers, and also Cruveilhier, remark upon the injurious effects of north and northeast winds in producing inflammation of the lungs in the aged.[3]

Laennec thought that prolonged exposure to cold had more effect than a sudden chill, but I cannot say that my own experience has led me to adopt this view. Nearly all the cases of Pneumonia which I have observed from traceable causes were owing to a temporary chill, such as a wetting, exposure to draughts of cold air when heated, and similar influences. There can be very little doubt but that Pneumonia, in many instances at least, must depend in great measure on predisposing constitutional or local conditions, whose nature is unknown, but whose influence is distinct. It is to their influence that the special localization of acute diseases arising from general in contradistinction to specific causes, is due; and it is also to the greater or less degree in which they predominate, that the relative facility of the production of such diseases may in great measure be attributed.

Excessive exertion appears to act as an occasional cause. Wunderlich quotes a statement of Barth's to the effect that he had traced this cause in 12 out of 125 cases, and Wunderlich says that he can confirm Barth's experience.

Traumatic causes do not easily produce a pneumonia of any extent or severity: the lung appears to have remarkable powers of recovery from direct injury.[4] Injuries and blows to the chest are however occasionally followed by Pneumonia without distinct evidence of direct laceration of the lung.[5] The mechanism of such influences appears in some cases very obscure. Thus in a case admitted into University College Hospital, under Sir W. Jenner, a patient struck his shoulder-blade on rising from a stooping position. He had previously been in apparently good health, though on admission he was found to be suffering from albuminuria, in addition to signs of pleuro-pneumonia on the side strück. Pericarditis also supervened, and the case proved fatal. The pneumonia was in the lower portion of the upper lobe, and there was also extensive pleuritic effusion on the same side, but there was no evidence of mechanical injury to the chest-wall or to the lung. The kidneys were fatty. It is probable that in this case the pre-existing kidney disease acted as a powerful predisposing cause to the pathological conditions found.

Pneumonia may, on the other hand, be easily excited by foreign bodies entering the lungs from the bronchi. This condition is said to be not uncommon in those cases of dementia when food finds its way into the bronchi, and where gangrene of the lung is very liable to supervene. Grains of wheat or beards of barley entering the bronchi are also occasional causes of Pneumonia.[1] Blood gravitating into the vesicular structure of the lungs in cases of pulmonary hemorrhage may occasionally act as an exciting cause,[2] and it is thought probable that the disseminated Pneumonia observed in diphtheria and capillary bronchitis may be, in part at least, occasioned by the gravitation or insufflation into the air-vesicles of the fluid secretions of the bronchial tubes.

It is very doubtful whether irritating vapors can produce true lobar Pneumonia. They may, however, produce a disseminated form of the disease, resembling closely the "lobular pneumonia" occasionally occuring in bronchitis.[3]

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[1] Anat. Path., liv. xxix.

[2] Pneumonie des Vieillards, Arch. Gén. 2e Sér., xii. p. 29.

[3] The mode of action of these causes will be further considered under the head of Pathology.

[4] Grisolle, pp. 43–4.

[5] See a case quoted by Grisolle, loc. cit. 316, from J. P. Frank, of a porter who had overstrained himself; also Duchek, "Abtheilungs-bericht Allgem. Krankenhauz zu Prag;" Prager Vierteljahresch. 1853, xxvii. p. 37—two cases where Pneumonia followed a blow on the chest; also Wunderlich, loc. cit. Bd. iii. Abth. ii. 13; also noted by Morgagni, "Epist." ii.; also a case by Andral, "Clin. Méd." iii. obs. vii. p. 293.

[1] Grisolle, p. 146.

[2] See Dr. Hermann Weber's, Dr. C. Bäumler's, and Dr. Sanderson's papers in the Trans. Clin. Soc. iii. This subject is a very wide one, and involves the disputed question whether hæmoptysis, unassociated in the first instance with tubercles, can originate a disease running the course of phthisis. I have more than once seen Pneumonia follow hæmoptysis in the course of early phthisis, but I have hitherto regarded it as probable that the hæmoptysis may be the result of the congestion which precedes Pneumonia acting on the weakened pulmonary vessels. In some cases of phthisical subjects, this appears to be the undoubted mechanism of the hæmoptysis observed; but in other cases there is a strong probability that the Pneumonia results from the hæmoptysis in the manner described.

[3] See Bretonneau, Rech. Infl. Spec. Tiss. Muqueux, Paris, 1826, p. 100. Gendrin (Hist. Anat. des Inflam. ii. 302) says that if an animal be made to breathe chlorine, the lungs are found studded with little solid nodules arising from an exudation into the air-vesicles. Gendrin considered these to be

H. *Epidemic Causes.*—The only positive data on this subject are those afforded during the prevalence of epidemics of influenza. This disease has certainly a considerable tendency to give rise to Pneumonia, which is for the most part of a catarrhal type. Thus Nonat[1] observed, during the epidemic of influenza in 1837, that of 300 deaths in the hospital Hôtel-Dieu, in Paris, in the month of February of that year, 80 were due to Pneumonia; and Laserre,[2] in La Pitié, observed in three months in 1842, during a similar epidemic, 31 cases of Pneumonia.

It would appear, however, that unhealthy conditions of crowding, with bad ventilation, strongly predispose to the disease when other causes, particularly measles, are present; and some evidence has lately been adduced to show that similar influences may operate independently of the presence of any immediate cause.[3]

Griesinger[1] has stated that in malarial districts Pneumonia has at times a tendency to assume an epidemic character. It may be doubted whether, independently of such causes, Pneumonia can be considered as an epidemic due to a specific poison, or whether its greater prevalence at certain seasons, and in particular years, producing an apparent resemblance to a zymotic disorder, has not resulted from some of the atmospheric agencies before alluded to.[2]

It has been asserted that Pneumonia and "typhus" fever have a tendency to appear simultaneously, and it has hence been concluded that some connection may therefore possibly exist between these diseases. This belief is disproved by the returns of the Vienna hospitals, and also by Huss's statistics; though Huss considered that during the prevalence of these disorders Pneumonia is liable to assume the typhoid form.

I. *Influence of other Diseases in the production of Pneumonia.*—There appear to be at least six categories under which Pneumonia occurring in the course of other diseases may be classified:—

1. It may be the immediate effect of the poison producing the primary disease, or of the altered composition of the blood thus induced,[3] and in this light it is probable that many of the pneumonias occurring in the course of the acute febrile diseases should be regarded.

2. It may be the result of accidental products accumulating in the blood, as is

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identical with some forms of tubercle. It may also be recalled that Cruveilhier produced similar results (to which he attached the same interpretation) by injecting mercury into the trachea. Reitz (Sitzb. K. K. Akad. zu Wien, 1867; Math. Nat. Wissch. Cl. lv. 3) has varied these experiments by injecting caustic ammonia into the trachea. I have repeated this experiment in a dog. The result was an intensely developed membranous exudation, extending throughout the trachea and smaller bronchi, but becoming more fluid and puriform in the latter. There was no uniform lobar consolidation in the lungs, but these were studded throughout with small yellow spots, solid, not at all prominent, rarely exceeding the size of a pea, somewhat irregular in their outlines, finely granular on section, breaking down in various parts into cavities which in some places attained the size of a hazel-nut. These were filled with a diffluent puriform matter, and when near the surface, they projected like blisters from under the pleura by which they were covered. Death had occurred on the third day after the experiment. Dinstl, however (Oest. Zeitsch. Prakt. Heilk. viii. 1862, and Schmidt's Jahrb. 1866), has occasionally seen Pneumonia arise from the inhalation of irritating vapors.

[1] Arch. Gén. de Méd. 3e Sér. tome ii. 1837, p. 16.

[2] Ibid. xv. 1842, p. 130.

[3] Thus Dahl, "Norsk Mag. für Laegevidenz," xxii. Hft. 6; Virchow's Jahresb. 1868, ii. 95, has twice observed an epidemic of Pneumonia in the prison of Christiania. The first of these was in 1847; the second was in 1866–7, when of 366 prisoners, 62 had Pneumonia, or one-sixth of the whole number. The servants working outside were equally affected with the prisoners. In other years Pneumonia has been a rare event in the prison. Prof. Boeck, who was consulted by the Government, considered that overcrowding had a great influence in the production of the disease. In the "epidemic" of 1866–7, the weather was very cold during a great part of the prevalence of the disorder, and Pneumonia was common also in the surrounding district.

[1] Infections Krankheiten; Virchow's Handbuch, Sp. Path. Therap. ii. 43.

[2] The descriptions of epidemics of Pneumonia are only to be found in older writers, and the nature of the disorder must in some of these cases be considered at least doubtful. Lebert, "Path. Anat." i. 651, says, however, that he has convinced himself of the existence of epidemics of Pneumonia in certain parts of Switzerland. Further information on this subject may be obtained in the following works: Hirsch, loc. cit.; Ozanam, Hist. Méd. des Mal. Epidém., Paris, 1835; Lebecq de la Cloture, Obs. sur les Malad. et Consid. des Epidémiques, Paris, 1776–1778; Max Simon, Étude Pratique rétrospective, et comparée sur le Traitement des Epidémiques au 17e Siècle, Paris, 1859. (Quoted by Lebert, loc. cit.)

[3] O. Weber, in addition to other internal inflammations, has succeeded in producing diffuse Pneumonia by injecting the blood of a febrile dog into another healthy one. (Pitha and Billroth's Handbuch der Chirurgie, i. 610.) See also Virchow, Ges. Abhand. 660, *et seq.* Also Billroth, Archiv für Klin. Chirurg. vol. vi.

seen in albuminuria, and possibly in diabetes,—or it may arise from the mechanical or infecting influence of solid materials formed elsewhere, and conveyed by the blood current to the lungs, as in thrombosis and in some cases of pyæmia.

3. It may be the secondary result of other diseases affecting the lungs or air-passages, as tubercle or bronchitis.

4. It may be the effect of mere passive congestion, mainly of mechanical origin, arising either from valvular disease of the heart or from weakness of the circulation, aided by defective respiratory movement and dependent position, and in many cases by collapse of lung in the course of some of the acute febrile and also in that of chronic exhausting diseases.

5. It may be the result of a direct extension of diseases affecting other organs, as when abscesses of the abdominal viscera communicate with the lungs. In some cases Pneumonia, secondary to pericarditis, may have a similar origin.

6. It may be a purely accidental complication.

In many of the acute febrile diseases no other cause can be assigned for the occurrence of Pneumonia than the presence of a blood poison.[1] In others the mechanism is more complex, as in diphtheria and measles, when the effect is probably in part due to the secondary effects of bronchitis or collapse; and even in some cases of typhoid fever Pneumonia may rise either from embolism,[2] or from secondary blood-poisoning resulting from the ulceration of the intestines.

The characters also of the Pneumonia arising in the course of other diseases vary considerably. In some, as in measles and hooping-cough, it mainly presents the characters of lobular or broncho-pneumonia; in others, as in variola, the inflammatory changes may be either lobar, or may be disseminated irregularly throughout the lungs. The appearances in diabetes may be either those of the acute lobar form, or the Pneumonia may occur in disseminated nodules, tending to undergo a necrobrotic or cheesy change, and which appear to be closely allied to, if not identical with, the tubercular process. In albuminuria either the acute lobar form may predominate with firm exudation, or the inflamed part may present a smoother section, together with softer consistence and a more translucent appearance, arising from coexisting pulmonary œdema.

Of the acute specific fevers, measles is that most commonly attended by Pneumonia. The frequency of the latter disease varies, however, in different epidemics, and at different periods of the same epidemic;[1] and it is a more common complication of the disease during childhood than in adult life. Typhoid fever stands next in order of frequency—typhus fever, according to the statement of Dr. Murchison,[2] involving a minor degree of liability to the disease. In both these diseases, however, the data are somewhat uncertain, owing to the liability to hypostatic congestion of the lungs, which is commonly found when they prove fatal. In typhoid fever especially, the Pneumonia tends occasionally to assume the lobular and vesicular forms of the disease.

In scarlatina, Pneumonia is less common during the earlier stages, but is by no means rare when in its later periods it is complicated by albuminuria.

In glanders and farcy, secondary Pneumonia is extremely common. It usually assumes a disseminated form and tends to pass into suppuration, presenting in this respect many features common to pyæmia.[3]

Pneumonia is occasionally observed in cases of erysipelas.[4] In some cases it appears to be due to secondary blood-poisoning, and to assume the disseminated form of pyæmic Pneumonia.[5] In other instances, however, it appears to be rather of the nature of an intercurrent phenomenon, and approximates more or less closely in its characters to those of the acute primary disease; and it appears not improbable that the Pneumonia may, under these circumstances, originate from the

[1] It has been noticed by Andral that Pneumonia may appear with the first invasion of the exanthemata, and that its occurrence at these early periods sometimes coincides with an imperfect development of the eruption. In a case of variola he observed, during the invasion, crepitation in both lungs, with a viscous rusty expectoration, which vanished on the appearance of the eruption. I have recently seen a case where crepitation and dulness at the base of the lung disappeared within twenty-four hours after the eruption of variola had taken place. (Cf. Clin. Méd. iii. pp. 409–460.)

[2] I have seen a well-marked instance of this, where both lungs contained infarcta surrounded by secondary Pneumonia.

[1] Barthez et Rilliet, Mal. des Enfants, iii. 264. These authors observed 65 cases of Pneumonia and lobular Broncho-pneumonia in 167 cases of measles. Bartels (Virch. Arch. xxi. pp. 75–6), in an epidemic in 1860, found Pneumonia or Broncho-pneumonia in 12 per cent. of his cases, but these complications contributed 80 per cent. of the deaths which occurred.

[2] Continued fevers, p. 184.

[3] It is remarkable that, although this is the condition most ordinarily found in the human subject, yet that in the horse these diseases are associated with peri-bronchitis, and in this respect closely resemble tubercle. (Cornil and Ranvier, Manuel Histol. Path.)

[4] Stokes, Dis. of Chest, p. 339.

[5] See vol. i. art. "Erysipelas," by Dr. Reynolds, p. 321.

same blood-poison as that which gives rise to the erysipelas. It has also been observed to arise by a propagation of the inflammatory action from the skin, extending through the mouth, fauces, and air-passages to the lung tissue.[1]

In the course of acute rheumatism, Pneumonia is a not very uncommon complication.[2] In some cases it appears in a form truly metatastic with the rheumatic affection of the joints;[3] but most commonly the joint affection persists during its continuance. The influence of acute gout in the production of Pneumonia appears to be much less marked than that of rheumatism.

Other febrile states associated with disordered conditions of the blood are frequently causes of Pneumonia; but at present these cases have not been fully analyzed with regard to the mechanism of its production. Thus the statistics of Mr. Erichsen[4] show that it is common after severe surgical operations, 45 per cent. of deaths from these causes presenting signs of inflammation of the lungs. It also appears to be common in puerperal fever.[5]

Grisolle states that five-sixths of children affected with gangrene of the mouth suffered from intercurrent Pneumonia. It is also very common in the course of scurvy and purpura. In the latter disease I have seen it assume anatomically the acute primary form.

Albuminuria, associated with disease of the kidneys, is again a very common cause.[1] Rayer found Pneumonia in one-twelfth of these cases. The collection by Jaccoud,[2] of Frerichs and Rosenstein's returns, shows that the affection was found in 52 of 416 cases, or in 12·8 per cent. Dr. John Taylor,[3] however, found the frequency of Pneumonia to be 24 per cent. Becquerel,[4] in 129 cases of Bright's disease, found Pneumonia in 20 per cent.; but in 100 cases examined by Dr. Bright[5] it was only found in 6 per cent. Rosenstein[6] considers that Pneumonia in this disease is nearly as frequent as pleurisy.

It has been already stated that Pneumonia is a very common secondary result of bronchitis. The relation of the two diseases is threefold. In some cases they appear to be due to a common cause, and acute primary lobar Pneumonia may originate simultaneously with a general bronchitis. In other cases it appears to be the result of a direct extension from the bronchi, and under these circumstances it may appear in the form of "lobular" or "vesicular" Pneumonia; but in other instances the disease thus originating is in the lobar form, and offers no distinctive characters from the primary disease.[7] In the third class the Pneumonia is produced by the intervention of collapse, the mechanism of which process will be further considered hereafter. Bronchitis in the adult, as it would appear from the analysis of Grisolle, often precedes Pneumonia, having been observed by him as an antecedent in 76 out of 201 cases, and in 53 of these the catarrh was recent, *i. e.*, it had commenced within a month or three weeks before the Pneumonia appeared. Such antecedent bronchial catarrh, according to Grisolle's experience, is less common in the summer months. My own observations would lead me to the belief that bronchial

[1] See a case by Gubler, quoted in a thesis by Labbé, "De l'Erysipèle," Thèses de Paris, 1858, p. 57. Vulpian, Pneum. Second.

[2] Dr. Fuller, in 268 cases of acute rheumatism, observed 28 of Pneumonia. Dr. Latham, "Dis. of Heart," i. 161, in 136 cases found Pneumonia in 18. Dr. John Taylor (Med.-Chir. Trans. 1845, vol. x. p. 565) only observed it three times in 86 cases. My own observations would lead me to the belief that this complication is not infrequent. I have seen several cases of this class. In the autumn of 1865–6, several cases of acute rheumatism simultaneously admitted into hospital suffered from Pneumonia.

[3] Grisolle, p. 173, cites three cases of this kind; one from Andral, "Clin. Méd.," iii. 463: and a fourth where, in two consecutive attacks of acute rheumatism occurring at intervals of some years in the same patient, Pneumonia appeared and disappeared "eight or ten times, following the same course, and having the same duration as the joint affection."

[4] Med.-Chir. Trans. vol. xxvi.

[5] Tonnelé (Arch. Gén. xxii. 487) found Pneumonia in one-twelfth of the fatal cases of puerperal fever. Grisolle, p. 165, says that perimetritis is associated with septic pleurisy, and not with Pneumonia, while uterine phlebitis is more commonly associated with Pneumonia.

[1] Dr. Grainger Stewart (Bright's Diseases of the Kidney) gives the following data of the frequency of Pneumonia in the different diseases of the kidney associated with albuminuria: acute nephritis, 21 per cent.; contracted or cirrhotic kidney, 7 per cent.; waxy kidney, 4 per cent.

[2] Leç. Clin. Méd. 1867.

[3] Loc. cit. p. 565.

[4] Seméiotique des Urines, 1841.

[5] Guy's Hosp. Rep., 1836.

[6] Path. Therap. Nierenkrankheiten, p. 198.

[7] Some authors, and Rilliet and Barthez in particular, consider that this form results from extension of the disseminated variety, and they have termed it "Pneumonie vésiculaire," or "disséminée," or "lobulaire généralisée." It does not appear to me that this distinction can be always maintained; and, further, it not infrequently happens that in cases of acute pulmonary pneumonia, in addition to the lobar form, disseminated nodules are found in other parts of the lungs.

catarrh may not infrequently precede by two or three days the symptoms of invasion of Pneumonia. Bronchitis commonly precedes the Pneumonia of the aged.[1]

Phthisis is so commonly complicated with Pneumonia, that the latter may, as stated by Dr. Addison, be regarded as the immediate cause of a large proportion of the phenomena of this disease. The question of their mutual relations belongs, however, rather to the subject of Phthisis than to that of Pneumonia.

Congestive conditions of the pulmonary circulation are also a common cause of the disease. The influence of cardiac affections in its production is a very important one, and Pneumonia tends to appear in their course in a proportion of from one-third to one-fifth (Grisolle and Dr. King Chambers).[2] It is probably mainly to the influence of congestion that the inflammatory changes appearing in collapsed portions of lung are due, and it is not impossible that it contributes in a considerable degree to the Pneumonia complicating capillary bronchitis. The influence also of congestion in the production of the inflammation of the lungs which attends the later periods of life, when it mainly occurs in the most dependent parts of the lung—the hypostatic pneumonia of Piorry—is unanimously admitted.[3] Pneumonia being found in a proportion of one-sixth to one-seventh of chronic and cancerous diseases,[4] and of one-fifth of chronic diseases of the nervous system.[5]

ACUTE PRIMARY PNEUMONIA.

Symptoms.—The *invasion* of the disease is sometimes preceded by *prodromata*, which may exist for one or two days, or even longer,[6] before the outbreak of the severer symptoms. They are, however, very frequently wanting: when present they may exist as before stated, as a slight degree of bronchial catarrh, or in the form of general *malaise*, chilliness, loss of appetite, headache, pains and aching in the back and limbs, and an earthy or icteric tint of skin.[2] In old people the disease may be preceded for one or two weeks by headache and vertigo, epistaxis and lumbar pains.[3] Pyrexia of a marked kind is stated to precede sometimes by some days all other signs of the disease, but these cases are exceptional,[4] and it may be questioned whether pyrexia in such instances has not been caused by a central but undiscovered Pneumonia. There are not, as far as I am aware, any authentic thermometric observations recorded of the temperature during the prodromal period,[5] but Huss states that a slight degree of feverishness is sometimes observed. In a large proportion of cases,

[1] Dilatation of the bronchi appears to be a very common cause of secondary Pneumonia. Thus Barth (Mém. Soc., obs. iii. 1856) met with Pneumonia in 12 out of 40 cases of bronchiectasis. Biermer, Theorie Anat. der Bronch. Erweiterung (Virch. Arch. xix.), found it in 12 out of 54 cases; and Rapp (Verhand. der Würzb. Med. Gesellsch.), in 21 out of 24 cases. The Pneumonia thus met with is in some cases lobar, in others lobular. It occasionally passes into gangrene.

[2] Med.-Chir. Rev., Oct. 1853.

[3] An interesting case of this kind is quoted by Vulpian, "Pneumonies Secondaires," from Rayer, "Mal. des Reins," ii. 293. The patient was obliged to maintain the sitting posture, and the lower portions alone of both lungs were found affected with Pneumonia.

[4] Grisolle.

[5] Calmeil, Dict. de Méd., ii. 196.

[6] Such prodromata may last from one to two weeks, or five or six days, and then the Pneumonia may appear after a slight further exposure to cold. (Grisolle, p. 157.) Zimmermann (Prager Vierteljahresch. 1852, vol. xxxii. p. 97) gives a case in a young man where the prodromata had lasted a week. It may, however, be doubted how far these symptoms can be regarded as being in any respect special forerunners of the inflammation of the lungs; or whether they are not rather to be considered as symptoms of a bad state of health which predisposes to the disease.

[1] Grisolle estimates the frequency with which prodromata are observed in the adult as about one-quarter of all cases. In those to which I have had access the proportion has been 15 out of 53. They are much less commonly observed in children and in old people. Durand-Fardel noted their presumed absence in 20 out of 50 cases (Mal. des Vieillards, 470).

[2] Andral (Clin. Méd. ii. 284, obs. iv.) gives a case of a female who, after drinking largely while heated, was seized with diarrhœa and bronchitis; after ten days the diarrhœa ceased, and signs of Pneumonia then appeared.

[3] Hourmann et Dechambre, Arch. Gén. 2e Sér. xii.

[4] Grisolle, p. 187; Traube, Deutsche Klinik, 1857, p. 22.

[5] The only case absolutely bearing on this subject with which I am acquainted is one by Monthus, "Essai sur la Pneumonie Double." A patient was in the hospital for abscess of the foot. Her temperature had been normal throughout. One night she got a chill from a draught of cold air. In the morning she felt ill, and the temperature was 100·4; within a quarter of an hour a rigor supervened. At the commencement of the rigor the temperature was 100·9. During the rigor and for an hour after, the temperature was 105·8, it then fell to and remained at 103·6. On the following day crepitation appeared in the lung, and the temperature was 104°.

however, the disease commences suddenly and without previous warning, in persons who up to the moment of seizure had felt perfectly well, and it is not uncommon for the invasion to occur during the night after the patient has gone to bed in his usual health.

The invasion is most commonly marked by rigors, which are generally of a severe character. They form one of the most constant features of Pneumonia in adults, and their frequency and intensity are greater in this than in almost any other disease, with the exception of intermittent fever, pyæmia, and puerperal fever.[1] They are, however, commonly absent in most cases of secondary Pneumonia, and also in that succeeding to long-continued bronchitis. The rigor usually only occurs at the commencement of the disease, and it is rarely repeated, though this is sometimes observed.[2] In some cases it may appear subsequently to other symptoms, such as pain or cough, and in other instances it commences suddenly, after *malaise* and a general feeling of illness have existed during some days. When rigors are absent, the invasion of the disease may be evidenced by other symptoms, such as great prostration and pyrexia. In children also it may be marked by symptoms indicating the early implication of the nervous system, such as convulsions, vomiting, and headache or delirium, which may occur suddenly and without previous warning, or by the milder symptoms of stupor, restlessness, and loss of appetite. In old people sudden prostration and a semi-comatose state may be the first symptoms observed.[3] Rigors may precede by a period of from twelve to twenty-four hours, or even in some cases, of from three to four days, all other symptoms and local signs of Pneumonia with the exception of Pyrexia.[4] More commonly, however, other symptoms occur early, and particularly pain in the side, dyspnœa, oppression of the chest, cough, and rusty expectoration. In some cases the earlier symptoms may be headache, or vomiting, or diarrhœa;[1] severe lumbago is also occasionally observed.

Of the symptoms indicating the pulmonary affection, pain in the side is one of the earliest and the most constant, and it may be the first symptom noticed, in some cases preceding the rigor.[2] It is commonly very acute, and its presence is the cause of great anxiety and distress to the patient. It usually corresponds to the site of the Pneumonia, but exceptions to this are occasionally observed, and it assumes at times the character of lumbago. Much discussion has arisen as to its cause, but probably in most cases it is to be attributed to concomitant implication of the pleura. It generally continues during the earlier stages of the disease, tending to diminish towards the third or fourth day, but sometimes lasting until the eighth or ninth. It is aggravated by deep inspirations and by cough, and it occasionally coexists with marked tenderness on pressure: I have observed it to be associated with considerable cutaneous hyperæsthesia of the affected side.

The other symptoms of the declared disease usually show themselves within twenty-four hours of the invasion, and the aspect of the patient is then to a certain degree characteristic. There is great prostration — a flushed but somewhat earthy or dusky tint of face, tending in some cases to lividity. The skin is pungently hot, sometimes dry, but not infrequently perspiring. The countenance is expressive of anxiety, particularly when pain is present; at other times the expression is dull and heavy. The respiration is accelerated, and when pain is severe it is shallow and irregular; and the expansion of the *alæ nasi* with the respiratory act is strongly exaggerated. Dyspnœa and a great sense of thoracic oppression are frequently but not constantly present. Speech is rendered difficult and

[1] Huss (loc. cit.) observed rigors in 80 per cent. of his cases. They occurred in 145 out of 182 cases observed by Grisolle, and in 110 of these they were the first symptom noticed. Louis observed rigors as the initial symptom in 61 out of 79 cases. I find their entire absence recorded in 9 only out of 53 cases. In 34 they were distinctly present.

[2] Louis, Rech. Fièvre Typh. ii. 128.

[3] In 35 cases of Pneumonia in old people, Durand-Fardel observed the phenomena of invasion to be as follows: In 7, rigors only; in 8, rigors and pain in the side; in 6, rigors and vomiting; in 8, pain in the side alone; and in 6, vomiting alone. Dyspnœa was rare at the outset, and was only observed in 12 out of 50 cases. It was comparatively constant at later periods.

[4] This state, when protracted, constitutes the "Febris Pneumonica" of older writers. The term is also applied to some forms of "Latent Pneumonia."

[1] Headache occurred among the first symptoms in 12, and vomiting in 9, out of 53 cases which I have been able to analyze. For the opportunities of making many of these observations I am indebted to the kindness of my colleagues, Sir W. Jenner, Dr. Hare, and Dr. Reynolds, who have allowed me access to the pulmonary cases under their care, and also have permitted me to use their case-books to supplement, when necessary, my own observations.

[2] In 201 cases analyzed by Grisolle, pain in the side was only absent in 29. In 182 it appeared within the first twelve hours in 121. In four it only appeared on the third or fourth day, and in two of these latter its invasion was marked by the recurrence of an intense rigor.

broken by the accelerated respiration, the dyspnœa, the cough, and the thoracic pain. There is a short hacking cough, attended with a labored expectoration of viscous, tenacious, and rusty sputa. The pulse is accelerated, it is full, and occasionally resisting, but more commonly it is soft, or small, or dichrotous. The decumbency is in most cases dorsal; orthopnœa is less frequently observed. Tremors and subsultus tendinum mark severe cases, which may also be complicated by convulsions or delirium. The urine is scanty and high-colored. There is complete anorexia and great thirst; the tongue is dry and furred, and the lips cracked; vomiting is sometimes present; the bowels are usually confined, but diarrhœa is by no means rare. These symptoms may last with unabated or even with increasing intensity, for a period varying from the third to the tenth day of the disease, within which time a notable improvement is usually suddenly observed; the temperature falls abruptly, the flush disappears and gives way to pallor; the skin becomes bathed with a profuse perspiration; the pulse and respiration, particularly the latter, fall in frequency; the dyspnœa and distress are markedly diminished; the cough becomes freer and looser, and the rusty sputa ordinarily disappear. In favorable cases the patient at once feels and declares himself better, and the appetite may return immediately; while in severe cases, or in weakly patients, in spite of the fall of temperature, an intense degree of prostration, amounting even to collapse, and sometimes ending fatally, ensues. When this crisis has taken place there is usually a rapid and continuous improvement both in the general symptoms and in the physical signs, which may, however, be occasionally interrupted by a relapse and by a return of the febrile condition after an interval of twenty-four, forty-eight, or seventy-two hours. In some cases, however, the crisis is indeterminate, the defervescence of the pyrexia is gradual, and the improvement slow and protracted. In unfavorable cases death may occur from asphyxia or collapse within the first ten days, without the subsidence of the pyrexia; or even, as before stated, after the temperature has fallen to the normal standard.

The symptoms now enumerated require, however, a more special consideration.

Respiratory System.— Accelerated respiration and dyspnœa are among the most marked phenomena of Pneumonia. The latter is not, however, constant as a subjective symptom, and seems to bear, in many cases, no relation to the rapidity of the breathing.[1] It is, however, occasionally the first symptom observed, especially in secondary pneumonias, and it may exist to an intense degree, producing a sense of impending asphyxia; in children it occasionally occurs in suffocative paroxysms threatening death.[1] Its intensity is commonly but not constantly in proportion to the rapidity of the invasion and of the extension of the disease. It is much aggravated by the coexistence of general bronchitis together with the Pneumonia. It has been said to be more intense when the inflammation affects the apex of the lung, but Grisolle has shown that facts do not confirm this opinion. In some cases the sensation of dyspnœa is probably masked by the prostration of the nervous system.

The rate of respiration is greatly quickened. The number of respirations per minute is seldom less than 30, often 35 to 40, and they may even reach 60 or 70. The acceleration of the breathing is generally proportionate to the extent of lung affected, but this is not invariably the case. It is increased by coexisting bronchitis, or by any cause interfering with the thoracic expansion, such as pregnancy. The acceleration of the breathing is proportionately greater than that of the pulse, and hence arises the perverted pulse-respiration ratio which is especially insisted on by Dr. Walshe as one of the earliest signs of Pneumonia.[2] This perversion may reach the limits of 60 respirations to 100 pulsations per minute; or in some cases, when the pulse remains slow, the ratio has been observed of 56 pulsations to 60 or 70 respirations per minute.[3] The respiration is commonly more rapid in children than in adults, and in them the anhelation may be extreme and the respiratory movements irregular. In some cases of asthenic Pneumonia the pulse-respiration-ratio may not vary markedly from that of health.[4] It is not improbable that the extreme degrees of frequency of respiration may in some cases be due indirectly to peculiar states of the nervous system.[5] The breathing, in ad-

[1] Dr. Walshe says that he has seen patients breathing at the rate of 50 or 60 per minute without any sense of dyspnœa. It is possible that in some of these cases the blood is sufficiently aërated by the accelerated breathing to prevent the sense of dyspnœa being felt. In some cases, when the respiration is less rapid, the sense of dyspnœa is extreme.

[1] Ziemmsen, loc. cit.

[2] Dr. Walshe (Dis. of Lungs, Edit. 1860, p. 366) says that the return to the normal ratio may, on the other hand, be one of the first signs of improvement.

[3] Ibid. [4] Ibid.

[5] Traube (Annalen der Charité, vol. i.) considers that the rapidity of the breathing is in part due to pain, and in part to the high temperature of the blood affecting the nervous centres, as it is diminished by the application of cold.

dition to being rapid, is commonly shallow, particularly when pain in the side is severe.

Cough is not only an almost constant, but it is one of the earliest symptoms.[1] It is short and hacking, and rarely paroxysmal, though it sometimes becomes so in children in the later stages of the disease. The violent paroxysms, resembling those of hooping-cough, mentioned by Rilliet and Barthez, are thought by Ziemssen to be more characteristic of Broncho-pneumonia. It is often, on the other hand, less frequent in old people and in children than in adults, and it has been observed in the former, that a cough previously existing, and caused by bronchitis, has ceased or has become greatly diminished on the invasion of Pneumonia. The cough often ceases when a fatal termination is approaching.

The expectoration which attends the cough usually presents characteristic features, depending on the admixture of blood. This is not, however, always apparent at the outset, when the sputa may be frothy and aërated. They soon, however, tend to become peculiarly viscous, adhering with great tenacity to the containing vessel, and owing to this quality they are often expectorated with great difficulty. They are at the same time transparent, having various tinges of reddish-brown or saffron, or they may be of a lighter tint, resembling apricot jelly or barley-sugar. The most common color is that characterized familiarly as "rusty," which aptly expresses their appearance. In rarer cases they may sometimes, in the earlier stages, present a brighter tint, or even a rose color, but this is by no means so strongly marked as in the earlier stages of acute bronchitis, though in Pneumonia the sputa of the first few hours are commonly of a brighter red than subsequently, and streaks and specks of blood may appear in them. Dr. Walshe remarks that profuse hæmoptysis is commonly a sign of coexisting tuberculosis. In the cases when I have seen this, the same connection has been distinct. Huss also confirms this observation, but adds that in the Pneumonia complicating heart disease, the sputa may contain an unusual quantity of blood.

In other cases the sputa may be more watery, almost diffluent, of a dark purple color, and occasionally offensive. This appearance, familiarly known as "prune-juice," and which probably results, in part at least, from the presence of œdema of the lungs, is commonly considered a sign of gray hepatization; but this connection is by no means invariable, for such sputa may coexist with red hepatization,[2] and may be absent when gray hepatization is found post mortem;[1] sputa of this character are, however, to be regarded as indicative of a grave form of the disease.

The sputa which have now been described may be regarded as almost pathognomonic of the pneumonic process in some of its forms, but variations in their characters may be sometimes observed, and they are said at times to present a greenish tint.[2] They may also at times exhibit appearances during some days, differing but little from those of bronchitic sputa, and without any blood-tinge which is appreciable to the naked eye, but in these cases they are commonly more tenacious and gelatinous than those seen in simple bronchitis.[3] In severe cases they may be simply purulent throughout.[4] In other instances the viscidity is less apparent, and the prune-juice sputa in particular are often diffluent and watery.

The rusty sputa are, however, the most usual form, and they commonly are present early in the disease, and are among its first symptoms.[5] The time of their appearance is, however, often considera-

[1] In 8-9ths of cases (Grisolle).

[2] Cases are recorded by Andral where prune-juice sputa preceded all the physical signs of consolidation (Clin. Méd. iii. obs. 28, p. 361), and when they coexisted only with red hepatization (Ib. obs. 39, p. 392); also another case where they appeared on the fourth day, but were replaced on the subsequent day by ordinary expectoration, the patient proceeding afterwards to recovery (Ib. obs. 40, p. 393). Durand-Fardel has also noticed them in the earlier periods of the disease (Mal. des Vieillards, 477). Huss also states that prune-juice sputa are by no means constant accompaniments of gray hepatization, but that they are common in the Pneumonia of drunkards, and also in tubercular Pneumonia.

[1] See Andral, loc. cit. obs. 24, pp. 350–1. Of two fatal cases of gray hepatization coming under my own observation, this character was not observed in one. In the other the sputa were diffluent, and had the tint of burnt sienna.

[2] Grisolle. These have not come under my own observation in the earlier stages. A greenish tint is not uncommon during the period of resolution. When icterus complicates Pneumonia, a greenish tint is sometimes observed. (Andral, loc. cit. obs. 55, p. 440.)

[3] Sputa of this kind are, however, more common in Pneumonia which is secondary to bronchitis, and particularly in the Pneumonia accompanying influenza.

[4] Only one such case has come under my own observation.

[5] In 191 cases observed by Grisolle, characteristic sputa existed on the second day of the disease in 71; they were present in 33 of 53 cases analyzed by myself. Of the cases in which they were absent, 4 were in children, in another the Pneumonia was secondary to albuminuria, and they were absent in one fatal case.

bly delayed, and they may not be seen until the fifth or sixth day, or even until the twelfth day. In one case coming under my own observation, there was not a vestige of expectoration until the tenth day, when the Pneumonia was rapidly approaching resolution, and the amount was then limited to two small rusty masses expectorated on each of two consecutive days.[1] When present, they commonly continue through the first five or six days, but they may preserve their rusty tint until the ninth day.[2]

In some cases, otherwise typical, no expectoration whatever occurs throughout the whole course of the disease. In others the characteristic sputa may be absent during the acute period, and only a moderate amount of bronchitic, or slightly purulent, or pigmented sputa may appear during resolution. In cases of gray hepatization and of abscess of the lung the sputa may be purulent or creamy-looking. When gangrene supervenes they become offensive, and fragments and débris of pulmonary tissue may be found in them. The entire absence of expectoration is said to be more common in Pneumonia of the apex than in that of the base of the lung.[3] Children under six years of age seldom if ever expectorate, but Ziemssen says that he has found the rusty tint in the sputa of infants when vomiting has taken place. They are also often absent or only mucoid in the Pneumonia of old age,[4] and in many cases of secondary intercurrent Pneumonia, and in that complicating delirium tremens. The sputa often cease or fail to be expectorated when the disease is approaching a fatal termination.[5]

Both purgation and bleeding diminish or check the expectoration.

When Pneumonia complicates other diseases of the lungs, the rusty sputa may be more or less masked by other forms of expectoration present, or they may replace these.

Remak[1] first described, as one of the phenomena of Pneumonia, casts of the air-vesicles and of the minuter bronchial tubes, which may be found in the sputa when these are floated in water. He regarded them as pathognomonic of the exudative period, but they are by no means constant.[2]

Histologically the main elements of pneumonic sputa consist of swollen epithelium cells, which have assumed, by imbibition, the spheroidal form; large mucoid cells, sometimes with double nuclei, and occasionally tinged with imbibed hæmatine; swollen cells of columnar epithelium, occasional granule cells, free oil-globules and blood-disks. Dr. Walshe says that true pus-cells are never found in the rusty sputa of Pneumonia. They may however appear when the disease is approaching resolution, and in this stage large round cells containing granules of black pigment become a very predominant feature, mingled with free nuclei, free pigment-granules, and much granular débris. Chemically the sputa contain mucus and albumen.[3] Sugar has been observed in them by Dr. Walshe[4] and by Dr. Beale,[5] and tyrosine by Griesinger.[6] They contain, in the earlier stages, a small amount of organic constituents, and an excess of fixed salts[7] in proportion to

[1] Grisolle questions whether rusty sputa expectorated only during convalescence may not be considered critical. In the case in question they only occurred after the thermometric crisis, and their amount is almost invariably too small to permit them to be regarded as a true critical evacuation.

[2] Exceptional cases are recorded where viscous and rusty sputa may continue during longer periods, as in a case by Dr. Stokes, "Diseases of Chest," 361, where a patient with broken ribs continued to expectorate sputa of this character for weeks after the physical signs of Pneumonia had disappeared. Andral also quotes a case where rusty sputa continued to the nineteenth day, lasting nine days after all physical signs had disappeared. (Loc. cit. 526.)

[3] Out of 14 cases where the sputa were entirely absent, in 7 the apex was the site. (Grisolle.)

[4] Rusty sputa were observed in 17 out of 61 cases of Pneumonia in old people observed by Hourmann and Dechambre, and in 18 out of 50 cases observed by Durand-Fardel.

[5] The absence of expectoration may, as suggested by Andral, be sometimes due to the absence of bronchitis, or to the viscosity of the exudation in the air-vessels. The absence of expectoration in some cases where resolution is very rapid is a remarkable evidence of the absorptive power of the lung.

[1] Diagnostische und Pathognostische Untersuchungen, 1845.

[2] Biermer says that in 25 cases he failed to find them six times. (Die Lehre von Auswurf, p. 52.)

[3] Scherer, quoted by Biermer, loc. cit. p. 114.

[4] Loc. cit. p. 367.

[5] Med.-Chir. Trans. vol. xxxv.

[6] Bleuler, Clin. Beobach. über Pneumonie, Diss. Inaug. (Zurich, 1865), p. 37. There was no icterus in this case. The patient recovered.

[7] Biermer, loc. cit. Beale, loc. cit. In three cases Dr. Beale found the following proportions of fixed salts and of chloride of sodium in the sputa:—

In 100 parts of solid matter.	1st case.	2d case.	3d case.
Fixed salts . . .	24·78	32·86	20·67
Chloride of sodium	10·12	18·11	12·67

In another fatal case where the sputa contained 9·83 per cent. of fixed salts in the solid matter, the blood taken from the heart con-

the serum of the blood, but this excess is reduced during resolution, when the fixed salts are diminished in amount, probably owing to their elimination by the kidneys. Among these salts the chlorides are sometimes in excess.

In some cases the sputa tend to assume an acid reaction. This was noticed by Dr. Beale, who suggested that it might be due to the pneumic acid discovered by Verdeil, and found by him to be increased in the inflamed lung.[1] The true explanation would appear to be that afforded by the observations of Bamberger, that they are markedly deficient in alkaline phosphates when contrasted with the sputa of simple catarrh.[2]

The expired air, as Nysten and Dr. Walshe have observed, is colder than natural, and the amount of carbonic acid excreted is also diminished.[3]

The physical signs indicative of the disease commonly make their appearance within twenty-four or forty-eight hours from the symptoms of invasion, but they may be undiscoverable for three or even four days, though probably when their appearance is thus delayed the Pneumonia may be central.

In the order of their typical sequence they may be stated to consist of the following signs, corresponding to the anatomical stages of engorgement, hepatization, and resolution: among them, however, certain varieties occur:—

(1) Altered characters of the respiratory sound, which may be either weaker or harsher than natural, and attended or immediately followed by fine crackling râles.

(2) Dulness on percussion, attended by bronchial or tubular or suppressed breathing, bronchophony, and increased vocal fremitus, together with diminished respiratory movement, chiefly affecting the act of expansion.

(3) The return of crepitation, usually in a coarser form;—gradual diminution of percussion dulness, together with the return of the respiratory movements and of the characters of the respiration and of the vocal resonance and fremitus to the healthy standard.

(1) *The Congestive Stage.*—The indications of this stage are in most cases somewhat uncertain. There may, however, occasionally be noticed, even at an early period, a deficiency of pulmonary tone on percussion, not amounting to absolute dulness, but presenting this character in an increasing degree as hepatization advances. In some cases, however, during the early stages, the percussion note may be distinctly tympanitic. Sometimes, as was first noticed by Dr. Stokes, an increased harshness of the respiratory murmur may be the first phenomenon observed, but this is not constantly present, although it may occasionally be heard at the confines of a part where hepatization is extending. In some cases, however, the respiratory murmur is weakened and loses in clearness and softness, acquiring almost *ab initio* the character of the "indeterminate" breathing of Skoda.[1]

The existence of fine crepitation in this stage is less constant; when present its characters may be best described in the terms of Dr. Walshe as occurring "in puffs more or less prolonged, but rapidly evolved, composed of a variable, sometimes immense number of sharp crackling sounds, all perfectly similar to each other; conveying the notion of minute size; dry; coexisting exclusively, except in rare cases, with inspiration; and, once established, remaining a persistent condition until superseded by other phenomena." The simile introduced by Dr. Williams, between this râle and the sound produced by rubbing the hair between the fingers close to the ear, is so truthful as to have become almost proverbial. For its proper evolution it is often necessary that a full inspiration should be taken. It requires, under these circumstances, to be distinguished from the râle produced on the first full expansion of a portion of the lung which has been previously in a condition of imperfect action, either from muscu-

tained only 2·82 per cent. Healthy pulmonary mucus, according to Hasse, may contain 18 per cent. of fixed salts, and the mucus of influenza, according to Wright, contains 8·9 per cent. (Quoted by Dr. Beale.)

[1] See Gaz. Méd. 1851, p. 777; also Robin et Verdeil, Chem. Anat. Phys. ii. 460–1.

[2] Wurzburg Med. Zeitsch. ii. Nos. 5 and 6. Bamberger's observations contain the following interesting facts in relation to the composition of pneumonic sputa:—

(*a*) They contain no alkaline phosphates, while catarrhal sputa contain 10 to 14 per cent. of alkaline earths.

(*b*) In catarrh the soda is to the potash as 31 to 20, while in Pneumonia the soda is to the potash as 15 to 41.

(*c*) Sulphuric acid in catarrh is equal to 3 per cent., in Pneumonia to 8 per cent.

(*d*) At the period of resolution the chemical character of pneumonic sputa approaches the catarrhal type.

[3] Walshe, loc. cit. This latter phenomenon is common to many acute diseases.

[1] Dr. Walshe, p. 355, states that when congestion is near the surface the respiration is weaker and harsher. When the part affected is deeply seated, the intervening healthy pulmonary tissue may give rise to puerile breathing. He has also observed a "fair number of cases in which exaggerated breathing, coupled with febrile excitement and slight pain in the side, were the earliest indications of a central Pneumonia eventually travelling to the surface."

lar weakness or from pleurodynia. The latter, however, disappears after one or two deep respirations, while the true crepitant râle is, as above stated, persistent.

The crepitant râle may often be mingled with sibilant or sonorous râles, or, in other cases, where pre-existing bronchitis passes into Pneumonia, with coarser bubbling râles.

The râle is often wanting in children, and both in them and in old people it is commonly coarser and less rapidly evolved than in the Pneumonia of adults.

In some cases, however, when the stage of engorgement passes rapidly into that of hepatization, crepitation is not heard, even though the Pneumonia be developed under direct observation.[1] Occasionally it may only be heard after bronchial breathing has appeared.

The mechanism of the crepitant râle is not yet determined. The two leading theories respecting its mode of production are (1) that it may be produced by air and the viscous exudation matter in the pulmonary vesicles, and (2) that it is due to the expansion of the parietes of the vesicles previously agglutinated together.[2]

In rare cases bronchial breathing may be heard during the congestive stage.[3] Vocal fremitus and vocal resonance are increased in proportion to the condensation of the pulmonary tissue, but true bronchophony is not heard.

(2) *Stage of Hepatization.*—The crepitant râle last described may disappear at various periods of the second stage, or it may continue throughout its entire course, becoming coarser as resolution advances.

The characteristic physical signs of the second stage depend, however, on the filling of the air-vesicles with the products of inflammation, by which the part so affected is distended to the degree of medium or full insufflation. In consequence of this all further expansion movement of the affected part ceases, though thoracic elevation continues, and a certain though not extensive degree of enlargement of the affected side may ensue.[1] The distended lung may even encroach on the mediastinum,[2] and may occasionally cause a slight displacement of the heart.[3] The prominence or obliteration of the intercostal depression is, however, not seen to the extent observed in cases of pleuritic effusion, although they sink to a less degree than normal during the act of inspiration.

The percussion note over the affected part loses its normal pulmonary resonance. In some cases it becomes in the early stages slightly tympanitic in quality, and it may retain this character anteriorly when the dull note posteriorly indicates complete consolidation of the latter region. Over lung completely consolidated, the percussion note may be almost toneless, and the sense of resistance is greatly increased, though neither of these qualities is so strongly marked as in the presence of extensive pleuritic effusion.

Instead of absolute tonelessness the note may, however, be tubular or amphoric. The tympanitic quality is less common when the consolidation has attained its maximum intensity,[4] but it sometimes returns during the progress of resolution. In the earlier stages it is often necessary to compare the percussion note on the two sides, in order to detect a slight degree of dulness on that affected. The contrast becomes increasingly marked as consolidation advances.

When the Pneumonia affects the base, the upper part of the lung often continues to give excessive or even tympanitic resonance; and a cracked-pot sound may sometimes be elicited here when the chest wall is elastic. The note under the clavicle is, however, rarely so markedly amphoric or tubular as that found in the same situation in cases of pleuritic effusion.[5] The limits of percussion dulness are sometimes sharply defined, but occa-

[1] Walshe, loc. cit. 356. Dr. Walshe states also that "the diagnosis of Pneumonia must be made once in every four or five cases independently of the crepitant rhonchus" (loc. cit. 337). In the writings of Laennec and Andral great stress is laid on the râle. The latter author describes, on the strength of its persistence, a case where the stage of engorgement lasted eight days, and ended fatally without hepatization. It is probable that this was only a case of capillary bronchitis. (Clin. Méd. iii. 297, obs. viii.)

[2] Walshe. Dr. Walshe, however, has observed a râle indistinguishable from true pneumonic crepitus in some cases of pulmonary œdema. (Loc. cit. p. 123.)

[3] Traube (Annalen der Charité, i. 286) says that bronchial breathing may occur during the stage of engorgement when the combined effect of œdema and of swelling of the pulmonary tissue is sufficiently great to expel air from the pulmonary vesicles.

[1] This point has been the subject of considerable discussion, but the enlargement appears to be settled in the affirmative. Dr. Walshe, however, states that general enlargement of the side is never the resultant of Pneumonia alone.

[2] Walshe.

[3] Ibid.

[4] This is noticed by Skoda, and referred by him to the lung still containing some air—a proposition also maintained by Dr. Hayden (Dublin Journ. 1866, xli.). Dr. Bäumler attributes it in some cases to relaxation of pulmonary tissue, in others to a note conducted from the larger bronchi. (Deutsch. Arch. Klin. Med. i. 145.)

[5] Walshe.

sionally they are indistinct. In the latter case the percussion note at the margins of the inflamed part may yield a tympanitic resonance, or may have its natural resonance impaired by a pneumonia extending deeply.

Blowing or bronchial respiration passing into tubular and intensely metallic breathing, distinguish this stage.[1] These characters usually succeed those of the first period with great rapidity. Grisolle describes, under the title of "bruit de taffetas," from its resemblance to the noise produced by the tearing of linen, an intermediate sound, occasionally heard between the disappearance of the crepitation and the supervention of bronchial breathing. The bronchial character is heard first during expiration, but it subsequently attends the inspiratory sound also.

In some cases, however, when there is no evidence of pleuritic effusion, all respiratory sound may be completely absent over hepatized lung. The cause of this is uncertain, for post-mortem evidence has shown that it does not necessarily depend on complete exclusion of air from a large tract of lung, since under such circumstances tubular and bronchial breathing may persist, and, on the other hand, respiration may be absent when only a small portion of pulmonary tissue is affected; nor does it necessarily depend on the obstruction of the bronchi by exudation matter.[2] In other cases tubular breathing may alternate with absence of respiration.[3] The intensity of the bronchial or tubular breathing appears to depend in some measure on the size of the bronchial tubes included within the portion of lung affected.

The vocal resonance is increased in intensity, and is at the same time altered in quality, acquiring the character known as bronchophony. The cough may also acquire a bronchial character. Intense whispering pectoriloquy may be occasionally heard.[1] The heart's sounds are also sometimes heard with undue intensity over the affected lung.[2]

The vocal fremitus is generally increased over the affected side. In comparatively rare cases, however, this is not observed; the difference between the two sides may be so slight as to be scarcely perceptible, or the fremitus may even be less on the affected side. In some exceptional cases, however, vocal fremitus, vocal resonance, and the respiratory murmur may all be simultaneously absent,—a condition when the diagnosis from a case of pleurisy might present some difficulties.[3] (See Diagnosis.)

In some instances pulsation may be felt over the affected lung. It is a disputed point whether this is due to the transmission of the cardiac impulse, or to increased pulsation in the arteries of the inflamed lung.[4]

The signs now enumerated are most distinct when the inflammatory consolidation has reached the surface. When it is seated in the deeper portions of the lung, and the more superficial layers are left unaffected, the physical signs may be comparatively obscure. Laennec thought that crepitation and bronchial breathing could be heard deeply; and this may sometimes be the case, though instances occur where neither of these signs are distinct. The signs also derived from alterations in the vocal fremitus, and resonance, are usually wanting under these circumstances.[5] Where inflammation of the pleura complicates the Pneumonia, friction is commonly heard during its whole course. It may, however, be absent during complete consolidation, owing to entire loss of movement of the hepatized lung, and also when effusion is extensive. When this takes place, the dulness increases in extent, and the resistance is greater. Bulging of the affected side becomes more distinct, and displacement of the heart occurs if the left side be affected; and sounds of respiration usually become weaker, and the bronchial breathing less distinct; but the intensity of this, and the site in which it is heard, depend on the proportion of fluid present.

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[1] "The tubular form (of respiration) occurs in perfection in but one condition of lung, that of hepatization; so true is this, that tubular and pneumonic breathing may be used as convertible phrases, but not infrequently Pneumonia runs its course without having produced true tubular breathing, diffused blowing alone being audible." (Walshe, loc. cit. 122.)

[2] This view is, however, affirmed by Skoda, who says that the auscultatory phenomena of respiration may be restored after coughing.

[3] Dr. Walshe (loc. cit. 360) has traced this in one case to pressure on the main bronchus. Other theories advanced have been that of Grisolle, that it may be due to complete loss of elasticity of the lung; or of Dr. Gairdner, that it is due to collapse from obstruction of the bronchi.

[1] Walshe. This, according to my own observation, is not very uncommon.

[2] Ibid.

[3] Wintrich, Virchow's Handb. vol. v. Abth. i. p. 299. In this case also the bronchi were obstructed by firm exudation matter. The case was mistaken for pleurisy, and paracentesis was attempted. There was no fluid in the pleura.

[4] The latter opinion is denied by Grisolle, but supported by Graves, Stokes, and Skoda, and admitted as a possibility by Dr. Walshe.

[5] This subject will be further alluded to under the head of "Diagnosis."

The fremitus is commonly diminished. Bronchophony may also be diminished below the fluid, or may continue at its level, or the vocal resonance may in the latter position assume an ægophonic tone.

The period necessary for the evolution of the different physical signs varies. The duration of the initial stage of congestion may, as has been already stated, extend over two or three days, and bronchial breathing and distinct percussion dulness may not appear until the second or even the fourth day, and this appears to be more commonly the case with Pneumonia of the apex. In other cases hepatization may advance so rapidly that a large tract of lung may be consolidated in from twenty-four to forty-eight hours, or bronchial breathing may be heard within twelve hours from the period of invasion.

The condition of the unaffected lung is usually that of increased functional activity. In some cases it is hyper-resonant on percussion, and the respiratory murmur over it, and over the sound parts of the affected side, is of an exaggerated or puerile type. In three cases I have observed that bronchial breathing friction, and moist râles were heard over the healthy side where resonance on percussion has been perfect, for a distance of more than a hand's breadth extending outwards from the scapula.[1] These signs disappeared *pari passu* with the return of the affected side to a normal condition. The vocal fremitus was not increased over the unaffected side, although a bronchophonic tone of the voice was conducted for a short distance, but not so far as the bronchial breathing. It seems difficult to explain these phenomena on the theory of consonance, and my own conviction is that they are due to direct conduction.

(3) *During the stage of resolution* the abnormal physical signs commonly disappear in an inverse order to that in which they originated. Improvement is generally first manifested by a reappearance of the crepitant râle. This râle — the *rhonchus crepitans redux* — is usually coarser and less rapidly evolved than that heard during the progress of hepatization; it tends to pass into a more liquid form—the subcrepitant râle—and occasionally it acquires a distinctly fine bubbling character. In some instances, however, resolution may proceed rapidly without the occurrence of redux crepitation. Sibilant and sonorous râles also appear in the affected part, and sometimes in other portions of the lung. The dulness on percussion gradually disappears; the tubular breathing diminishes in intensity, it loses its metallic quality, and both it and the bronchial breathing pass into blowing respiration, which finally becomes indeterminate or simply weak. Similar changes occur in the bronchophonic tone of the voice, but the vocal fremitus and resonance usually continue intensified as long as the percussion note remains less resonant than natural.

The signs which persist the longest are some dulness on percussion, and the subcrepitant or fine moist râle, and the latter may often remain during a prolonged period after the other physical signs have disappeared. In some cases, however, when the resolution is very rapid, the redux crepitation may be wanting, and the dulness and altered characters of the respiration may vanish within twenty-four hours, giving place to a weakened or indeterminate respiratory sound. Friction also may continue long after the other physical signs have disappeared.

Generally, though occasional exceptions are observed, the parts last affected are those in which the signs of resolution first appear. In some instances, however, I have noticed the dulness disappear in irregular patches over the consolidated part. When a whole lung has been consolidated, the resolution usually commences at the apex. If this is not the case the existence of tubercle may be suspected.[1] In cases of double Pneumonia the lung last affected may first show signs of improvement; but occasionally the resolution of that first attacked may progress, while hepatization is still advancing in the other.

(4) The physical signs *of gray hepatization* and of *diffuse suppuration of the lung*,[2] present nothing characteristic. Occasionally a high metallic bubbling râle, as described by Stokes, supervenes, while dulness on percussion still persists; but I have observed this in a case where, post mortem, the lung was found to be almost entirely in a state of red hepatization. The formation of a circumscribed abscess (a very rare event in Pneumonia not arising from secondary deposits in pyohæmia) is only discoverable by the local signs of the formation of a cavity, together with profuse purulent expectoration, which is often offensive, and in which the elastic tissue of the lung may sometimes be found. Gangrene of the lung — also a rare event in primary Pneumonia — is mainly to be recognized by the signs of a cavity, coupled with the peculiar fetor of the sputa and the expectoration of débris of pulmonary tissue.

Circulatory System.—The pulse is almost invariably accelerated, though exceptional

[1] Barthez and Rilliet (i. 460) describe bronchial breathing as being sometimes heard close to the spine on the unaffected side.

[1] Walshe, loc. cit. 372.

[2] It may be doubted whether the latter ought to be described as a separate condition. (See Pathology.)

cases occasionally occur, particularly in old people,[1] when this is not observed. In adults, in cases of moderate severity it usually ranges from 90 to 120[2] pulsations per minute, but it may reach 130 or 140, and in children 160, 180, or 200, or it may be so rapid as to be uncountable. The extreme degrees of frequency of the pulse in children are commonly only observed in the earlier periods of life. In some cases the pulse may become notably retarded before the fatal issue.[3] A pulse above 130, or even 120, is, except in children, a very unfavorable sign.[4] The frequency of the pulse commonly, but not always, bears a certain proportion to the acceleration of respiration, and a similar proportion may within certain limits be observed between the frequency of the pulse and the degree of temperature attained.[5]

In characters a pulse of moderate frequency is commonly during the earlier periods full, but soft; it may, however, be tense and incompressible.[6] These characters tend, however, to diminish by the fifth day, when the pulse usually becomes smaller and often acquires a dichrotic character. A rapid pulse is, however, generally both small and weak. A small pulse may at times be associated with signs of distension of the right ventricle, particularly when the Pneumonia is extensive and other signs of defective aëration of the blood are distinct, but it may occasionally be observed when these are not marked, and when the distension of the right side of the heart is not demonstrable either by percussion or by increased post-sternal or epigastric impulse. It is, however, *à priori*, extremely probable, and it is also confirmed by post-mortem observation, that overloading of the right cavities of the heart is the direct result of the obstructed pulmonary circulation, and the immediate effect of this will be that a proportionately diminished amount of blood is propelled by the left ventricle into the systemic arteries, though the general injurious effect on the aëration of this fluid is partly compensated for by the increased rapidity of the circulation.

The diagnosis may in some cases be aided by the palpation and auscultation of the heart. If the cadiac impulse be strong and the sounds full when the pulse is small, the over-distension of the right ventricle is probably present. In other instances the impulse is weak and the sounds less distinct than natural, and the enfeebled pulse must then be attributed to impaired cardiac power.[1]

A small pulse may therefore be attributed in many cases to the first-named cause, though enfeebled cardiac innervation has probably in some instances a considerable share in its production. A dichrotous pulse must, however, depend in a greater degree on weakened cardiac power and also on diminished tonicity through impaired innervation of the muscular coat of the arteries. The dichrotous character is often extremely marked about the period of the crisis.[2]

The heart's action is commonly more accelerated in weakly people, and also by coexisting cardiac disease, so that a rapid pulse in Pneumonia may occasionally draw attention to this previously unsuspected complication.[3]

Intermittence of the pulse is sometimes observed in adults; it is much more common in the Pneumonia of old age, independently of any discoverable cardiac disease. In children the pulse, when very rapid, is frequently unequal, but not distinctly intermitting.

Occasionally, as remarked by Dr. Graves, a murmur may be heard over the heart during the height of the disease, and may disappear during the progress of resolution. I have also observed this in one case; the murmur was systolic, and was limited to the apex. In Dr. Graves's case it was heard over a large extent of the affected side. No satisfactory explanation has been offered of this phenomenon. It is difficult to attribute it to polypoid concretions of fibrine, seeing that its dis-

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[1] Walshe, loc. cit.

[2] In a quarter of Grisolle's cases it was, however, below 100.

[3] Thus in a case by Grisolle, in an old man, the pulse was only 58 for twenty-four hours before death.

[4] Out of 184 cases of recovery, Griesinger observed a pulse of 120 to 150 in fifty-four patients above the age of 15. Twenty-seven patients, having a pulse of this frequency, died. Hence nearly one-third of all the patients above 15 under Griesinger's care with a pulse above 120, died. The dangerous significance of this symptom rises to an extreme with advancing age. (Bleuler, loc. cit.)

[5] Ziemssen, loc. cit. 217. Griesinger, loc. cit.

[6] It may seem superfluous to point out the fallacy which may arise from rigidity of the arteries from calcification in advanced life, but this condition requires constantly to be remembered in estimating the "strength" of the pulse in acute disease.

[1] See some excellent critical remarks on this subject in M. Jaccoud's Clin. Méd. The test proposed by M. Jaccoud of "radial recurrence, *i. e.* of the blood finding its way back by the collateral circulation to the radial artery when compressed superiorly, is, I believe, fallacious as an evidence of cardiac power. It may be observed in the weak and dichrotous pulses of advanced phthisis.

[2] For sphygmographic tracings of different varieties of the pulse in Pneumonia, see Appendix A.

[3] Traube, Symptomen der Krankheiten der Respirations-Organe, p. 31.

appearance was not attended by any of the phenomena of embolism.

Evidences of impeded circulation through the lung are also observed in the cyanotic tint of the lips, and less commonly of the fingers (though this is sometimes seen in children), and also in the occasional distension of the jugular veins,[1] which may sometimes pulsate; a similar pulsation has been seen to extend to more distant parts of the venous system.[2]

Epistaxis is sometimes observed. It may be one of the earliest symptoms, or it may appear among the phenomena of the crisis. I have observed it under both sets of circumstances, but not so frequently as has been noticed by some authors.

The condition of the blood will be described under the pathology of the disease.

Digestive System.—This also participates in the general pyrexial state. Thirst is marked, and the appetite is lost. The tongue varies in appearance; sometimes it shows but little alteration, but usually it is coated with a thick creamy fur. In severe cases it tends to become dry and brown, and sordes form on the teeth, and the lips are dry and cracked. Difficulty of deglutition is occasionally observed in old people.[3]

Vomiting has been already stated to be an occasional symptom of the invasion. I find this recorded in eight out of fifty-three cases, most of whom were adults; it is much more common in children, occurring, according to Barthez and Rilliet, in one-half, and according to Ziemssen in three-eighths, of all cases of Pneumonia. It usually ceases after the first or second day, but it may sometimes continue throughout the pyrexial period, and even subsequently.[4] I have known a case in which erysipelas followed Pneumonia, and where vomiting continued during three weeks, and placed the patient's life in considerable danger.

Diarrhœa is also an occasional symptom of the invasion, though not so frequently so as vomiting, with which, however, it may coexist. It rarely continues unchecked throughout the case, except in very young children; it sometimes appears at the period of the crisis or during resolution.[1]

When the gastric symptoms are severe, they have given rise to the description as a special variety of a *gastric* or *bilious form of Pneumonia* (in which, however, the complication with icterus is not included). This variety, which was first described by Stoll, has been the subject of much discussion, and it appears to be a very ill-defined one. A certain number of cases, indeed, occur in which the symptoms of gastro-duodenal, or enteric catarrh, are very distinct. I have met with three or four such; but all gradations can be observed between these and the more ordinary symptoms evincing participation of the digestive tract in the disturbance occasioned by acute pyrexia.

When the condition is a marked one, the complexion is more opaque and earthy than usual. There is greater prostration, and often the headache is more than commonly severe. The tongue is much loaded, nausea is present, or vomiting may persist throughout. The epigastric region is sometimes tender. Constipation is present in some cases, diarrhœa in others, and the latter often appears towards the crisis. Huss found this form of complication most frequent in the summer months.[2] The ordinary state included under this term does not appear to exercise much influence on the mortality of the disease, though cases presenting its more decided features are usually protracted in their course.

Nervous System.—Headache has been already spoken of as an almost constant symptom. It may be very severe, and in such cases it is greatly aggravated by the cough. It usually, however, tends to diminish after the first three or four days. Delirium is also common,[3] but except in patients of dissipated habits, in whom it may assume the characters of delirium tremens, it is rarely violent. It may, however, occasionally appear in so sud-

[1] Grisolle (loc. cit. 257) thinks that this may be occasionally due to pressure by the hepatized lung. It has been observed by him on the affected side in Pneumonia of the apex, and he cites Bouilland as having similarly seen distension of these veins limited to the affected side.

[2] Stokes (loc. cit. 331), quoting from Graves, says that the pulsation was seen in the back of the *hand*. Graves (Clin. Méd. ii. 41) says *head*. (Query a misprint.)

[3] Wunderlich, Abth. iii. B. ii. 363.

[4] Louis (Fièvre Typh. ii. 465) records gastric symptoms, pain or vomiting, in 17 out of 24 cases of Pneumonia which died, and in 23 out of 58 which recovered. In many, these symptoms occurred late in the disease, and it may be questioned whether they were not in part due to antimonial treatment.

[1] Diarrhœa appears to have been very common in Louis's cases, amounting to one-third.

[2] Huss found gastro-intestinal catarrh in 5 per cent., "acute enteritis" in a little more than 1 per cent.

[3] Its frequency is variously estimated by different observers. Louis and Andral stated it at nearly 20 per cent.; but others—Grisolle, Briquet, and Huss—have shown that it does not appear in more than from 8 to 12 per cent. It often, however, occurs to a slight degree at night, and hence may fail to be noticed. Grisolle says that it is more common in males than in females, in the proportion of 21 to 6.

den and severe a form as to be mistaken for acute mania (Grisolle), but most commonly it exists only as a calm wandering, or as an incoherent talkativeness. Huss remarks, contrary to some previous statements on this subject, that delirium is not especially common in Pneumonia of the upper lobes, but that it is most liable to occur when a large tract of lung is affected — either in the single or double form. Under these circumstances, Huss attributes its appearance to cerebral congestion. In the Pneumonia of old people it is particularly common. Huss says that it is most frequent in patients who have been bled. It usually occurs during the height of the disease, and commonly makes its appearance at the time of the evening exacerbation of the pyrexia; it rarely continues more than four or five days. I have known it to make its first appearance in the prostration following the subsidence of the fever.[1] In other cases I have observed it to commence immediately before the crisis, and to continue subsequently. Both of these events are, however, rare—the period succeeding the crisis being more commonly characterized by a subsidence of pre-existing nervous symptoms.

Its appearance in a marked form is a sign of danger, and is indicative either of the prostration of the patient or of the severity of the disease. Grisolle says that three-fourths of his patients presenting this symptom died; but the treatment by bleeding to which they were subjected must be taken into account in estimating this degree of mortality which certainly is not corroborated by my own experience, and though the symptom tends to occur in a large proportion of fatal cases, others may preserve a perfect intelligence to the last moments of life. The delirium, in fatal cases, tends to pass into an imperfect coma. A comatose condition independently of delirium is sometimes observed; it is most common in old people and in children,[2] and in the former there may be a complete prostration of the mental faculties, extending even to a failure in the pronunciation of words.[3]

In drunkards Pneumonia is so constantly associated with nervous disturbance as to have led Huss to describe a special form, the *Pneumonia Potatorum*. The delirium may assume the form of active delirium tremens, with sleeplessness, delusions, and noisy talkativeness, associated with tremors of the limbs and uncertainty of pronunciation—symptoms which may sometimes appear with the first invasion of the disease; or in weakened patients, the subjects of chronic alcoholism, the state may be one of profound prostration and stupor, alternating with a low muttering delirium. In both these forms the general signs of Pneumonia may be indistinct or may be masked by the nervous symptoms, though in the first class the invasion may be sudden and acute, and attended with rigors. Pyrexia is, however, present in both varieties, and is a valuable clue to the mischief in the lungs.

Tremors are not uncommon in weakened patients independently of delirium.

Convulsions are rare and quite exceptional in the adult. They are, however, very common in children,[1] particularly under five years of age, in whom they often attend the invasion of the disease, and they are specially prone to occur if dentition is advancing or difficult. In other cases they occur towards the fatal termination. They are sometimes general and epileptiform; sometimes they appear only in the form of spasm or rigidity of one limb, or of some of the muscles of the face or the eyeballs; occasionally also a stiffening of the muscles of the neck, passing into opisthotonos and a tetanic state, has been observed.[2] I have known a state of partial paralysis to remain subsequently in the limbs affected.[3] When the convulsions are general, and occur in the earlier stages of the disease, they are seldom repeated; but if this is the case, they generally end in a fatal coma. Partial convulsive movements may, however, recur more frequently.

In other cases in children the cerebral disturbance may resemble those seen in the earlier stages of tubercular meningitis, being marked by prostration, headache, delirium, and strabismus — symptoms whose deceptive character is further increased by attendant constipation. Barthez and Rilliet state that these symptoms

[1] The observations of Heintze (Arch. der Heilk. 1868) appear to show that the occurrence of delirium in Pneumonia is not specially connected with excessive elevation of temperature. In the cases observed by him it was much more frequent in cases of Pneumonia of the upper lobe than in that of the lower, in the proportion of 40·17 per cent. of the former to 25·5 of the latter. As regards season, it was more common in the cooler than in the hotter months of the year.

[2] Grisolle relates a case of a young adult who remained perfectly insensible without movement for twenty-six hours, but finally recovered.

[3] Hourmann and Dechambre, loc. cit.

[1] Barthez and Rilliet give to the affection of the nervous system in children the title of "Pneumonie Cérébrale," which they subdivide into "Pneumonie Eclamptique" and "Pneumonie Meningée."

[2] Weber, Path. Anat. des Neugeborenen und Säuglinge, ii. 61. These symptoms were attended by inflammatory changes in the cerebro-spinal arachnoid sac.

[3] This, according to Barthez and Rilliet, is very rare.

are, however, rarely accompanied by the automatic cries, by the sighing respiration, the grinding of the teeth, or by the expression of indifference, and by the rapid changes of color which characterize tubercular meningitis. Ziemssen, however, remarks that all these may be exceptionally observed, and that the coma may be so deep as almost to simulate death.[1] The *collective* appearance of this group of symptoms is fortunately of extreme rarity in the Pneumonia of children.

Disturbances of vision occurring suddenly, with undue sensitiveness to light, a false coloring of surrounding objects, and associated with a dilated condition of the pupils, have been occasionally observed. In these cases ophthalmoscopic examination has revealed undue distension of the veins of the retina; these symptoms disappeared soon after the resolution of the Pneumonia.[2] Deafness was observed in one case by the late Dr. Hillier,[3] and this symptom may at times add to the difficulty of diagnosis from typhoid fever. It does not, however, appear to be a common complication.[4]

The urine is diminished in quantity and increased in specific gravity during the acute period of the disease. The decrease in water may reduce the amount passed to little more than one-half the normal quantity. At the same time the excretion of urea is vastly augmented, amounting sometimes to 85·5 grammes or 1326 grains in twenty-four hours,[5] though usually the amount varies between 35 and 55 grammes (761 and 858 grains).[6] This large amount of excretion necessarily represents destruction of tissue, for it is found at a period when very little food is taken. It usually reaches its height during the pyrexial period, increasing daily in amount until shortly before the crisis, though differences are observed in the period at which the maximum is attained. After the crisis, in spite of an increase of food, the amount may fall within one or two days, to or below the normal standard. In other cases an excess may be passed for some days during the period of resolution,[1] and the normal amount may only be attained on the fourteenth day.

The uric acid is also increased, and probably to a greater proportionate degree than the urea, and generally during the pyrexial period. It may reach at the crisis the amount of 37·7 grains,[2] or even the enormous amount of 103 grains[3] excreted in twenty-four hours. Like the urea, an excess may continue to be passed for some days after the pyrexia has disappeared.[4]

Large deposits of urates tend to occur during the whole period of the disease.

The sulphuric acid also appears to be slightly increased; the phosphoric acid is lessened, and the free acidity is said to be diminished.[5]

The chloride of sodium is markedly diminished, and sometimes its excretion is entirely suppressed during the height of the disease, even when hydrochloric acid or chloride of sodium is taken internally.[6] The hydrochlorate of ammonia continues in some cases to be excreted. The chloride of sodium reappears during resolution, and may for some days after be passed in excessive amounts, showing that it has been retained in the system; and the excess of chlorides may persist in the urine after that of the urea has ceased.[7]

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[1] In a case where this was observed by Ziemssen, the coma ceased with the crisis on the fifth day.

[2] Sichel, Gaz. des Hôpitaux, June, 1861. Seidel, Deutsche Klinik, 1862, p. 269.

[3] Dis. of Children, pp. 40–42.

[4] Griesinger (Bleuler, loc. cit.) met with it five times in 228 cases.

[5] Parkes on Urine, 271. There will be found here a complete list of authors who have investigated this subject.

[6] The estimates of the normal amount of urea vary considerably. The normal daily average for an adult man under ordinary conditions of life may probably be regarded as 500 grains.

[1] Dr. Parkes (loc. cit.) says that he has found 50 or 60 grammes per diem during the period of resolution.

[2] On the tenth day. Zimmermann, Prager Vierteljahresch. 1852, vol. xxxvi. p. 118. The average normal amount appears to be from 6 to 9 grains daily.

[3] Huss, loc. cit. p. 47. This amount must be regarded as very exceptional, as would appear from other analyses given by the same author.

[4] Dr. Parkes considers that this may probably be due to some of the uric acid being retained in the system, owing to its being less easily got rid of than "the diffusible urea." Zimmermann (loc. cit.) for the case quoted above gives the following averages:—

	Grains per diem.
Stadium Incrementi	15
Crisis	37·7
Stadium Decrementi (21 days—average)	13·5
First 7 days of Stadium Decrementi	21·26
Second 7 days "	11·9
Third 7 days "	8·29

[5] For these statements the author is indebted to Dr. Parkes's work. Huss, however, says that both these acids are diminished, at least in the form of their salts.

[6] For the chief investigations on this point see Redtenbacher, Zeitsch. der K. K. Gesellch. der Aerzte zu Wien, 1850, by whom the discovery of this peculiarity was first announced; and Dr. Lionel Beale (Med.-Chir. Trans. xxxv.), by whom this subject was further investigated; also Dr. Parkes, loc. cit.

[7] For remarks on the relative excretion of

Rigler has found that iodide of potassium, when given internally, is also retained in the system during the height of the disease, but that during resolution it is excreted by the urine.[1]

In some very exceptional cases the urea and uric acid appear to be retained in the system during the febrile period, even when there is no albuminuria, and are excreted in large quantities during convalescence, forming a sort of pseudo-critical discharge.[2] Dr. Parkes states that these patients are more liable to diarrhœa during convalescence, and that possibly some elimination of the retained matters may then take place by means of the intestinal mucous membrane. Patients presenting these phenomena of retention are also liable to a more protracted convalescence than those whose urinary excretion is large throughout the disease.

During convalescence the amount of water passed is increased, but that of the urea tends to fall below the normal amount, while the chlorides, as before stated, are commonly increased in quantity.

Albuminuria, usually slight in amount, is a more frequent complication of Pneumonia than of almost any acute disease, except typhus.[3] It is found commonly during the height of the disease, more rarely during convalescence, but it may appear for the first time as late as the twenty-third day. In most cases it must be regarded as one of the general phenomena of the disease, due probably to the kidneys being affected by the same cause which sets up the inflammation in the lungs. Its presence is also indicative to a certain degree of the intensity of the cause, for cases in which it occurs are generally more severe in their character and more fatal in their issue than those in which it is not found.[4] It is very common also in the Pneumonia which appears as part of the general phenomena of some morbid blood poison, as in diphtheria and other conditions, to which further allusion will be made (see Pathology). It is very commonly attended with epithelial casts, and sometimes with blood in the urine.

Bile pigment is not infrequent. The biliary acids are less common. Fibrine and cystine have also been found. The vesical mucus is increased, and the urine tends to decompose early (Dr. Parkes). I have observed retention of urine in one case associated with severe cerebral symptoms.

The skin is pungently hot, but many variations are observed with respect to perspiration. It may appear shortly after the rigor, and subsequently give place to a dry pungent heat, or the skin may be dry until the crisis is attained, or perspirations may continue throughout the entire course of the pyrexial period. Andral thought that sweating was a favorable sign, but I have observed it more than once in fatal cases, and even in those where the temperature has not been markedly elevated. Profuse sweating usually attends and follows the crisis.

Louis remarked that sudamina were rare in Pneumonia.[1] They have been abundant in three of the cases which I have observed; a few also may often be seen when sweating is copious.

Herpes is a very common complication.[2] It most usually appears on the face, and particularly about the lips and angles of the mouth, but it may occur occasionally in other situations.[3] I have seen a tonsillitis having the characteristic appearances of the herpetic form appear on the fifth day of a Pneumonia. It seldom appears before the third or fourth day, but I have known an eruption which from the description I concluded to be herpes precede the Pneumonia by a period of some weeks, the patient remaining out of health in the interval. It may also appear during the crisis, and, in rare instances, during convalescence.[4]

The face, as has been stated, is flushed,

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the chlorides in the urine and sputa, see Appendix B.

[1] Beiträge zur Statistik der Pneumonie; Wien Med. Woch. 1858, No. 48 (Canstatt's Jahresb. 1858).

[2] Parkes, loc. cit.

[3] Parkes, loc. cit. Dr. Parkes quotes the following statistics. He found it in 6 of 13 cases, or in 46·1 per cent.; Finger in 15 of 33, or in 45·4 per cent.; Becquerel in 9 of 21, or in 42·8 per cent.,—collectively representing 30 cases of albuminuria out of 67 cases of Pneumonia, or a ratio of nearly 45 per cent. Metzger, however, did not find it once in 48 cases. In 32 cases which I have analyzed, it was found 10 times, or in rather more than 31 per cent.; Martin Solon and Ziemssen each found albumen only twice in 24 cases.

[4] In seven non-albuminous cases, Dr. Parkes met with only one death; while in five where albumen was present during the height of the disease, three died. Of the ten cases in which I find albuminuria to have been present, five died, but in one of these the disease of the kidneys was probably of old standing. Griesinger (Bleuler) found albumen in the urine in 63 out of 121 cases. Of these, 42 recovered and 21 died. In 22 cases where the amount of albumen was considerable, 8 died.

[1] Fièvre Typh. ii. 111.

[2] Ziemssen observed it in half of the cases of children under his care. Geisler ("Ueber die prognostiche Bedeutung des Herpes bei der Pneumonie," Arch. der Heilk. 1861, ii.) found it in 43·2 per cent. of 421 cases in Wunderlich's wards. In cases under my own care it has been less frequent than this.

[3] Thomas has observed it around the anus. (Arch. der Heilk. viii. 478.)

[4] Six days after the resolution of the fever. (Thomas, ib.)

particularly over the malar bones.[1] The flush may be bright in tint, or it may tend to a cyanotic or violet tinge, particularly in children. With the flush there is, however, usually an opacity or earthy tint of the skin around the eyes and lips. In rare cases the whole surface of the body may be of a bright-red tint, so as even to give rise to the suspicion of the presence of one of the eruptive fevers.[2] The flush tends to disappear with the progress of the disease; occasionally, and particularly in children, and sometimes in old people, there may be an earthy pallor throughout, which may be attended with a bluish tinge of the eyelids. Pallor of the face is most commonly observed during the crisis.

The *temperature*[3] of the body in Pneumonia has only been made the subject of accurate thermometric observations within the past twenty years, though many of the more important facts bearing on this subject had been previously stated by earlier observers.[4] It is, however, to Von Baerensprung,[5] Traube,[6] Zimmermann,[7] Wunderlich,[8] Thomas,[9] and Ziemssen[1] that we owe the revival of observation and most of our accurate knowledge on this subject.[2]

One of the most marked features of Pneumonia,[3] which is almost sufficient to distinguish it from other diseases, is the sudden and considerable rise of temperature which marks its invasion, and which, with some exceptions, is then maintained, with slight morning remissions and evening exacerbations, throughout its course until a crisis occurs. The rise of temperature during the rigor is common to most diseases in which this phenomenon occurs,[4] but its subsequent maintenance at a very high standard during the succeeding first hours and days of the disease is limited to a small class of inflammatory affection.

An instance of this has been already given. I have known a case in which the temperature had reached 105° within a few hours of the first feeling of illness, although the usual rigor was absent; and others may be quoted from different observers who have had an opportunity of witnessing the earliest phenomena of invasion. Thus Zimmermann[5] relates a case where, after prodromata of a week's duration, the temperature within three hours after the initial rigor reached 102° and within twelve hours it attained the height of 104°. Thomas[6] observed a temperature of 105° within nine hours of the invasion; Ziemssen, within four hours after the initial vomiting in a child, found a temperature of 102·5°; within twelve

[1] Unilateral flushing of the cheek on the affected side, and attended with a higher temperature than on the opposite side, was described by Gubler (Union Méd. 1857) as very common in Pneumonia and also in other pulmonary affections, and was attributed by him to the implication of the pulmonary branches of the sympathetic plexus. Other observers, as Barthez and Rilliet, have controverted this opinion, and have shown that the cheek on the side opposite to the affected lung may show an excess of hyperæmia. Jaccoud (loc. cit. p. 28) observed in an attack of Pneumonia in his own person, that a local flush, attended by a disagreeable sensation of heat in the cheek on the side opposite to the affected lung, preceded the Pneumonia for twenty-four hours, during which time, with this exception, he felt in perfect health. The Pneumonia then commenced suddenly with rigors. He states that he has met with five similar instances.

[2] Barthez and Rilliet (loc. cit. i. 522).

[3] In all ensuing statements on this subject, the temperatures quoted will be those of Fahrenheit's scale. Quotations from other observers have been reduced to this standard.

[4] Thus Donné (Arch. Gén. de Méd. 1837) observed a temperature of 103°, and Roger, in a more extended series of researches (Arch. Gén. de Méd., Sér. iv. vol. vi.) stated that Pneumonia had a higher temperature than almost any other disease, and that in the majority of cases this exceeded 104° Fahr.

[5] Müller's Archiv, 1851–2.

[6] Annalen der Charité, i.; Ueber krisen und kritischen Tagen.

[7] Various writings in "Med. Zeit. des Vereins für Heilkunde im Preussen," specially in "Prager Vierteljahresch." 1852.

[8] Various papers in "Archiv für physiol. Heilkunde;" "Das Verhaltniss der Eigenwärme im Krankheiten."

[9] Archiv für Heilkunde, 1864–5.

[1] Pleuritis und Pneumonie im Kindesalter.

[2] Among English authors the most valuable observations are those by Dr. Parkes, Med. Times, 1866; by the late Dr. Waters, St. Barth. Hosp. Rep. vol. ii.; Dr. Compton, Dublin Quarterly Journal, xlii.; Dr. Grimshaw, ib. 1866; and Dr. Maclagan, Edinb. Med. Journal, 1869.

[3] Grisolle (loc. cit. 163) says that in some cases the course of Pneumonia is apyrexial throughout, though the physical signs and rusty sputa are present. Grisolle's statement is made apparently irrespectively of thermometric observations. Wunderlich, however, repeats the statement (Eigenwärme im Krankheiten, p. 337). Such cases must, however, be excessively rare, and require data as to the day of the disease upon which they came under observation. The majority of cases in hospital practice are rarely admitted before the third day, and it must be remembered that even at this early period the temperature may in some cases fall from a pyrexial height to the normal standard. I have never seen a case of Pneumonia unattended by pyrexia free from this suspicion.

[4] This was observed by De Haen, in Intermittent Fevers, Rat. Medendi, Ed. 1761, i. 117.

[5] Prager Vierteljahresch. 1852, xxxvi. p. 97.

[6] Archiv für Heilk. 1864.

hours this had reached 104·6°, and within twenty-four hours the temperature was maintained at 103·5°.

The highest temperatures are most commonly observed on the second or third day of the disease, but exceptions to this rule are not infrequent. In some instances the maximum temperature, preceded by a very sudden rise of from one to two degrees Fahr. above the previous average, may occur immediately before the crisis. The highest recorded temperatures in cases ending favorably are 106·7° in the rectum[1] (Ziemssen), and 107° (Kocher), but they rarely exceed 105° or 106°. In fatal cases, however, there may be a considerable rise before death, as to 106·9°, 108·9° (seventh day), or even to 109·4° (fourteenth day—Thomas[2]); a slight post-mortem rise is also occasionally observed. In the fatal cases which have come under my own observation this ante-mortem rise has not been noticed, but in most of these the temperature had been only moderate throughout. The higher temperatures, according to my own experience, are however, rather the exceptions than the rule.[3] In the majority of cases it has seldom exceeded 104°, and a large number run their course without the temperature of 103° being attained. As a general rule the milder cases are those in which the pyrexia is least, but cases may end fatally in which the temperature has barely exceeded 102°. In old people especially, in whom Pneumonia is comparatively the most fatal, the temperature is very commonly lower than in adults.

After the invasion the pyrexia generally runs a certain definite course, with a series of regular daily exacerbations and remission, which commonly occur respectively in the evenings and mornings, representing in this respect, though with some irregularity, an exaggeration of the normal diurnal variations,[4] and which according to their relative extent have given rise to various classifications.[1] Usually the daily course is that the morning temperature from 6 to 9 A. M. is the lowest, but it seldom falls more than 1° or 1·8° Fahr. below that observed in the evening, and the temperature at these periods of remission never, or only in the most exceptional cases, reaches the normal standard. In the forenoon or early in the afternoon the fever again increases, commonly reaching its maximum intensity early in the evening, or sometimes even at mid-day.[2] From this point the temperature falls towards midnight, when a second slight exacerbation occasionally occurs, which does not however reach the same height as that of the afternoon.[3] Subsequently to this the temperature continues to sink during the night until an early hour the following morning, from which a gradual rise takes place, culminating in the exacerbation of the succeeding afternoon. In very rare cases the rise of temperature takes place early in the morning, when the highest temperature is observed, under which circumstances the corresponding remission is noticed at mid-day, or a continuous fall takes place until the evening.

During the course of the acute disease the morning remissions and evening exacerbations maintain in typical cases a very uniform standard of temperature until the period of the crisis is attained, unless fresh extensions of the pneumonic process occur, when a sudden increase of the temperature may be usually observed. In the earlier periods of the disease or during the *stadium incrementi* a more

[1] This was observed on the sixth day in a child who at the time was sweating profusely.

[2] This terminal elevation of temperature is sometimes preceded by a marked remission. It is sometimes gradual, extending over a period of from twelve to twenty-four hours, but it may take place very rapidly, *i. e.*, within six hours. The temperature has been known to rise on the supervention of severe brain symptoms from 101·7° to 108·7°, or 7 degrees Fahr.

[3] Out of twenty-seven cases whose temperature has been carefully taken throughout, in one only was a temperature of 105·8° attained on the sixth day, the crisis occurring on the eighth day. Griesinger (Arch. der Heilkunde, i.), out of seventy-two cases, only observed the temperature higher than 103° Fahr. in nineteen.

[4] See Von Baerensprung, Müller's Archiv, 1851, pp. 160 *et seq.*; Ib. 1852, p. 251.

[1] Those proposed by Thomas and Wunderlich are as follows:—

(*a*) A subcontinuous course, with daily variations of from $\frac{2}{5}$° to $\frac{3}{5}$° Fahr.

(*b*) Subremittent, with daily variations of $\frac{3}{5}$° to 1·5° Fahr.

(*c*) Remittent, with variations from 1·5° to 2·5° Fahr.

(*d*) Intermittent, a very rare form, with complete apyrexial periods in the daily course.

There is a form of Pneumonia described as accompanying intermittent fever where there also appear to be complete apyrexial periods corresponding to the type of the fever.

[2] This occasional irregularity renders a mid-day observation on the temperature necessary in all cases when scientific accuracy is required. In fact, unless frequent observations are made, the period of the *maximum* elevation of temperature may escape observation.

[3] This second exacerbation may sometimes be anticipated; that is, when the afternoon exacerbation occurs early, a rapid fall may take place until early in the evening, and the second rise may take place early in the evening instead of at midnight.

marked remission may occasionally occur, and may even be repeated more than once. This is followed in some cases by an intenser exacerbation, but in others the temperature of the succeeding rise falls below the average standard of the case. When an exacerbation of the fever follows this remission, it is also frequently attended by an extension of the pneumonic process or by a secondary inflammation of some other organ.[1]

The pyrexia tends to subside abruptly by crisis or gradually by lysis, the resolution by crisis being however the most usual form, and the rapidity with which this takes place in typical cases is again almost peculiar to this disease. Thermometric phenomena of the crisis may commence either at the period of the morning remission or of the post-meridial exacerbation. If at the former, the temperature, which on the preceding evening may have maintained its previous height, is found on the following morning to have fallen to the normal or nearly to the normal standard, and the succeeding exacerbation on the following evening is less by 1 or 2 degrees Fahr. than those previously observed. From this period a gradual fall of temperature ensues, and within forty-eight hours from the commencement of the crisis it has usually reached the limit of health, or it may have fallen below it, and after this no further elevation ensues.[2] The extent of the fall of temperature is sometimes very remarkable when the fever has been severe, amounting even to 9·7° Fahr. in sixty hours.[3] This is sometimes intensified by the fact that when the fever has been high and the patient is weak, and in children particularly, the temperature may sink during the critical defervescence to 1°, 2°, or 2·5° below the normal, and may continue at this low point for forty-eight or seventy-two hours. I have observed in a child a temperature of 96·5° (axilla) with a cold skin and profuse perspiration maintained in spite of artificial warmth for forty-eight hours. Such cases in children, however, do not commonly end unfavorably. Variations in the phenomena of the crisis are, however, not uncommon. Sometimes immediately before it occurs the temperature may rise to a higher point than those previously observed. In other cases, for twenty-four or forty-eight hours previously, both the morning and the evening temperature may show a lower range before the final rapid decline takes place. In some, again, the crisis is marked rather by successive falls of temperature during the periods of remission, those of exacerbation maintaining during twenty-four or forty-eight hours the same height as before, but finally participating in the decline—a course which may be regarded as presenting simply a modification of the ordinary rhythmical progress of the disease.

Sometimes, after the crisis has distinctly appeared, the fall of the temperature is suddenly checked, and a temporary exacerbation may occur, attended by a cessation of the critical perspiration and by a return of the restlessness and of the other febrile symptoms.

For some days after the disappearance of the fever there is also a tendency to temporary trivial exacerbations from slight causes, such as a meal or a slight exertion; but these, within certain limits, do not interrupt the progress of convalescence.[1]

The course of the fever is, however, subject to other variations, which are due commonly to the progressive invasions of other portions of the lung or of the opposite lung, and the crisis may be disturbed by a relapse.

The former may occur while the fever is still present. They are often marked by an increased intensity of the succeeding exacerbations, which may give the pyrexia a remittent type.

Relapses most commonly occur within the first three or four days succeeding to the crisis.[2] I have, however, known a relapse take place as late as the sixteenth day, when the temperature had been natural since the eighth day.[3] They are marked by a sudden rise of temperature from the normal or subnormal standard previously attained. The duration of the pyrexia in these relapses is, however, commonly shorter than that of the primary attack, usually terminating within three or four days; but cases are recorded where even a third relapse has ensued.[4]

[1] Kocher, Behandlung der croupösen Pneumonie mit Veratrum Preparaten; Würzburg, 1866.

[2] Griesinger (Bleuler, loc. cit.) found this rapid fall of temperature in 112 out of 146 cases. The normal temperature was reached within twelve hours in 37 cases; within twenty-four hours in 32 cases; within thirty-six hours in 43 cases. In 41 cases the fall of temperature was more gradual.

[3] Ziemssen, loc. cit. 211.

[1] Monthus (loc. cit.) remarks that the apyrexial period following an attack of double Pneumonia is marked by a rather higher temperature, and more readily shows slight subsequent exacerbations than when the Pneumonia has been unilateral.

[2] Monthus, loc. cit. 206.

[3] The duration of the pyrexia in the second attack was only two days.

[4] See a case by Ziemssen, p. 186, of Pneumonia of the upper lobe. The maximum temperature of the original attack was 102° Fahr. On the ninth day the temperature fell to normal. On the tenth day there was a return of the fever with invasion of the

It is therefore very important to maintain thermometric observations for some days after the normal temperature has been attained—the more so as relapses with invasion of other portions of the lung are seldom attended with a return of the initial rigors, and the increased temperature may give the first indication of the extension of the disease.

In other cases the temperature falls by a gradual lysis, which, in some cases that I have observed, has only reached the normal standard on the twelfth or fourteenth day.

In a third series, again, the crisis is incomplete, and the course of the pyrexia is protracted. There is very often noticed on one of the days intervening from the seventh to the tenth inclusive, a marked fall both of the morning and evening temperature; but this does not reach the normal, and on the succeeding days pyrexia persists, though not usually at its previously high standard. A slow defervescence then ensues which may be protracted over two or three weeks, and is attended with a somewhat irregular course of the temperature—that in the morning being often nearly normal, while in the evening it may be on some days 100° and on others 102° Fahr., occasionally rising to 103° or 104°, and on the succeeding evening it may again only be 100°. These cases are generally attended with a protracted disappearance of the physical signs—the consolidation and bronchial breathing with fine râles lasting, together with the pyrexial state, for three weeks or a month, but gradually disappearing and ending in perfect recovery.[1]

The circumstances determining a more protracted course of the pyrexia are not always clearly discoverable. Cases where bronchitis passes into Pneumonia, and which belong rather to the clinical category of broncho-pneumonias, often evince this tendency; but I do not think that this peculiarity is sufficient under all circumstances to remove a case in which it is observed from the category of the primary form, as it is occasionally seen when the mode of invasion and the earlier course are typical of this condition; and it must, therefore, I think, be regarded as a somewhat exceptional variation.

The cases in which I have observed this course are most commonly those which are accompanied by an extreme degree of gastric catarrh, or when the Pneumonia attacks persons of weakly constitutions, or those of previously dissipated habits. In some cases also where bleeding has been practised the recovery has been slow.[2] A very widespread opinion now exists that venesection tends to retard convalescence. The presence of tubercle or the tubercular diathesis appears also to protract the course of acute Pneumonia. Such patients may in many instances recover entirely from the inflammatory consolidation, but in others the resolution is imperfect, and the disease, although in rare instances,

middle lobe, and with a temperature of 104·9°. On the fourteenth day a second remission of the pyrexia took place, followed on the fifteenth by a return of the fever (temperature 102·7), and with invasion of the lower lobe. The final crisis and permanent recovery occurred on the eighteenth day. In another case by the same author, with Pneumonia of the left lower lobe, the crisis occurred on the fifth day. On the sixth there was a severe return of the fever, followed on the eighth day by the physical signs of consolidation of the right upper lobe, while the resolution of the lung first affected continued unimpeded. The final fall of temperature began on the eleventh day and continued through the twelfth, when convalescence was re-established. Grisolle says that relapses occurred with him in the proportion of once in 28 cases. Briquet met with 16 instances in 92 cases. Grisolle quotes a case in which three consecutive relapses took place, the last being on the twenty-seventh day. Commonly the course of the relapse is shorter than that of the first attack, rarely lasting more than three or four days. In the case, however, quoted, each attack lasted nine days, and the last was very severe. (There is some confusion in Grisolle's statement with respect to the duration of the relapses.)

[1] In one case under my own observation, a boy aged 15, previously in good health, got chilled. Pneumonia supervened with rigor on the following morning; admission on the third day of disease with well-developed Pneumonia of the lower two-thirds of the right lung, and severe gastric catarrh. Temperature on fourth day, 105°; on the eighth day it fell to 99°; on the ninth and tenth days it was 99° and 98·4°; on the eleventh day it rose to 100° without any discoverable increase of the Pneumonia. It then fluctuated between 100° and 102°, reaching to 103° on the fourteenth day, and only fell gradually to the normal on the thirty fifth day. The physical signs only completely disappeared by the sixtieth day.

[2] This was the case in that by Zimmermann before quoted. The case was peculiar in its course. The temperature on the first day was 104° in spite of VS to 2 lbs. and repeated on the second day to 14 oz. Up to the third day there were only the physical signs of congestion, but the respiration was slightly bronchial in one place. On the third day there was a distinct remission in the morning (99·8°), but followed by an evening exacerbation to 105·8°. On the fourth day, rusty sputa, dulness, and bronchial breathing appeared in the lung. A second imperfect crisis occurred on the ninth day, with a subsequent elevation of temperature on the tenth, reaching 103° on the seventeenth day, and with irregular intermissions maintaining a temperature of 100° to the twenty-fifth day.

passes into the condition of a chronic tubercular Pneumonia.[1]

Pneumonia of the apex is said by Ziemssen and Bleuler to tend to maintain a high temperature during a longer period than that of the base,[2] and that in non-tubercular patients, though the protracted course may give rise to the suspicion of this complication. This, however, is not invariably the case, for I have known the crisis to occur in a well-marked case of Pneumonia of the apex as early as the fourth day. Ziemssen thinks that such cases may be distinguished from tubercular Pneumonia by the constantly maintained high temperature; but my own experience would show that this sign cannot be relied on, since I have observed that the elevation of temperature in cases of protracted simple Pneumonia is not always continuous, while it may be so in some cases of tuberculosis and of tubercular Pneumonia.

The coexistence of pleuritic effusion certainly tends in some cases to render the thermometrical crisis incomplete and to protract the period of defervescence. It also, as might be expected, delays the disappearance of the physical signs; the complication with pericarditis has a similar influence in the crisis. Ziemssen remarks that neither pleurisy nor pericarditis, when occurring in the course of Pneumonia, has any necessary tendency to raise the temperature above the standard of the individual case.

The period of the crisis has been a subject of considerable discussion and of careful thermometric observation. The recognition of this tendency has been common to many observers, and it was pointed out by Laennec. Andral[3] thought that the seventh, fourteenth, or twenty-first days were the most common periods of its occurrence—supporting the doctrine of special critical days advanced by Hippocrates. Grisolle disputed this opinion. Traube (Ueber krisen und kritischen Tagen) has again revived it, and has asserted that in acute diseases, and especially in Pneumonia, the crisis usually occurs on the third, fifth, seventh, ninth, or eleventh days, and that therefore it has a preponderating tendency to appear on uneven days. This, however, has been denied by different observers, whose observations show that the crisis is by no means so constant on the uneven days as Traube believed, but that in a large proportion of cases, amounting respectively to 20 and 25 per cent. and collectively to 46 per cent. of the whole number, it tends to occur on the fifth and seventh days.[1]

The cases of which I possess sufficiently accurate thermometric observations give very similar results, though in smaller numbers. Out of twenty-seven cases ending favorably, a distinct thermometric crisis occurred in eighteen, and two more were admitted on the fifth and eighth days respectively with the physical signs of Pneumonia, but with a normal temperature, which was maintained subsequently. These, therefore, may justly, I think, be added to the above, making the proportion of cases terminating by crisis, as compared with those not thus ending, as twenty to twenty-seven.

The following were the days[2] in which a crisis was observed:—On the fourth day, one case; on the sixth, one; on the seventh, six; on the eighth, two; on the ninth, four; on the tenth, two; and on the eleventh day, two cases. The period of complete defervescence varied from twelve hours (four cases) to seventy-two hours (one case). In the remainder it

[1] This course is, however, very rare in the acute primary disease. Most of the forms of tubercular Pneumonia run the course of catarrhal or broncho-Pneumonia.

[2] See a case by Ziemssen (loc. cit. pp. 180–2) of Pneumonia of upper lobe, in a child aged nine months. The pyrexia lasted thirty-one days, and the physical signs only disappeared three weeks after the subsidence of the fever. Bleuler (loc. cit. p. 19) states that of the cases observed by him and Griesinger when the inflammation affected the apex of the right lung, in one only did the fall of temperature occur before the sixth day, and in three-fourths of these cases it took place after this date, while in more than half the cases of Pneumonia of the base defervescence ensued from the third to the fifth day.

[3] Clin. Méd. iii. 516.

[1] The days of crisis observed by Wunderlich (Spec. Path. Therap. Abth. iii. B. ii. p. 334), Ziemssen (loc. cit. 174), Thomas (Arch. der Heilk.), and Bleuler (loc. cit.) may be best expressed in a tabular form:—

Crisis, Number of Cases.

Day of disease.	Wunderlich.	Ziemssen.	Thomas	Bleuler	Total.
1st	0	0	0	0	0
2d	0	0	2	0	2
3d	10	9	6	6	31
4th	11	3	6	13	33
5th	14	31	11	22	78
6th	14	5	5	26	50
7th	19	35	10	32	96
8th	4	4	4	24	36
9th	3	9	0	12	24
10th	0	0	2	6	8
11th	0	8	0	1	9
12th	0	0	0	3	3
13th	0	3	0	1	4
14th	0	0	0	0	0
	75	107	46	146	374

[2] I have reckoned the day of invasion as the first, the next day as the second day.

varied from twenty-four to forty-eight hours.

Two other cases terminated by gradual lysis, one on the twelfth and another on the fourteenth day, the temperature gradually falling to the normal.

In five others the duration was protracted without complications, which would account for the persistence of the pyrexia, except in one instance, where there was considerable pleuritic effusion. In this case an imperfect crisis took place on the tenth day, but the temperature remained elevated until the forty-sixth day. Of the remainder three recovered perfectly, though the pyrexia only ceased on the twenty-fourth, thirtieth,[1] and thirty-fifth days respectively.[2] In the fourth there was, however, a suspicion of tuberculosis. The Pneumonia, which had invaded the whole right lung, and which was complicated with pleurisy, resolved imperfectly, and occasional pyrexia remained until the eighty-first day.

As far as I have observed, I do not think that cases where the temperature is much elevated, *i. e.* above 104°, necessarily have a longer duration than those in which the pyrexia is less marked. The pyrexia in the former may end rapidly by an early crisis, and in the latter its disappearance may sometimes be considerably protracted. My own observations would also tend to confirm Thomas's opinion that the extent of lung affected does not necessarily delay the appearance of the defervescence, though cases supporting the contrary opinion, which has been advanced by Ziemssen, may sometimes be met with.

Together with the disappearance of the fever, the aspect of the patient markedly changes. The flush disappears, and profuse sweating is almost constantly observed.[3] The face may be pallid and sunken, and, as before stated, the general condition may be one of such intense collapse as to lead to immediate fears of a fatal issue, which indeed sometimes occurs at this period.[4] The pulse becomes small and often dicrotous, and generally falls in frequency. It seldom, however, attains the normal standard, and is liable to irregular exacerbations for some days later, quite irrespective of any corresponding variations of temperature. Children particularly may be for hours partially unconscious and almost incapable of being roused, with a cold skin bathed in colliquative perspiration.[1] A catarrhal flow from the nose is sometimes also observed in children at this period simultaneously with the perspiration.

The respiration at the same time falls in frequency. The pain in the side, if this has persisted up to the period of the crisis, disappears or is much relieved. The cough becomes looser; the expectoration loses its tenacity, and the rusty character diminishes, though it may not finally disappear until some days later. In its further course and during the resolution of the Pneumonia the sputa gradually assume a bronchitic character. The most marked appearance is, however, that of black pigment, which takes the place of the rusty tinge of blood, and the early appearance of which is a favorable sign. The amount of this pigment in some cases, when the resolution is retarded, is sometimes very considerable: I have seen the sputa during many days almost black from its presence.

Other phenomena are occasionally observed, some of which have been regarded as truly *critical*, that is to say, as in part conducing to the fall of temperature; others, however, must be looked upon as accidental, or as a result of the subsidence of the fever. Among the former, whose influence in really producing a fall of temperature must be considered doubtful, are hemorrhage and diarrhœa. Hemorrhage is occasionally observed in the form of epistaxis, more rarely as hæmaturia, and occasionally it proceeds from the bowels.[2] Diarrhœa is more common,[3] but it must be remembered that the crisis may take place without any of these events, and their appearance is as a whole decidedly exceptional, the only constant critical discharge (with the exception of the changes in the amount of the urinary secretions) being that from the skin.

Erysipelas is mentioned as an occasional critical phenomenon.[4]

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[1] This case was a man of dissolute habits. An imperfect crisis took place on the ninth day. The general symptoms were very severe, with profuse puriform sputa after the second week, giving rise to strong suspicions of gray hepatization.

[2] This case has been already alluded to (see note, p. 179). There was in this case an imperfect crisis.

[3] Herpes also may appear as a critical phenomenon at this time.

[4] I have seen two cases of this nature.

[1] An excellent and life-like description of this condition is given by Ziemssen, loc. cit. 167.

[2] I have only seen one case of this nature.

[3] Huss, p. 53, says that diarrhœa most commonly occurs on the seventh day in cases of Pneumonia characterized by severe gastric disturbances, but that the convalescence of such cases is usually protracted.

[4] Grisolle quotes from Serres a case of a patient who had several attacks of Pneumonia, each terminating in an attack of erysipelas. I have only seen one such case. The erysipelas appeared three days after complete defervescence, and the resolution of the Pneumonia and the subsequent recovery of the patient were greatly protracted.

When the nervous system has been profoundly implicated during the pyrexial period, the symptoms of such disturbance also commonly disappear during the crisis. Delirium or extreme restlessness usually pass, particularly in children, into quiet sleep. In adult persons, and especially in those of dissipated habits, this may not be the case; I have seen symptoms closely resembling delirium tremens persist during forty-eight hours after the normal temperature has been reached and maintained.

The physical signs of the disease may begin to improve coincidently with the disappearance of the fever.[1] The commencement of the resolution, however, is more commonly observed after the first twenty-four or forty-eight hours of the apyrexial period. In some cases it is so rapid that all the physical signs of the disease may have totally disappeared in twenty-four hours from the first appearance of improvement.[2] I have seen this in one case where the whole lower lobe has been implicated, and it may occur without any marked increase of the expectoration, or even when this has been scanty and quite insignificant in quantity. Indeed it may be said that, generally speaking, the proportion of exudation removed by expectoration must be small in comparison with the whole amount present in the lungs. Commonly, however, the course of resolution is more protracted. Grisolle states that all the physical signs had only completely disappeared in 37 out of 103 cases who left the hospital between the twentieth and the fifty-fifth days. Dr. Stokes gives the following results of 24 cases, dating from the commencement of resolution. In nine the physical signs had disappeared at the end of a week; in nine more at the end of fourteen days; in five at the end of three weeks; and in one they lasted a month. In 26 cases of which I have notes of the total[3] disappearance of the physical signs, their duration after defervescence was as follows:—In one case, two days, in three, three days; in one, four days; in one, five days; in one, six days; in three, seven days; in one, nine days; in nine, from ten to fifteen days; in five, from twenty to twenty-five days; in one, from twenty to thirty. Two other patients left the hospital with physical signs still remaining on the twenty-fourth and eightieth days.[1] In some of the cases of longer duration the Pneumonia was complicated with pleurisy, and when much effusion has been present some dulness at the base may remain almost indefinitely, as I have seen in one or two cases not included in this list. The coexistence of tubercles may also indefinitely protract the resolution. Patients whose health has been previously bad are also liable to a retarded resolution; but this is not always observed. The same tendency has been noticed in cases where the defervescence is not marked by a crisis, or only by an imperfect one.[2]

Dr. Stokes has observed that retraction of the chest walls may follow an attack of Pneumonia. This has been disputed by Grisolle and Woillez; but Dr. Walshe has seen it take place when the Pneumonia had been unattended by liquid effusion into the pleura. I have also observed it in one of the cases of protracted Pneumonia before mentioned.[3]

The recovery of strength and of flesh is generally very rapid. The appetite often returns almost with the cessation of the pyrexia. Wachsmuth observed in a patient whose loss of weight in four days

[1] Grisolle states that the improvement in the physical signs may precede the disappearance of the pyrexia. Grisolle's statement appears, however, to be made independently of thermometric observations. I have never seen a case where this occurred before a marked form of temperature, and it must be remembered that Grisolle regards a rapid pulse as one of the phenomena of the fever. It has been already stated that the pulse may remain rapid after the fever has subsided.

[2] Ziemssen gives a case of a child where the physical signs had disappeared before the end of the eighth day of the disease.

[3] This includes the final disappearance of all râles as well as dulness and bronchial breathing. Crepitation or fine moist râles may, as has been before stated, often persist for days, or even weeks, after all other signs have disappeared. Probably the weakened resistance of the vascular coats leaves, during a lengthened period, a tendency to a certain degree of œdema, particularly in the lower portions of the lung.

[1] Bleuler (loc. cit.) gives the following periods of resolution in 150 cases: One day, 5 cases; two days, 2 cases; three days, 4 cases; four days, 21 cases; five days, 21 cases; six days, 30 cases; seven days, 13 cases; eight days, 11 cases; nine days, 5 cases; ten to fifteen days, 18 cases; fifteen to twenty days, 6 cases; more than twenty days, 7 cases, among which were included 3 cases of Pneumonia on the right upper lobe.

[2] Ziemssen remarks that in cases where the crisis is early, resolution may be short, but my own experience has not confirmed this.

[3] In a boy in whom the pyrexia and physical signs lasted together sixty days (see note, p. 179), there was observed when he left the hospital some flattening inferiorly of the right (the affected) side. One month later, when he presented himself for examination, the measurements were: At nipple—right side, 12 inches; left, 12 inches. At sixth rib—right side, 11 inches; left, 11½ inches. There was also some procidentia of the shoulder on the right side. The amount of effusion present here was throughout extremely small, but some dulness still remained at the right base, attended with weak breathing.

amounted to a daily average of 24 oz. in the twenty-four hours, and in whom the loss of weight continued for forty-eight hours after the crisis, that in the succeeding four days nearly 2 lbs. were regained.[1] I have repeatedly observed that from 7 to 14 lbs. may be gained in weight during the first few weeks of convalescence.

An attack of acute Pneumonia is seldom succeeded by secondary diseases, except in patients liable to tubercle. Ziemssen has observed in children that œdema of the lower extremities may be caused by a pure hydræmia, independently of albuminuria, which, however, is sometimes present to a slight degree. Dr. Walshe has observed the same phenomenon associated with coagulation in the veins.

Gubler[2] and Macario[3] have each observed instances of general paralysis following Pneumonia, but these cases are fortunately rare.

The *termination* of Pneumonia is not, however, always favorable. It may end fatally or it may give rise to local abscess or to gangrene of the lung, or finally it may pass into a chronic state.

In some cases, which may prove fatal during the acute stage, the pyrexia may persist to the last, and may, as before stated, increase rapidly towards the close of life. In others, however, no elevation of temperature occurs, and it may even sink to normal before the fatal termination: I have seen in one case, in a patient aged 62, death occur after the crisis had taken place forty-eight hours previously, and in whom during the first portion of this period the symptoms might on the whole have been considered favorable.

Most commonly death ensues during the acute period of the disease, when it is usually preceded either by intense prostration or by extreme dyspnœa. The pulse becomes small and extremely rapid and dicrotous, and the respiration is commonly greatly accelerated. Expectoration becomes difficult, or ceases, while large coarse metallic râles are heard in the trachea and larger bronchi, and fine and medium-sized râles indicative of œdema of the lung, extend over the non-consolidated portions. The face becomes livid, the extremities cold, and the skin is often bathed in profuse perspiration, which is colliquative when the temperature is low. A semi-comatose state supervenes towards the last, but in some instances intelligence is preserved to within a few minutes of the fatal issue. In children, coma or convulsions are very common. In old people death may often take place suddenly and unexpectedly.[1]

Sometimes, particularly in children as described by Ziemssen, death may occur at a later stage. The fever does not maintain the high standard of the earlier periods of the disease, but persists together with the physical signs. The pulse remains accelerated, the skin becomes intensely pallid; emaciation, reducing the patient to the extremest degrees of marasmus, progresses rapidly; and the patient dies in the third or fourth week. In other cases there is observed an incomplete remission, followed by a return of the fever, and the patient gradually sinks in the course of the second week.

Some cases, however, presenting these characters lapse into a more chronic stage; the fever and physical signs may persist during many weeks, but the former may subside, while the lung remains permanently consolidated with signs of dilatation of the bronchi.[2]

No special condition of the lung is necessarily associated with a fatal termination in the earlier periods of the disease, but the red or gray hepatization, or even diffuse suppuration, may be found in different cases under circumstances which are otherwise apparently similar.

The termination in *abscess* is very rare. Huss says it only occurs once in fifty or sixty cases, and usually only in patients of bad constitution. According to this author, it is most commonly met with in males over forty years of age, and he states that it was more common when bleeding formed part of the treatment than it has proved since this was abandoned by him. The period of this termination, as determined by profuse purulent expectoration, has varied, according to Grisolle, between the fifteenth and twenty-eighth days. Profuse expectoration may continue for three months subsequently. The site of the abscess is usually at the apex; one case, however, has been recorded by Dr. Stokes, where a cavity in the midst of pneumonic tissue was found at the base of the lung. The signs of this condition have been already described.[3] Cases in which it occurs usually run a protracted course, though death ordinarily, according to Grisolle, takes place before the thirteenth week. Pyrexia persists, and the expectoration, which is at times intermittent, consists of large quantities of puriform matter. The pyrexia tends to assume the character of hectic

[1] Zur Lehre von Fieber, Arch. der Heilk. 1865, p. 236. In this case the temperature had been very high, 106·10, and the defervescence was gradual after the crisis.

[2] Arch. Gén. 1860–1.

[3] Gaz. Méd., Par. 1858. Huxham says: "I have seen in some cases (though few indeed) a complete paraplegia." (On Fevers, p. 183.)

[1] Cruveilhier, Path. Anat., Liv. xxix.

[2] These cases will be again considered under the head of Chronic Pneumonia.

[3] See *ante*, p. 170.

fever, but from the rarity of the disease thermometric observations are wanting. Emaciation progresses as long as the fever remains, and many cases end fatally, sometimes with the signs of pyohæmia, in other instances by rupture of the abscess into the pleural cavity, and occasionally by sudden suffocation resulting from the filling of the bronchi with pus. Others, however, progress more or less completely to recovery; in these the abscess cavity may either cicatrize, or it may remain patent but completely quiescent, and revealed only by physical signs more or less distinctly indicating its existence.[1]

The termination in *gangrene* is almost equally rare with that in abscess, and Grisolle has even doubted whether it is a cause or a consequence of the latter. Some well-authenticated instances are, however, recorded, and it appears that an epidemic constitution may at times predispose to its occurrence.[2] It commonly appears late in the disease; but it has been seen as early as the fifth day (Huss). In fifty-three cases of which I possess observations, I have found two instances of gangrene,[3] and in both these it was irregularly diffused through scattered spots of pneumonic infiltration. Its site, according to Huss's observations, is most commonly in the lower lobe, and it has almost invariably occurred in exhausted constitutions. Gangrene is much more common in tubercular Pneumonia. Its physical signs have been already described. In addition to these its advent is usually marked by a sudden and intense prostration of strength, with a rapid weak pulse and sunken countenance. The characteristic sputa are, however, the only positive signs, when developed suddenly in the course of a primary Pneumonia. It appears to be almost invariably fatal.

COMPLICATIONS OF PNEUMONIA.—Some of these affecting the kidneys and nervous system have been already described. Others, however, deserve mention.[4]

Laryngitis, though not mentioned by Huss, is an occasional complication. Grisolle quotes Serres as having collected the histories of ten cases, and Dr. Walshe says that œdema of the glottis may be one of the causes of a fatal termination.

Bronchitis is a more frequent complication. Grisolle says that it has occurred in one-fourth of his cases, that it is seven times more common in males than in females, and that it is most frequent in the winter months. It affects both lungs, though it sometimes appears in excess on the affected side. Its intensity varies greatly in individual cases. Its presence, when general, however, increases the dyspnœa and the lividity of the face. It also renders the sputa more abundant and the cough more frequent. It is seen from Huss's tables that it tends (at least when severe) to increase the mortality of the primary disease.[1]

Pleurisy is also very common. There are, indeed, very few cases of Pneumonia reaching the surface of the lung in which the visceral pleura is not implicated. Effusion, according to Grisolle, occurs in about 15 per cent. The amount of fluid is commonly in inverse ratio to the extent of lung implicated. Its signs are naturally, almost invariably, found at the base, whatever the site of the Pneumonia. Its influence on the pyrexia and on the progress

nia. The following table from Huss gives a relative estimate of the frequency of other complications, and of their influence on the mortality. This table appears to include cases of both catarrhal and acute primary Pneumonia; but while some chronic diseases are mentioned, the omission of others, as cancer, is remarkable. Huss, however, does not treat of the secondary Pneumonias complicating other diseases.

	Recoveries.	Deaths.	Total.	Per ct. of deaths.
Pleuritis	92	12	104	11·53
Bronchitis capillaris, acute	120	20	140	14 28
Bronchitis chronica	36	6	42	14·28
Emphysema pulmonum	20	6	26	23·07
Tuberculosis pulmonum	24	12	36	33·3
Pericarditis	10	12	22	54·54
Endocarditis	1	3	4	75
Phlebitis after bleeding	0	2	2	100
Valvular disease of heart	16	7	28	30·43
Meningitis cerebralis	0	2	2	100
Erysipelas faciei	11	1	12	8·33
Catarrhus intestinalis	110	13	123	10·56
Enteritis et entero-colitis, acute	31	6	37	16·21
Colitis chronica	0	2	2	100
Icterus	21	2	23	8·69
Bright's disease	20	26	52	50
Acute articular rheumatism	20	2	22	8·69
Intermittent fever	60	6	66	9·09
Chlorosis	20	5	25	20
Delirium tremens	144	36	180	20
Chronic alcoholism	12	4	16	25
Total	774	185	959	

[1] Of 20 cases, Huss states that 12 died, 4 recovered completely, and 4 only partially. A case of cicatrization of a supposed pneumonic abscess has been recorded by Dr Stokes.

[2] Hughes (Guy's Hosp. Rep. 2d Ser. vii. 1848) found 28 cases of gangrene in 200 post-mortem examinations of Pneumonia. At one time it was noted that several cases of gangrene appeared during the prevalence of an epidemic of influenza, and that as many as six cases occurred in one week.

[3] See also notes to Section on the Morbid Anatomy of Pneumonia, "Gangrene."

[4] Under this head I only propose to treat of such complications as may appear secondarily to or simultaneously with Pneumo-

[1] The inclusion of cases of Broncho-pneumonia in Huss's statistics must, however, be remembered.

of resolution has been already considered. Unless very considerable in amount, or when occurring on the site opposite to the pneumonic lung, it does not very materially modify the mortality. Under the latter circumstances, however, it may dangerously lessen the respiratory surface. Pneumothorax has been mentioned as an occasional complication; but its existence is very doubtful, and is entirely unsubstantiated by post-mortem evidence. Probably the tympanitic note occasionally heard over the non-consolidated parts has given rise to error in this respect.

Pericarditis, though a less common event, is a very dangerous complication. Huss's statistics show that it proves fatal in more than half the number of cases affected. In some cases it appears to originate in the same cause as the Pneumonia, or it may be caused by a direct extension of the inflammatory affection—(it may, however, occur, and apparently with about equal frequency, in pneumonias of the right and left side)—or, finally, it may in some cases be due to secondary septic effects resulting from the absorption of inflammatory products in the lung.[1] Its influence on the pyrexia and on the phenomena of resolution have been already described.

The evidence of other cardiac lesions secondary to Pneumonia is but slight, but in some cases there appears to be a tendency to the formation of fibrinous concretions in the cavities of the heart.

Icterus.—A slight icteric tinge of the conjunctiva is by no means uncommon. Distinct jaundice is also an occasional complication.[2] It may in some cases be produced by congestion of the liver, arising from the impeded circulation in the lungs; in others it is probably due to coincident gastro-duodenal catarrh. It is more common in the summer than in the winter months. It appears to be more frequently associated with Pneumonia of the right than with that of the left lung; but it must be remembered that the former is much more liable to be affected. The theory of its production by direct extension of the inflammatory action from the lung to the liver is now generally considered untenable.[3] I have met with one case in which icterus preceded the attack of Pneumonia; it usually, however, follows the invasion of the disease. According to Grisolle, the liver can very rarely be felt to be enlarged.[1] Gastric symptoms, and particularly nausea and vomiting, tend to accompany this condition.

Parotitis is a rare complication, but it is one whose appearance seriously increases the gravity of the prognosis. Most of the cases of Pneumonia in which it occurs prove fatal.[2] Grisolle states that its progress is very rapid, and that it tends to pass into suppuration or gangrene. In the former case, the pus may burrow deeply among the muscles of the neck, or may open into the external ear. The pus is, however, usually infiltrated, so that but little escapes on incision. It appears to be most common in advanced life. The only case in which I have met with it was in a girl aged fourteen.[3]

In rare cases an *inflammatory condition of the joints* occurs in the course of Pneumonia. Grisolle reports four such. In all these the joint affection was multiple, but it was not migratory. Three of these cases proved fatal. In the only one examined the joints contained pus, and Grisolle considers it probable that the affection was septic in its nature, since in all the fatal cases the lung was found in a state of suppuration. In one case, a patient of Dr. Reynolds, a man of dissipated habits, effusion came on in the knee-joint on the day after the crisis, attended with a slight rise of temperature. The Pneumonia resolved perfectly, but the swelling of the knee became chronic.[4]

Variations in the Clinical Aspect of Acute Pneumonia.

Many of these, depending on the severity of the coincident affection of the digestive or of the nervous system, have been already described. Three classes, however, deserve some mention, viz.,

[1] Dr. Parkes, Clinical Lecture, Med. Times and Gaz. 1860; i. 187.

[2] It occurred in 7 per cent. of Grisolle's cases, in less than 1 per cent. of 237 cases analyzed by Roth, Würzb. Med. Zeitsch., i. Nos. 3 and 4. Cvostek (Canstatt's Jahresb. 1867) met with icterus in the proportion of 21 per cent. of 147 cases, and the mortality in these cases was 23·8 per cent. The average mortality of the whole number of these cases was 16·8 per cent.

[3] Out of 20 cases observed by Grisolle, 16 were associated with Pneumonia of the right lung. In these cases, however, the right and lower lobes were affected with equal frequency.

[1] Andral reports a case (Clin. Méd. iii. p. 441, obs. lv.) of icterus accompanying Pneumonia, where the hepatic region was painful and resistant. The stools were natural, though all the tissues were stained with bile. Post-mortem, the liver was found softened, and of a deep red color. The biliary passages were free; and bile could easily be expressed from the gall-bladder into the duodenum.

[2] Two such cases are related by Béhier, Conférences de Clinique Médicale.

[3] This case has already been referred to as an instance of Pneumonia passing into gangrene.

[4] This patient was transferred to the surgical wards, and I am unable to trace his subsequent history.

Latent Pneumonia, the so-called Typhoid Pneumonia, and Pneumonia assuming an intermittent type.

Latent Pneumonia.—The class of *Latent Pneumonia* is an ill-defined one, and in many cases in children the accompanying cerebral affection may mask the ordinary symptoms of the disease.

It is very rarely that in vigorous adults the inflammation of the lungs does not present characteristic clinical features, but in old people many of these are often absent. In cases also where Pneumonia is secondary to other diseases, the chief symptoms may be altogether wanting. In old people the disease may be only revealed by prostration, headache, and delirium, and none of the usual phenomena of invasion may be present. Cough also and expectoration may be entirely absent, or the latter may fail to present the characteristic rusty tint, and may be transparent and viscous, or simply puriform. Subjective dyspnœa is also less frequent, though some acceleration of the respiration and the perversion of its normal ratio to the pulse rarely fail to be observed.

The flushed face is also less frequent in the aged than in adults, and the countenance is often pale, earthy, and sunken. The skin may be dry and hot, but it may fail to communicate to the hand the pungent feeling of heat sometimes described; or it may be relaxed and perspiring throughout. Fever is, however, almost always present, though seldom ranging so high as in adults and in children. Its presence is, however, a valuable indication for a careful investigation of the chest, since Pneumonia is one of the few febrile affections to which elderly people are liable. The disease, however, may be so entirely latent that its presence in a state of gray hepatization may only be revealed post mortem after a sudden and unexpected death.[1]

The Typhoid Form of Pneumonia is very common in elderly people, and might be described as a sub-variety of the Latent form. Its occurrence, judging from my own experience, must be rare in this country, though some of the severer, and particularly of the fatal cases, tend to assume towards their close some of the characters described. Dr. Stokes, however, has found it more common in Dublin. It has also been described by Huxham as occurring in scorbutic patients, in whom it is often associated with dysentery, attended by bloody stools. Huss remarks that it occasionally occurs sporadically, but only in those who have been exhausted by toil, want, or other depressing influences. It is very doubtful whether the reported epidemics of this character have been pure Pneumonia, or not rather typhoid fever.[1] Many of the cases in which Pneumonia occurs as a complication of other diseases tend to assume this type,[2] but it may occasionally be met with as the primary disease. It may be described as a form of Pneumonia marked by intense prostration and by the signs of profound depression of the nervous centres. Its invasion is often gradual; the initial rigor may be slight or nil, and pain in the side may be absent or slight; the cough and sputa are often present at the outset, but the latter may be merely viscous, or may present the characters of prune juice. Stupor, alternating with a constant low muttering delirium, and associated with tremors and subsultus tendinum, with a fixed but vacant expression of countenance, and with complete abolition of senses of sight and hearing, and also in some cases of the faculty of speech, are its most prominent features. The tongue is dry and brown, and sordes form on the teeth. Incontinence or retention of urine are sometimes observed. The pulse is small, but markedly accelerated. Sloughs may form on the more prominent parts. These symptoms may continue through the whole course of the disease, which usually ends fatally on the tenth or twelfth day, or later. The course in cases of recovery is commonly protracted, and resolution is very slow.

Wunderlich describes, as a variety of Pneumonia, a class of cases attended with early breaking down of lung-tissue (*Jäuchige Pneumonie*), which present a great resemblance to the typhoid form. The sputa are fetid and of a dirty color. The fever is high, and prostration sets in early. Sweating is profuse, and there is a tendency to colliquative diarrhœa. Their course is protracted, and they tend to a fatal termination. When recovery

[1] Hourmann et Dechambre, Pneumonie des Vieillards (Arch. Gén. de Méd. 2e Sér. xii. 37). These authors state that of 49 cases of Pneumonia in old people uncomplicated by disease of the heart or brain, 21 were latent. It is almost always latent when occurring in old people with cardiac or cerebral affections. See also Cruveilhier, Anat. Path. liv. xxxii.

[1] This would appear to be the case in the epidemic quoted by Grisolle, as described by Torchet, at Noyers, Mém. Acad. Imp. 1838.

[2] Dr. Stokes (loc. cit. p. 339) describes various forms: (1) As a complication of "Enteritis, or Gastro-Enteritis;" (2) As a complication of true typhus; (3) Occurring in cases of bad erysipelas; (4) Occurring in cases of diffuse cellular inflammation; (5) Occurring in cases of delirium tremens from excess; (6) As a consequence of phlebitis; (7) As apparently the sole disease.

takes place the fever subsides, and the sputa lose their fetid odor and peculiar color, and become simply purulent. I have seen one fatal case of this kind associated with dysentery, and with sloughs in the mucous membrane of the stomach.[1] Some forms of Pneumonia occurring secondarily to dilatation of the bronchi are very prone to assume this character.

Intermittent Pneumonia.—Among the inhabitants of malarial districts the symptoms of Pneumonia, and in particular the pyrexia, often assume an intermittent type.

The invasion is commonly attended with rigors, followed by pyrexia and sweating; but with these symptoms of ague the physical signs of Pneumonia may simultaneously make their appearance. In some cases, after the first twenty-four hours the fever ceases, and during the apyrexial period a marked improvement is said to take place in the physical signs: the dulness diminishes and the râles disappear, while the respiration over the affected part may be merely weak, or may in some cases retain the bronchial character. A second invasion, however, occurs with increased severity after twenty-four or forty-eight hours, with a return of the physical signs. The subsequent intermissions are less complete, but the pyrexia in such cases has always a distinctly remittent character, which may assume either the quotidian or tertian type; the cessation of the pneumonic signs in the early stages is, however, more complete in the latter than in the former variety. It is said that quinine, if given early, will cut the disease short; but if this is not effected, the Pneumonia tends to become double, and of a dangerous character.[2]

In some cases, however, of Pneumonia where there is no evidence of malarial infection, the type of the pneumonic pyrexia is distinctly intermittent, with apyrexial periods whose duration may vary from twelve to thirty-six hours. The remissions are attended with marked sweating, and also with an alleviation of the chief symptoms, though the physical signs usually remain unchanged during this period. The exacerbations are sometimes, but not always, attended by a return of the rigors which marked the primary invasion. This class of cases is rare, and the conditions determining their peculiarities are not fully explained. In some instances the exacerbations appear to be due to an irregular progress of the Pneumonia, but in others no determining cause, either of the remissions or of the return of the fever can be discovered.[1]

[1] Dr. Stokes (Cyc. Pract. Med. iii. art. "Gastritis") has also observed this form of Pneumonia associated with severe gastro-enteric disturbance. An instance of this form of Pneumonia is given by Dr. Laycock, "Fetid Bronchitis."

[2] See Morehead, Diseases of India, p. 349 et seq. Most of the other authorities on this subject will be found quoted in Grisolle's work.

Pathology.

A. Morbid Anatomy.—The different anatomical changes which may be found in the course of acute sthenic Pneumonia have been ordinarily described under the terms of *Engorgement*, *Red Hepatization*, *Gray Hepatization*, *Suppuration*, and *Resolution*.

Dr. Stokes has, however, described a stage of *arterial injection* antecedent to that of engorgement, and characterized by a brighter color and by dryness of the pulmonary tissue. Opportunities for observing this condition are extremely rare, and its very existence has been called in question by Rokitansky and by Skoda. There is, however, reason to believe in the probability that such a state may precede the subsequent changes of the inflammatory period, and the auscultatory signs of harsh respiration, which have been described by Dr. Stokes as attending it, have been recognized by many and different authors.[2]

(1) *The stage of Engorgement* is characterized by intense congestion of the pulmonary vessels and by commencing œdema of the lung.

The tissue is of a deep reddish-purple tint. It is heavier than natural, and has lost some of its resistance and elasticity. It pits on pressure, and is more easily torn than a healthy lung. On section a large amount of blood-stained serosity escapes from the cut surface, and in the earlier stages this is frothy from the admixture of air. During this period the tissue is still crepitant, and floats in water to a degree corresponding with the extent to which the condition has advanced. Under the microscope, the capillaries of the pulmonary artery are found to be loaded with blood. The epithelial cells of the air-vesicles are seen to be enlarged and granular, and occasion-

[1] See Wunderlich, Die Eigenwärme im Krankheiten; Thieme, Die Intermitterende Pneumonie, Diss., Jena, 1865; Griesinger, Virchow's Spec. Path. Therap., ii. p. 43. In none of the reported cases of this condition with which I am acquainted has the condition of the spleen been mentioned.

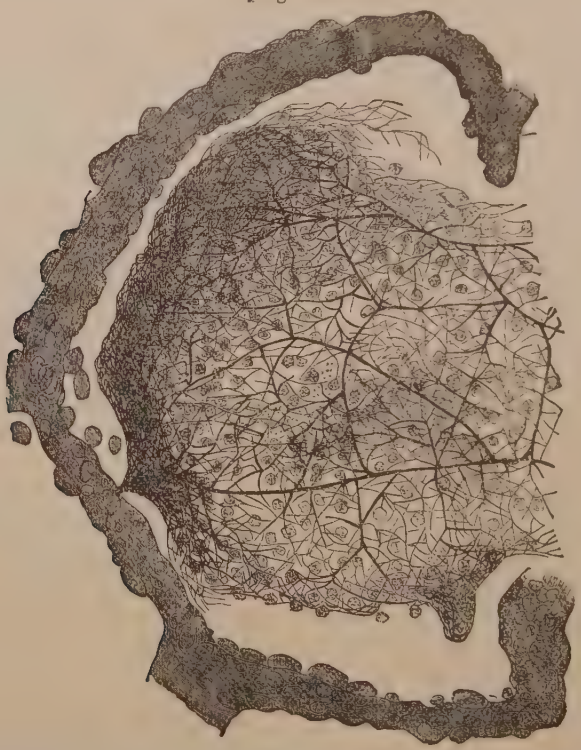
[2] The reality of its existence must, however, in part depend on the question of the increased arterial supply from the bronchial vessels, since congestion of the capillaries of the pulmonary artery does not give this tint.

ally they exhibit a commencing division of their nuclei: some exudation-corpuscles may also be seen in the alveoli, mingled with red blood-corpuscles which have escaped from the capillaries.

The question of the vessels chiefly concerned in the pneumonic process has been largely discussed without any definite settlement having been arrived at. It has been maintained by some that the inflammatory changes are mainly dependent on the bronchial artery as the nutritive vessel of the lungs,[1] and Virchow's observations have shown that the most typical pneumonic changes may ensue in parts of these organs whose supply from the pulmonary artery has been completely arrested by the occlusion of branches of this vessel.[1] It is by no means, however, certain that the nutrition of the lung is exclusively conducted by the bronchial artery; and it is not at all improbable that the branches of the pulmonary artery, whose participation in the process of congestion so vastly exceeds that of the bronchial capillaries, may have no inconsiderable share in the exudative processes which distinguish the condition of hepatization.

(2) *Red Hepatization* is the term generally adopted for the appearance observed in the second stage. In it the lung has

[Fig. 25.



Croupous Pneumonia.—Red Hepatization.—Showing the fibrinous coagulum in one of the pulmonary alveoli, inclosing within its meshes numerous leucocytes, which are already commencing to undergo fatty metamorphosis. A few leucocytes are also seen on the alveolar walls, and the alveolar epithelium is swollen and granular. × 200. (Green).]

become solid; it sinks in water, and the section is that of a solid tissue. It is firm, as if the lung had been artificially injected with size from the bronchi; but it has lost its elasticity and resistance, it tears easily, and breaks down into a pulp under pressure. Its section is less livid than that of a simply congested lung, and is of a dull, reddish-brown tint (sometimes likened, but not very exactly, to mahoga-

[1] This question, according to Virchow, was first raised by Boerhaave. (See Van Swieten, Comm. in Aph. Boerhaave, ii. 712.) It has also been ably discussed by Dr. Morehead, Dis. of India, ii. 311.

[1] Ges. Abhand. p. 369 et seq. Dr. Waters (Dis. of Chest, p. 30) believes that the pulmonary artery is exclusively distributed to the walls of the air-vesicles. Since, however, it has been shown that some of the products of inflammation may escape by the veins, it is possible that this may explain such cases as those described by Virchow; though some doubt still remains as to whether the bronchial arteries may not participate in the process more than Dr. Waters's suggestions would lead him to believe.

ny), which, however, becomes brighter after a short exposure to the atmosphere. It is also opaque, and has lost the glistening transparency of ordinary pulmonary tissue.[1] The color is not absolutely uniform, but mingled with the reddened tint is a grayish appearance, as if Chinese white had been mixed with the coloring matter. Very little serosity exudes on section, but a dirty, rusty-looking, reddish fluid with a certain degree of viscidity may be expressed or scraped from the surface. A characteristic appearance of the section in the Pneumonia of adults is the granular look which it presents, and which is still more distinct when the tissue is torn. The granulations are small and uniform; they give the torn surface the appearance seen on the exterior of a nutmeg, and they may easily be separated on scraping the tissue. This granular appearance is less distinct in children, and varies also in degree according to the amount of œdema present.[2]

During this stage the interlobar septa, and even the larger bronchial vessels, are still distinct, and participate but little in the inflammatory changes, but the latter are sometimes filled with solid exudation-matter. The vesicular character of the lung is, however, entirely destroyed, being replaced by the granular look just described.

The tissue is greatly increased in weight, and, according to Gendrin, its specific gravity when compared with that of healthy lung may be as 1·15 or 1·9 to 1.

The lung is expanded by the exudation present to the fullest capacity of its normal dimensions. It is possible also that it may somewhat exceed this. The possibility of its thus retaining the impress of the ribs has been largely discussed; but it has been definitely settled in the affirmative.

The pleura almost invariably participates in the inflammatory changes when the part affected is superficial. It loses its normal translucency and becomes opaque, and it is generally covered with a layer of fibrinous exudation.

When the stage of red hepatization has lasted some days, its color becomes paler and whiter. This is due to individual granulations becoming whiter in aspect, either singly, or in groups scattered through the surrounding reddened tissue; and this change produces a mottled look in the inflamed part. Coincidently with this change of color there is a gradually increasing loss of the solidity of the affected tissue: the exudation liquefies, and more fluid can be expressed from the cut surface, and the state may gradually pass into that of gray hepatization, though it is very questionable whether perfect resolution does not often take place without the latter being fully attained.

The two conditions are, however, frequently found intermingled, and the lung then acquires a marbled appearance, which, as Laennec remarked, may closely resemble some forms of granite.

(3) *Gray Hepatization.*—In this condition the cut surface of the affected part is of a uniform gray tint, generally presenting, however, a somewhat greenish or olive tinge. The redness of the preceding stage has disappeared entirely, and the granular character has become less distinct. The tissue has lost its firmness and has become soft and pulpy, and allows a dirty-looking, puriform, gray fluid to be abundantly exuded, both on scraping and on pressure. Sometimes a further stage of softening is reached, though this is, comparatively speaking, very rarely observed. Many minor variations of appearance are presented in this state, which usually is found in persons of bad constitution or in cases where Pneumonia is secondary to other diseases. The difference in the appearances observed, depend, however, for the most part, on the greater or less amount of œdema present, and in the comparative indistinctness of the granulations. In some instances these are entirely absent, and the tissue is uniform, smooth, and glistening. Under these circumstances a large amount of serum may escape on pressure, containing but few solid elements, and not presenting, therefore, the milky, puriform detritus usually observed. Such conditions are not uncommon in cases of Pneumonia proving fatal in the course of Bright's disease, when attendant œdema of the lung complicates the inflammatory process. In some instances also this condition appears capable of remaining for some time in a chronic state, when it may form one of the stages of transition between acute and chronic Pneumonia. This is particularly the case in some forms of phthisis, but it is also seen independently of the complication with tubercles. The consolidated lung still retains the gray marbled appearance, and some serosity may escape on pressure, but the tissue gradually acquires a more resisting character, and does not break down easily into detritus.[1]

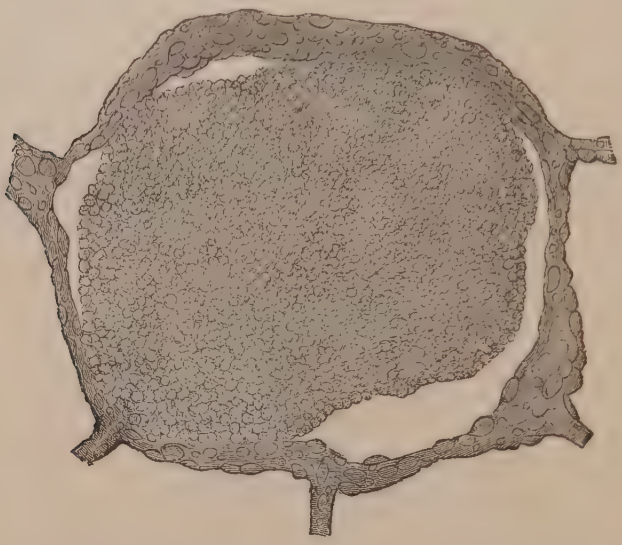
[1] It is to be remarked that this translucency is preserved in conditions of collapse, and that the dead opacity of appearance is one of the best characteristics of the pneumonic process.

[2] See Appendix C.

[1] I doubt much whether cheesy changes in the exudation, as described by Niemeyer, are commonly observed in this form of Pneumonia, independently of tubercular formations in the walls of the air-vesicles. Such an event may be possible, but I believe that cheesy masses commonly found in such lungs in phthisical patients are usually, though not

(4) *Suppuration of the Lung.*—In this state the lung presents a yellower appearance than that seen in the gray hepatization. The granular character is lost, and a diffluent puriform fluid exudes from the cut surface. The whole tissue of the lung is softened and pulpy, and breaks down with the greatest facility under very slight

[Fig. 26.



Croupous Pneumonia.—Gray Hepatization.—Showing the large accumulation of cellular elements within one of the pulmonary alveoli, which in some parts have undergone such extensive fatty degeneration that their distinctive outlines are no longer visible. × 200. (Green.)]

pressure, and it may thus give rise to the false impression that an abscess has been formed. The condition is not, however, specifically distinguished from either of those last named, in respect of the changes in the pulmonary tissue, since pus-cells are present in all stages of the pneumonic process; and the greater degree of softness and the changes of color observed in the so-called gray hepatization and suppuration of the lung are only due to the increasing anæmia caused by the pressure of the accumulated products of inflammation in the interior of the air-vesicles, and by the progressive degrees of fatty degeneration in the cell-forms thus produced: while the gradual softening is attributable to the liquefaction of the previously solidified exudation.[1]

(5) During *the stage of Resolution* the liquefied exudation matter, and the cell-forms which have degenerated and broken down, are gradually absorbed. The expectoration is often in such cases so insignificant as by no means to account for the elimination in this manner of these products and the greater part must necessarily be removed by absorption. Opportunities for the observation of lungs in this condition are rare. I once found, three weeks after the physical signs had disappeared, a considerable amount of œdema remaining in the affected parts, together with a marked loss of elasticity of the tissue[1].

It is a matter of some interest that Pneumonia has been described as a disease of intra-uterine life. F. Weber[2] mentions it as existing in two forms, a white hepatization and a red. The former, however, is now generally considered to be a syphilitic affection. The red hepatization of intra-uterine life occurs as a lobar Pneu-

invariably, the result of a secondary tubercular growth. The discussion of this question, however, belongs to that of Phthisis, and cannot be entered upon here.

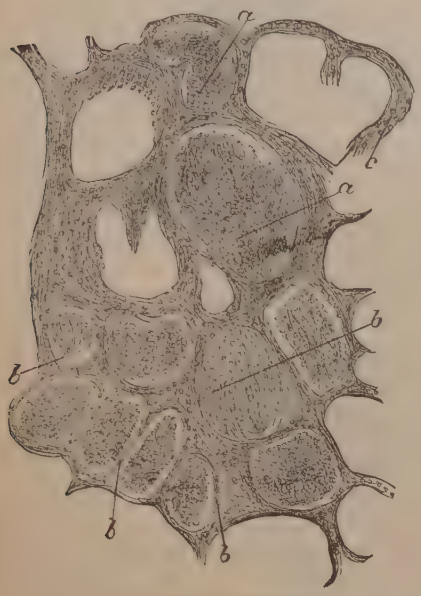
[1] It has been repeatedly affirmed that this condition is not a true "suppuration of the lung;" as stated by Gluge, and certainly it does not specifically differ from the previous stages, since pus-cells are produced throughout the whole pneumonic process. The question is one of terms rather than of a reality, but as the contents of the air-vesicles are more purely puriform than in the earlier stages, there appears to me to be no objection to retaining the expression.

[1] An accident prevented my making a microscopic examination of this lung. Similar conditions have been described by Laennec and Grisolle. Laennec's description of the process is very minute, and subdivided according to the different stages. It would appear, however, doubtful whether these can be so perfectly defined as was attempted by him.

[2] Path. Anat. des Neugeb. und Säuglinge, ii. 41 et seq.

monia. It is most commonly met with during epidemics of puerperal fever, which Weber believes may produce blood-poisoning in the mother before delivery.[1] The lung is very much gorged with blood, and is softer than in the ordinary form of red hepatization, though resembling the Pneumonia found in some conditions of blood dyscrasia. The disease usually proves fatal within a few hours after birth.

Fig. 27.

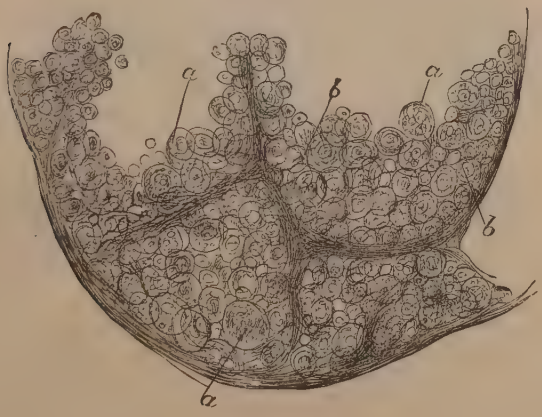


Air-vesicles of Inflamed Lung.

The microscopical examination of a pneumonic lung[1] is at once sufficient to show that the inflammatory products are almost entirely accumulated in the interior of the air-vesicles. This is seen in Fig. 27 (× 100 diam.); and the same appearance persists throughout all the stages of the process, including that of gray hepatization, in which, as originally remarked by Gluge,[2] the elastic fibres are still distinct. The walls of the vesicles are, however, somewhat swollen, but this is almost entirely owing to the congestion of the capillaries, and there is an entire absence of any interstitial growth or exudative process within or external to them. In some parts, *b b*, in hardened preparations, the contained masses of cells separate from the walls of the air-vesicles, leaving the latter intact.

When examined with a higher power (Figs. 28 and 29, × 700), the alveoli are seen to be occupied by a considerable variety of cell-forms held together by a tenacious material, and mingled with a number of free red blood-corpuscles (Fig. 28, *b*). The amount of these latter, however, varies greatly, but in some instances it may be so excessive as to form a large proportion of the material filling the alveoli. In the earlier stages of the process, the epithelial cells of the alveoli and smaller bronchioles are seen in different stages of transformation and proliferation. They are greatly enlarged, measuring from $\frac{1}{1500}$ to $\frac{1}{1000}$ or $\frac{1}{2500}$ of an inch in diameter. They tend to assume the

Fig. 28.



Alveoli in Pneumonia.

[1] Forster (Handb. der Path. Anat., 2d Ed. ii. 248) says that he has met with this change under similar circumstances.

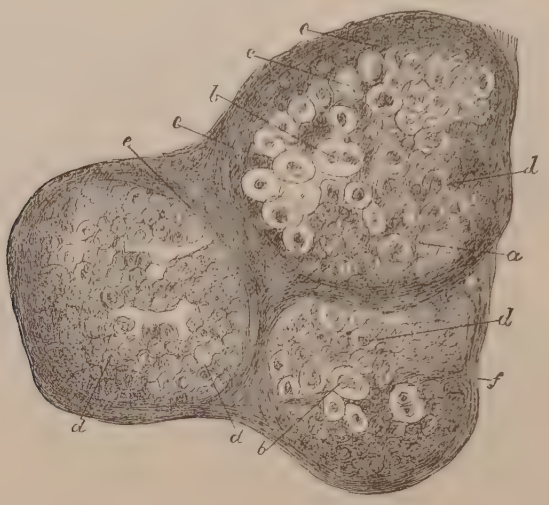
[1] On this subject see also Dr. Da Costa, "Amer. Journ. Microscop. Science," 1855; and Rindfleisch, "Lehrb. der Path. Gewebelehre."

[2] Anat. Microscop. 1838.

round form, but some (Fig. 30, *a b d*) are at times irregular in shape. They are for the most part very granular.[1]

In the early stages they are cloudy and opaque, but they clear with acetic acid, showing that they contain an excess of

Fig. 29.



Alveoli in Pneumonia.

fibrinous matter; but as the process advances, the granular character is mainly due to the accumulation of fat drops in their interior. The nuclei in these cells are sometimes single, and show a distinct nucleolus (Fig. 28, *a*; Fig. 30, *a c c*); but

Fig. 30.

Altered Epithelial Cells in Pneumonia.

in the majority of instances the nuclei may be seen in all stages of multiplication and division (Fig. 29, *b b*; Fig. 30, *b e d*), until several nuclei are found accumulated in the interior of the cells (Fig. 30, *f*).

Large cells may, however, at times be found in other conditions in which the nucleus has disappeared, and the cell may only present a clear hyaline cavity in its interior, which gradually increases in size until, in some instances, only a narrow margin of the granular cell contents is seen surrounding the central space (Fig. 29, *c*; Fig. 30, *k k*). Together with these there are seen various forms of pyoid cells, some of which present one, and others two nuclei or more (Fig. 30, *f*). They

[1] In Figs. 28 and 29, the preparations from which the drawings were made were put up in Canada balsam or Damara gum, and the antecedent modes of preparation (immersion in turpentine and chloroform) dissolved out the fat granules.

are smaller than the foregoing, and average from $\frac{1}{500}$ to $\frac{1}{300}$ of an inch in diameter; the nuclei vary in size from $\frac{1}{3000}$ to $\frac{1}{1000}$ of an inch. Many round cells are also seen in which no nucleus is apparent (Fig. 30, *h*). Some of these correspond in appearance with that presented by the nuclei of the larger cells; others bear the closest resemblance to lymphoid cells or to the white corpuscles of the blood; others again are larger than these (Fig. 30, *i*). The whole of these cells are finely or coarsely granular, the granules being mainly of a fatty nature. They are often stained by imbibed hematine, and in the later stages pigment granules tend to accumulate in increasing numbers in their interior. They are seen in Figs. 28, 29, to be irregularly scattered among the larger epithelial cells.

As the process advances, the granule cells become more numerous, and the epithelial cells in great measure disappear. This is due to the fatty disintegration of the latter, which may be seen in all stages of this change, large tracts being filled with coarser granule cells, and with the compound granular bodies of Gluge. They break down and their nuclei are set free, until the interiors of the alveoli are almost entirely occupied by the smaller-sized round nucleated and non-nucleated cells (Fig. 31), in which large quantities of fat granules become accumulated. These

Fig. 31.

Alveoli in advanced Pneumonia.

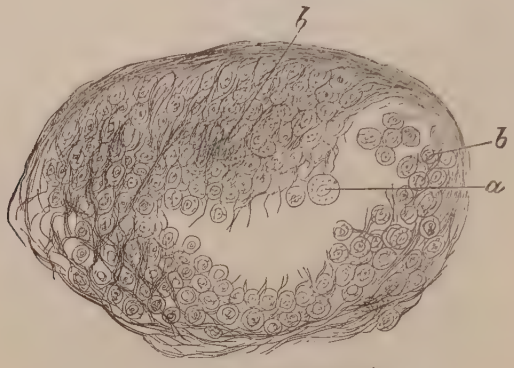
appearances are most common when the stage of gray hepatization is reached; but similar conditions are often found in parts which to the naked eye still present the aspect of red hepatization. In the earlier periods the cells are agglutinated together by a material of a cohesive nature, which is usually considered to be fibrin, but of the nature of which no very precise chemical proof has been afforded;[1] but it may sometimes present a fine network like that seen in whipped fibrin from the blood. Its cohesive nature is, however, distinctly seen in the fact that the granules may be scraped or washed out entire from the cut surface, and these not infrequently present the forms of casts of the smaller bronchi and infundibula, and consist of masses of the cells now described. If a section of the lung in this state be carefully washed over with a camel's-hair pencil, cells are seen still remaining between and imbedded among the elastic fibres of the alveoli, mingled with an adventitious network of a fibrinous nature (Fig. 32). These fibres have not, however, the definite outline and the regular arrangement seen in the process of growth which characterizes tubercular formation, and, though in section some cells are seen irregularly scattered over the walls of the alveoli, no interstitial growth appears to take place in these during the process of

[1] In the earlier stages of the pneumonic process, during the period of engorgement, the air-vesicles are loaded with a clear but very tenacious fluid, which, however, becomes cloudy on the addition of acetic acid. During the height of the consolidation acetic acid effects a partial clearing of the effused material, while during the stage of liquefaction the qualities of this fluid in respect to the reaction with acetic acid revert to the first stage. (Rindfleisch, Lehrbuch der Path. Gewebelehre, 363.)

acute Pneumonia. In the later stages of the process the material holding the cells together loses much of its cohesive properties, and becomes more fluid, and, together with the cell-forms observed, great numbers of free oil globules and of granules of protein matter become apparent. In this stage, scrapings of the tissue yield only cells and a tenacious fluid, and neither the granules nor the casts of the bronchi can be separated entire.

The full discussion of the pathology of

Fig. 32.



Alveolus in advanced Pneumonia.

these processes, involving as it does the whole question of the nature of the changes of tissues in inflammation, can necessarily be only briefly dwelt upon here. The points of greatest interest in relation to it are those regarding the nature of the exudation process, and the origin of the cells which are produced in such excess in the interior of the air-vesicles. Until the publication of Cohnheim's researches, the opinion generally received was that of Virchow, that the coagulable material was derived from blood-plasma, changed during its passage through the inflamed tissues, and that the cell-forms found were the result of increased growth from pre-existing tissues. Cohnheim's[1] statement, that the so-called pus-cells in inflammatory processes consist chiefly of the white corpuscles of the blood which have passed through the walls of the bloodvessels, has been absolutely adopted by Professor Axel Key[2] in respect to Pneumonia, though here the kind of proof obtainable in the mesentery and in the tongue of the frog is necessarily wanting. That such a passage takes place in these parts in the frog can be easily verified, but that this migration of the white corpuscles is the sole source of the vast increase of cells found in inflamed parts appears to me inconsistent with facts. I believe that all the cells and nuclear elements (centres of nutrition) of a tissue participate in the inflammatory process, and multiply in number by division.

The illustrations which I have given of these processes during Pneumonia are, I think, sufficient to confirm this proposition, and the same fact may be abundantly seen in the frog's mesentery, where, in parts when no escape of corpuscles from the bloodvessels is taking place, and even before this process has commenced, a great increase of the nuclei in the tissue may be observed without a single corpuscle having migrated from elsewhere into such parts.

The tendency of all irritative growth is to approximate to what may be considered as the primary or lymphoid cell-forms, and hence "pus-cells," which in some cases are undistinguishable from the white corpuscles of the blood, are produced wherever rapid growth of this nature occurs. I believe, therefore, that while a number of the cells in the pulmonary alveoli may be those which have escaped from the bloodvessels of this part, another series are produced in the manner above described, and which in their final stages are undistinguishable from the former.[1]

With respect to the exudation, it may be held, when spontaneously coagulable, to consist mainly of the blood-plasma; but the condition of this product in the early stages of Pneumonia suggests at least a doubt whether it is merely a transudation, or not rather, as Virchow has

[1] See Appendix D.

[2] Hygeia, 1868, p. 530. Translated by Dr. W. Moore (Med. Times and Gazette, 1869, 452).

[1] That a local production of white corpuscles takes place either in the lymphatics or in the bloodvessels (and probably in both) of an inflamed part, is, I think, probable from the great increase of these in the blood generally. If this were not the case their exit at the seat of inflammation ought largely to diminish their relative number in the systemic blood, whereas precisely the reverse is observed.

taught, that it owes some of its properties to transformations which it has undergone during its passage through the inflamed tissues.

The terminations of acute Pneumonia in *abscess* and *gangrene* are very rare.

Abscess[1] is probably the rarer of these. It is due directly to the breaking down of the lung-tissue, and it is most commonly found in parts which are the seat of gray hepatization. The size of such abscesses varies from that of a bean or a pea to a cavity of some inches in diameter.[2] They may in rare instances give rise to pneumothorax, and a case is reported of an opening being effected into the pericardium.[3] They are sometimes found surrounded by a thickened wall of false membrane, but more commonly they merely form irregular excavations in the softened tissue which may hang in irregular necrotizing rags in their interior.

Gangrene has been already stated to be rare in acute primary Pneumonia, but its occasional occurrence seems to be indisputable.[4] It may invade considerable tracts of tissue. The distinction between some forms of gray hepatization and true gangrene is not always very sharply defined. The former may be found rapidly breaking down into a pulpy detritus of a dirty and blackened appearance, but wanting the characteristic odor of a gangrenous lung. Such states appear to occur most commonly in persons of bad constitution or under peculiar conditions of blood-poisoning.[1] This state corresponds with the Ichorous (*Jauchige*) Pneumonia of Wunderlich, to which allusion has been already made. The condition is usually accompanied by intense typhoid prostration, and the Pneumonia only appears to be part of a constitutional state which is of the extremest gravity.

When true gangrene takes place, the part affected is dark and stinking, and is commonly reduced to a pulpy débris. The gangrenous fragments are not infrequently found floating in a pseudo-cavity amid fetid putrilage. Huss attributes its origin to thrombosis occurring in the branches of the pulmonary artery—a view originally entertained by Carswell;[2] but in some instances it may be due to the directly destructive effect of the inflammatory process destroying the vitality of the tissue, or to an arrest of the circulation by the excessive accumulation of its products in the interior of the air-vesicles. The portions affected are commonly surrounded by tissue in a state of gray hepatization.

SITE.—The most frequent *seat* of acute primary Pneumonia is in the lower lobe of the right lung.

The excess of frequency of the affection of the right lung, independently of the seat of the disease, over that of the left, is

[1] Huss estimates its frequency as once in 50 or 60 cases; Laennec only saw five instances, but he probably overrated their frequency as diagnosed by physical signs. Chomel met with it three times; Louis, Andral, and Grisolle have each observed only one instance. Morehead (loc. cit.) in 189 cases only found five instances. Of twenty-five cases collected by Grisolle from different sources, eight occurred above ætat. 70, twelve above ætat. 50, and three above ætat. 45. According to the statements of Barthez and Rilliet, and of all other authors, abscesses are very rare in the primary Pneumonia of childhood, contrasting in this respect with the effects of Broncho-pneumonia. They appear to be proportionately more frequent in Pneumonia of the upper than in that of the lower lobes, especially when the relative liability of these parts of the lung to the primary disease is considered. Multiple abscesses are most commonly the result of pyæmia, and have been described under this head. (See vol. i. of this work.) Rare instances may be found of abscesses in the lung caused by those formed in the different organs of the abdominal cavity perforating the diaphragm. Several cases of this kind have been collected by Dr. Stokes. (See also Ulcer of Stomach, vol. ii.) Foreign bodies entering the lung are also an occasional cause.

[2] A remarkable case of the cicatrization of an abscess is reported by Dr. Stokes. Laennec describes one involving the greater part of the middle and lower lobes.

[3] Béclard, Bull. Soc. Anat. 1863, p. 356. (Grisolle.)

[4] Andral, Clin. Méd., obs. vol. iii. 63 and 68. Willigk (Prager Vierteljahresch. xxxviii. p. 13) found gangrene in 52 out of 583 post-mortems of Pneumonia=3·3 per cent. It occurred in 3·6 per cent. of the males, and 2·9 per cent. of the females. Huss, in 2166 cases of Pneumonia, met with only 12 instances: all were in males, aged from 35 to 55, and all the cases were in patients of exhausted constitutions. Dr. West (loc. cit. p. 318) says that "the lung in childhood shows a much greater tendency to pass into a state of gangrene than in adult age." This tendency, however, is not seen in the acute Pneumonia of children, and instances of such an occurrence only occur singly in the works of different authors. (See Steffen, loc. cit.) Ziemssen (loc. cit.) met with only one case out of 201 instances of primary Pneumonia in children.

[1] See a case by Bamberger (Deutsche Klinik, 1850, 115) of Pneumonia of this nature passing into abscess. It occurred five days after parturition. A case is reported by Dr. Laycock (Fetid Bronchitis, p. 27) of acute gangrenous Pneumonia destroying nearly the whole of one lung, and proving fatal within a month. Tubercles were present in this case.

[2] Illust. Elem. Forms of Disease, art. "Mortification."

variously stated by different observers[1] in the proportions of 5 to 3 or 7 to 4. This predominance of the right side over the left exists from the earliest infancy, but diminishes somewhat with advancing age.[2] The same relative proportions obtain equally for both sexes.

The lower lobe is affected more frequently than the upper in both lungs collectively in the proportion of about 3 to 2.[3] The proportion remains nearly the same for childhood[4] and adult age; but in more advanced periods of life there is a great tendency to invasion of the upper lobes. This proportion, however, according to Dr. Stokes, varies from year to year and it appears also to be sometimes influenced by epidemic causes, rendering the upper lobe more liable to suffer than the lower.

In relation to the lung affected, Pneumonia of the upper lobe is singularly more common in the right than in the left lung,[5] and with a relative frequency which is greatly in excess of the proportion observed in the affections of the two sides, when considered independently of the locality of the inflammation.

The middle lobe of the right lung is still less frequently affected. Dr. Walshe states that Pneumonia having this site is usually either the result of endocarditis, or that it depends on blood-poisoning.

Double Pneumonia is comparatively much less frequent than the unilateral affection, except in the case of Bronchopneumonia.[1] The liability of old people and children to this form of the disease has, however, led to an exaggeration of the frequency with which both lungs may suffer. It is comparatively rare that both lungs are attacked simultaneously, and the invasion of one is commonly secondary in point of time to that of the other. No difference appears to exist in the relative liability of either side to be followed by the attack in the opposite lung.

The Mode of progressive Extension of the disease is usually direct from the site first implicated. Exceptions to this, however, occur when the opposite side suffers subsequently, and also sometimes when the upper lobe is invaded after the disease has commenced in the lower lobe of the same side. It is also not very uncommon in fatal cases to find disseminated nodules of pneumonic change, varying in size from a hazel-nut to a walnut, scattered irregularly around, and sometimes at a considerable distance from the larger mass, and separated from it by apparently sound, or sometimes by unduly hyperæmic tissue.

The Rate of Evolution of the different stages of the process appears to be very variable. A lung may remain in a condition of red hepatization during eight or ten days, or even for some weeks; while in others the condition of gray hepatization may be found as early as the fourth or fifth day.[2] Both Laennec and Huss consider the stage of engorgement to last from one to three days; whether it can persist longer than this without producing some consolidation of lung is, however, doubtful. It is not uncommon for twenty-four hours to elapse before the physical signs of consolidation become apparent; but the duration of the stage of engorgement may be so short that a large tract of lung may be consolidated within a few hours after the first rigor.[3] The duration

[1] Huss, of 2616 cases, found 53 per cent. in the right lung, 32 per cent. in the left, and double Pneumonia in 15 per cent. Grisolle, in 1430 cases, collected from various authors, found 742 cases in the right lung, 426 in the left, and double Pneumonia in 262. The latter number he regards as doubtful.

[2] Hourmann and Dechambre found in old people the proportionate frequency of Pneumonia of the right lung to that of the left, as 34 to 27.

[3] This is Andral's statement. Wunderlich, in a calculation of 660 cases, from different authors, gives the following numbers: Lower lobes, 397 cases; upper lobes, 180 cases; affection of a whole lung, 83 cases. Grisolle, from a calculation of 264 cases, gives the proportions as—lower lobe, 133; upper, 101; middle lobe, 30 cases.

[4] Barthez and Rilliet (i. 516) found in 122 cases: Upper lobe, 42; lower lobe, 65; affection of the whole lung, 3; double Pneumonia, 12 cases.

[5] Grisolle states that Pneumonia of the upper lobe in the right lung is two and a half times more common than in the upper lobe of the left. The observations of other authorities show a still more striking difference. Barth states the relative frequency of Pneumonia of the upper lobe of the right lung to that of the left as 18 to 1; Briquet as 18 to 4; Barthez and Rilliet as 9 to 1. Ziemssen, in 234 cases, gives the following numbers: Right side collectively, 126 cases, upper lobe, 57; lower lobe, 55; middle lobe, 14. Left side collectively, 106 cases; upper lobe, 27; lower lobe, 79.

[1] The proportion of 15 per cent. given by Huss represents nearly the average frequency of its occurrence, but the smaller numbers of some observers show different ratios. Grisolle states its frequency to be 11 per cent. The Vienna returns for 1860 place it at 5 per cent. Barth, in 125 cases, at 6 per cent. Willigk, on the other hand, loc. cit., found double Pneumonia in 50 per cent.

[2] I have seen this in a child under æt. 1, when the disease began acutely. Laennec (Forbes' Trans. p. 206) states that the stage of purulent infiltration may be reached in thirty-six hours.

[3] Dr. Stokes, loc. cit., p. 120, remarks that in some instances of typhoid pneumonia, there is no evidence of an antecedent stage of engorgement, but that of the lung may become solid without any crepitant râle preceding this change. He further observes, however, that this rapidity of progress is not common in the sthenic forms of the disease.

of the stage of red hepatization was stated by Laennec to vary from one to three days, and of suppuration from two to six days. Huss reckons the former as lasting from five to seven days. The periods of resolution have been already referred to.

The termination in abscess or gangrene usually occur at later periods; but even the latter, as has been already stated, may be found very early in the course of the disease.

THE PATHOGENESIS of acute primary Pneumonia is involved in considerable obscurity, and has been the subject of much discussion.[1] Two opposite theories have been advanced respecting its origin, both of which are supported by certain facts and are opposed by others.

These theories may be briefly stated in the following terms:—

(1) That Pneumonia is a "specific" fever, of which the disease in the lung is only a local effect.

(2) That it is a purely local disease, of which the pyrexial and other phenomena observed are only the immediate consequences.

The second hypothesis, as such, appears to be scarcely a tenable one, and even the first appears to require some modification.

The arguments in favor of the first hypothesis are mainly derived from the comparative rarity of discoverable causes for the origin of Pneumonia,[2] and from the suddenness of the crisis while the inflammation is still at its height.

The question of the mode of origin of the disease has been already considered under the head of etiology. It appears, however, deserving of remark that the theory of a "specific" cause can scarcely be maintained for Pneumonia in the same sense as that in which the term is employed for the contagious pyrexial diseases. The causes of Pneumonia are manifold, and the disease may originate under such diverse conditions, that it seems impossible to attribute it to any single blood-poison.

On the other hand, the most probable hypothesis to explain its origin is that of an altered composition of, or the existence of some morbid material in, the blood, which from its special qualities may affect a particular organ, or, as is more probable, may, under local predisposing causes, excite inflammation in that part of the system which in any given individual is the most liable to suffer as a *locus minoris resistentiæ*.

It is not improbable that some of the antecedent symptoms, the malaise, the pains in the limbs, the headache, and the slight jaundice occasionally observed, may be due to the blood alteration; but it must be remembered, that in a not inconsiderable proportion of cases, the outbreak of the pneumonic fever is sudden, without being preceded by any of these prodromata. The nature of the alterations in the blood capable of producing the disease are, like those of all other spontaneous inflammations, entirely unknown; and the hypothesis of an antecedent condition of hyperinosis advanced by Naumann[1] seems to be disproved by some of Zimmermann's analyses. It would appear indeed from these that the excess of fibrine observed in the blood of pneumonic patients is almost entirely a secondary phenomenon, and that it is, as Virchow affirms, a consequence and not a cause of the inflammatory process in the lung.[2]

[1] See especially on this point a clinical lecture by Dr. Parkes, Medical Times and Gazette, 1860. i. 187.

[2] It has already been stated (see Etiology) that experimental attempts at the production of a disease resembling acute primary Pneumonia by direct irritation of the lung, have invariably failed.

[1] Ergebnisse und Studien aus der med. Clin. zu Bonn, 1858. Naumann has afforded no direct proof of an increase of fibrine in the blood antecedent to an attack of Pneumonia. He says, however, that symptoms resembling the prodromata of Pneumonia are sometimes associated with hyperinosis.

[2] Thus, in "Prager Vierteljahresch," 1852, vol. xxxv., in a patient bled, after signs of the stage of engorgement had lasted five days, the blood contained only 1·13 per 1000 of fibrine. In a venesection practised 36 hours later, and when signs of consolidation had supervened, the fibrine amounted to 4·41 per 1000, and in a third venesection practised after 6 days it had risen to 7·16 per 1000. Zimmermann argues further, that this rapidly increasing quantity of fibrine in the blood, in the later stages of Pneumonia, is not due to venesection, for in other cases bled *for the first time* on the third, fourth, and eighth days respectively the fibrine amounted to 7·2, 8·0, 9·1, and 9·6 per 1000. Also in "Analyse des Blutes," p. 370, he has, as the result of more extended observations, found that in eight cases of commencing Pneumonia the blood contained either a normal or a less than normal amount of fibrine. In eight other cases where venesection was practised within the first 24 hours, the proportion of fibrine was between 3 and 7·5 per 1000, and in eight other cases, the blood in the second 24 hours of the disease contained fibrine varying from 3 to 7·5 per 1000. Zimmermann's method of analysis leads him to estimate the amount of fibrine in the healthy blood as lower than that given by many observers; but this only adds strength to his estimate of the proportion observed in inflammatory diseases. He places it at 1·689 per 1000 (loc. cit. p. 17), while Becquerel and Rodier estimate it at 2·5, and Andral and Gavarret at 3 per 1000. The increase of fibrine in the blood during the progress of Pneumonia is abundantly

The theory of hyperinosis as a cause of Pneumonia has also but little support in the diseases with which it is commonly associated, for though it is a not uncommon complication of acute rheumatism, in which this condition of blood is present, it also occurs in other diseases when the amount of fibrine is below the normal standard,[1] and in some of these, as in typhoid fever, the supervention of pneumonia increases the proportion of fibrine in the blood.[2]

It would appear from the consideration of the various diseases with which Pneumonia may be associated, that many, and probably different, blood-poisons may have the power of exciting inflammation of the lungs.

That the lungs should be especially liable to become affected by causes of this nature cannot be regarded as extraordinary, when we consider the importance of their functions as purifying agents of the blood. Nor does it seem improbable, from the complexity of the lymphatic structures which they contain, that other changes in the composition of the blood, in addition to its mere aëration, may be accomplished by their means, though of the nature of these changes we are as yet ignorant.

The lung, from its embryological development and anatomical characters, is closely allied to the glandular organs, and it is on these that blood-poisons produce their most marked effects. It is further to be noted that other organs of this class are not unfrequently simultaneously affected. The frequent association of albuminuria with Pneumonia can scarcely be regarded as a mere accidental complication, and it is by no means improbable that the kidneys are, under these circumstances, implicated by the same cause as the lung. Other glands also occasionally suffer, as the parotid: gastro-duodenal catarrh and some degree of affection of the liver are also frequent complications. In addition to these the serous membranes tend also to become implicated as part of the primary disease, and when these relations of Pneumonia are regarded as a whole, it appears that those organs are most likely to suffer which are most commonly affected by recognizable conditions of blood-poisoning. The fact that cases of Pneumonia presenting these complications are more severe and dangerous than the simple disease, would also tend to show a greater intensity of the primary cause, for their mortality is disproportioned to what might be expected (particularly in cases where parotitis is present) from the mere existence of these inflammations, if regarded as purely local disorders. The argument is still further strengthened by the profuse sweating which often attends Pneumonia, and also by the frequent co-existence of herpes, which is so commonly associated with disordered blood-states.

In some cases Pneumonia indeed is known to be caused by recognizable conditions of this nature, as by septicæmia, but in the case of the acute primary disease it is most probable that the poison is one engendered within the system. In the cases where a discoverable cause exists, such as a chill, it is probably due to retained products of secretion injuriously affecting the composition of the blood. It is also not improbable that Pneumonia secondary to uræmic poisoning may have a similar origin; while in the cases where no discoverable cause exists, we only stand, as has been already remarked, in the same position with respect to Pneumonia as we do to other idiopathic local inflammations.

Whether the blood-poison is eliminated by the exudation process must remain a matter of hypothesis, though the sudden cessation of the pyrexia when this stage has advanced to a certain degree would appear to lend some support to this view, and particularly when we remember the analogy, and even the various phases of transition, which exist between exudative and secretory processes.

The sudden outbreak of the pyrexia occurring simultaneously with the supervention of the inflammatory changes in the lung would, however, appear to show that the implication of the nervous system indicated by the fever is largely due to the alteration of the composition of the blood produced by the local process. We have no evidence of any distinct alteration antecedent to this, and much that a large proportion of the subsequent changes in the blood are due mainly to this cause.

All local inflammations produce in this

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confirmed by other observers. Thus Andral (Ess. Hæm. Path. p. 87), in 90 cases, found in

7 cases	fibrine	4—5 per 1000.
17	"	5—6 "
19	"	6—7 "
15	"	7—8 "
17	"	8—9 "
9	"	9—10 "
6	"	10 and upwards.

The largest amount in Pneumonia recorded by Andral is 10·5 per 1000, and Zimmermann (loc. cit. p. 13) also found 10 per 1000.

[1] I have already advanced reasons for doubting whether the Pneumonia which is secondary to many of these diseases differs essentially in anatomical characters from that of the acute primary disease. The firmness of the exudation varies in degree, but this may be influenced by the nature of the disease to which it is secondary.

[2] Andral, Ess. Hæm. Path. 17.

respect similar results,[1] and it is interesting to remark that the pyrexia following a purely traumatic Pneumonia may have the same typical course as is observed in that of idiopathic origin.[2] That the intensity of these blood alterations, and particularly the increase of fibrine, should be so especially marked in Pneumonia, and may in part be referable to the peculiar relations of the organ (to which reference has been already made), is very probable. Pyrexia *per se*, independently of local inflammations, has not, except in the case of acute rheumatism, any marked proclivity to the production of hyperinosis. The large excretion of urea during the height of the pyrexia and its diminution during the progress of resolution (even where effete materials from the lung must continue to be absorbed into the blood), conclusively show that this phenomenon is due to increased tissue-changes throughout the system, produced probably by perverted nervous action, and which are only secondarily referable to the process in the lung. The increased destruction of red blood-corpuscles shown by the simultaneous increase of pigment in the urine, is perhaps referable to both the general and local conditions, since it frequently persists after the excess of urea has ceased to be observed. Zimmermann has further remarked that the decrease in their number, noticed by Andral and Gavarret, may be due not only to this cause, but to a subsequent defective formation arising from the abnormal conditions under which the white corpuscles are formed during the process of local inflammation and in the pyrexial period.

The disorder which on a lesser scale presents the greatest analogy with acute Pneumonia is perhaps acute tonsillitis, where we have the same short initial state, a similar intensity of rigor and prostration, a similar sudden invasion of pyrexia, and a similar rapid decline of this before the local inflammation has shown any signs of abatement. In tonsillitis also we have frequently an equal difficulty with Pneumonia in verifying a distinct cause, and a certain amount of evidence at least exists in the case of the so-called "hospital sore throat," that it may also be produced by other poisons than those originating within the system from the impeded exercise of the functions of the skin.

The associated PATHOLOGY of Pneumonia has been already almost sufficiently described under the complications of the disease. A few points only deserve further attention.

In the Lungs themselves.—The mucous membrane of the bronchi is more or less injected, but the tubes seldom present much evidence of the dilatation observed in broncho-pneumonia. Plastic exudations, moulded to the shape of the tubes are very common in the smaller bronchi. In some cases, however, this process may extend to the larger bronchi, which may be found thus obstructed through considerable areas.[1]

Acute Emphysema is sometimes observed in parts adjacent to the hepatized portion.

Œdema surrounding the consolidated part is more common, and may, by its extension and by its appearance on the opposite side, prove a source of much danger to life.

The Bronchial Glands are usually swollen and medullary in appearance. They are only in the worst instances subject to suppurative changes.

The Pleura is almost invariably inflamed when the hepatized part is situated at the surface of the lung. Effusion is, however, less common than the formation of false membranes.

In the Heart the complication of pericarditis has been already alluded to. In some cases this is due apparently to direct extension of the inflammation, for it is most common when a part of the left lung in juxtaposition with the pericardium is the seat of the Pneumonia. It appears, however, to arise sometimes under circumstances inexplicable by this cause, and it may then, according to the date of its appearance, be held to be due to the same cause as that in which the Pneumonia originated, or to the secondary blood-poisoning[2] caused by the absorption of the inflammatory products from the lung.

The right side of the heart is usually in fatal cases found distended and containing large and firm clots. Bouillaud[3] thought that Pneumonia predisposes to ante-mortem polypoid concretions, and this opinion is confirmed by Hasse,[4] who adds that he has found secondary infarcta in the spleen from this cause.

One of the most important consequences of Pneumonia on the circulation is the occasional occurrence of thrombosis in the pulmonary vessels leading to the affected part. This event, caused in all probabil-

[1] See Andral, loc. cit.; also Zimmermann, Arch. der Phys. Heilk. 1848.

[2] See an interesting case from Mr. Hilton's practice, Medical Times and Gazette, 1867, i. p. 144, where Pneumonia supervened after a broken rib. The temperature rose abruptly to 105°, and fell by crisis on the seventh day.

[1] This condition appears to have been first described by Reynaud, Mém. oblit. des Bronches, Arch. Gén. de Méd. 1835, iv. p. 157.

[2] Parkes, loc. cit.

[3] Traité Clin. des Maladies Du Cœur, ii. 716.

[4] Loc. cit. 214.

ity by the retarded circulation in the lung,[1] is not uncommon, and may, by extending to the larger branches of the pulmonary artery, be a source both of immediate danger from sudden death, and may also, in great probability, retard the process of resolution and the subsequent convalescence.

Catarrh of the Gastro-intestinal Mucous Membrane is by no means uncommon. The characters of the appearances found have been already described in the section devoted to diseases of the stomach. In some instances, however, this proceeds to a more serious stage by producing dysenteric ulcerations of the colon.[2] Hemorrhage from the large intestines and stomach have been described by Barthez and Rilliet.[3]

The Liver is found congested, and the gall-bladder occasionally distended, but, even when icterus has been present, there may be no demonstrable obstruction of the ducts.

The Spleen is commonly congested, softened, pulpy, and opaque; characters which it presents after death in most of the acute febrile diseases.

The Brain rarely shows any other change than congestion; but in a few instances, when delirium has been violent, there has been found purulent infiltration of the subarachnoid space on the convexity of the hemispheres, and also of the base, which may also extend to the membranes of the cord. In many cases of delirium, however, the brain is found perfectly healthy.[4]

The influence of primary Pneumonia in the production of *other diseases* appears to be but slight.

That any permanent effect is produced on the heart appears to be disproved by Grisolle's statistics. Nor does the occurrence of Pneumonia, in the course of a cardiac disease already existing, appear to have any specially unfavorable effect upon the cardiac state. Its effect on *tubercular patients* appears, however, to be more doubtful. It is perfectly true, as Dr. Walshe has stated, that patients with tubercles already formed in the lungs may recover rapidly and completely from intercurrent acute Pneumonia, and Grisolle found that twenty-two patients of tubercular diathesis affected with Pneumonia all recovered perfectly.[1] In some cases, however, of tuberculosis, the convalescence is protracted and the cure imperfect, and in others the inflammation of the lungs tends to be followed by rapid softening and cheesy change. In fact, intercurrent Pneumonia must always be regarded as one of the greatest dangers of tubercular patients. Resolution is imperfect—the affected parts tend to pass into gray consolidation, and in such parts fresh formations[4] of tubercles rapidly form and disintegrate.

Diagnosis.—The diagnosis of the existence of acute Pneumonia essentially depends on the recognition of an acute febrile disease associated with the physical signs of consolidation of a portion of the lungs. Without this combination its presence cannot be affirmed with certainty in the earlier stages, though it must be remembered that patients may first come under observation at later periods, pre-

[1] Virchow, Ges. Abhand. 222. It appears first to have been described by Baron, Arch. Gén. 1838, ii. 17, who first had the merit of distinguishing this event from the effects of inflammation of the coats of the artery. Malherbe, Journ. de Nantes, 1843 (Constatt's Jahresb. 1843) first referred it to the retarded circulation. See also Mr. Paget's Memoir on this subject, Med.-Chir. Trans. xxvii.; Carswell, Illust. Princ. Forms of Dis., art. "Mortification;" Cruveilhier, Path. Anat., liv. xxxii. p. 2, who distinguishes the site of the coagulation as being in the artery, and not in the veins.

[2] The result of Dr. Bristowe's observations on this subject (Path. Soc. Trans. viii. 66) have led him to regard dysentery as a very common complication of Pneumonia. Out of 16 cases of acute primary Pneumonia proving fatal, he found dysenteric ulceration of the large intestine in four. The possibility of some epidemic influence may perhaps be regarded as not improbable in these cases, since the period over which part of Dr. Bristowe's observations extended included one of the recent epidemics of cholera.

[3] Loc. cit. i. 352.

[4] Grisolle. Louis, Fièvre Typh. i. 359, ii. 37. Immermann and Heller found that out of 30 cases observed in Erlangen during the years 1866 to 1868, nine presented post-mortem signs of meningitis. They attribute this condition in part to the simultaneous occurrence of epidemic cerebro-spinal meningitis; Deutsch. Arch. für klin. Med. v. (Virchow's Jahresb. 1868). Weber (Path. Anat. der Neugeborenen und Säuglinge, ii. 61) has also found cerebro-arachnitis during an epidemic of Pneumonia.

[1] Huss also states, p. 24, that in northern climates acute Pneumonia has very little influence in the production of tubercle. He quotes, however, p. 162, from Gellersted "Bidrag till den Tuberculose Lungostens Nosographie och Pathologie," a statement that of 310 cases of phthisis, 23·5 per cent. had within a longer or shorter period suffered from one or more attacks of Pneumonia.

[2] These changes belong, however, more particularly to the history of phthisis; and their pathology, being in many points disputed, would involve too wide a discussion to be entered upon here, since by some authors the process of Pneumonia complicating phthisis is placed in a separate category of "catarrhal Pneumonia," or "infiltrated tuberculosis."

senting the physical signs of consolidation of the lung, but after the initial fever has subsided. It is, however, important that its early stages should be recognized before the signs of consolidation are distinct. Under this head certain phenomena connected with the mode of invasion deserve special prominence.

Among these perhaps the most important and constant is the pyrexia, which, although not pathognomonic, still presents very marked and distinctive features, and is so invariable a symptom that the diagnosis of Pneumonia during the acute stage can scarcely be made in its absence. Whether or not the invasion be preceded by rigors, the sudden rise of temperature in a subject, previously non-febrile, should always excite suspicion, and it may be remarked that this rise of temperature may precede by hours, or even days, the appearance of the distinctive physical signs in the lungs. The use of the thermometer is also often a mode of recognizing the invasion of Pneumonia when its symptoms are obscure, and appearing in the form of vomiting or convulsions in children, or of the prostration with which it often commences in old people. The rise of temperature in most of the acute febrile diseases is commonly gradual; in Pneumonia it is sudden, and maintains a higher elevation, during the first forty-eight or seventy-two hours, than is commonly seen either in these or in tubercular meningitis.[1]

The other phenomena of invasion which are most distinctive are the acceleration of respiration and the perversion of its ratio to the pulse. If to these and to the pyrexia are joined cough, rusty sputa, and pain in the side, the diagnosis of Pneumonia becomes one of infinite probability. Of the last-named symptoms, the relative acceleration of respiration is perhaps the most valuable, if, as Dr. Walshe remarks, hysteria be excluded, since expectoration may be absent, both in adults and children, or in the former the blood-stained tint may be wanting, and on the other hand, appearances of a very similar character to those seen in the first stage of Pneumonia may sometimes be observed in the sputa accompanying cardiac disease, and also in the early stages of congestive bronchitis. It may be noted, however, that in the last-named diseases fever may be entirely absent, or if present in bronchitis, the elevation of the temperature is rarely so considerable or so sudden as in the commencement of Pneumonia.

The distinctive features presented by the physical signs have been already fully described. When, however, in the commencement of the disease the inflammation first attacks the central parts of the lung, the signs of consolidation may be masked by healthy pulmonary tissue nearer the surface. Under these circumstances harsh breathing or weakened respiration may be the only phenomena observed.

Crepitation, when present, and when the possibility of œdema and of pulmonary apoplexy are excluded, is a valuable aid; but it is not unfrequently absent, and consolidation may take place so rapidly that it may not be heard in the earlier stages. As a rule it only furnishes further grounds for suspicion, until dulness on percussion, bronchial or tubular breathing, and bronchophony are established. The chief fallacy attending percussion is the occasional production of a quasi-tympanitic note over portions of lung, below which deeper-seated consolidation exists.[1] A comparison of the two sides is, however, in children, often indispensable. Percussion of the chest of children should also be gently practised for reasons which I have already stated.

The superaddition of the auscultatory phenomena of the breathing and voice, and the increase of vocal fremitus over the affected part, if occurring collectively, render the diagnosis absolute; and as this combination of phenomena is the most frequent, Pneumonia may commonly be recognized with facility.

In exceptional cases, however, variations in these signs occur, which require some care in the diagnosis, particularly when one or more of them are wanting. This is sometimes the case in central Pneumonia, when the respiration may remain harsh or blowing, and crepitation and bronchophony may be absent. In some of these cases the diagnosis of Pneumonia can only be of relative value, depending on the presence of the characteristic pyrexia accompanied by rusty sputa.

The diseases of the lungs with which Pneumonia is most likely to be confounded are pleuritic effusion, œdema of the lungs, collapse, and certain forms of acute phthisis.

The question of the diagnosis of Pneumonia from *pleurisy with effusion* only occurs when the former affects the base of the lung or the whole organ.

In typical instances of the two diseases, the distinctive physical signs may be briefly contrasted as follows: In *Pneumonia* the affected side is not distinctly bulged, and the intercostal spaces are not obliterated. Neither displacement of the

[1] Children are, however, liable to such sudden elevations of temperature from very slight causes, so that less reliance can be placed on this sign in them than in adults.

[1] The tubular note over the larger bronchi may, however, sometimes prove deceptive to beginners.

heart, nor liver, nor diaphragm are observed. The dulness does not encroach upon the opposite side, or only to a very moderate degree. The dulness is less absolute than in pleuritic effusion, and has often a tubular tone. It does not change its site with the position of the patient, and the percussion note over the upper non-affected parts, though sometimes tympanitic, is never tubular or amphoric. The respiration over the affected parts is marked by a bronchial, or tubular, or metallic quality. The vocal resonance is strongly bronchophonic, and the vocal fremitus is increased. Crepitation may be heard in forced breathing or coughing.

In *pleuritic effusion* the side is bulged and increased in diameter, the intercostal spaces are obliterated and may even be prominent, and fluctuation may sometimes be perceived over them. Displacement of the heart or liver, according to the side affected, is proportioned to the extent of the effusion. When this is extensive the dulness also encroaches on the opposite side. The percussion note is toneless, the sense of resistance is great. A tubular note, as observed by Dr. Walshe, is sometimes producible under the clavicle. In some instances the level and seat of dulness change with the position of the patient. The respiration below the level of dulness is weakened or absolutely suppressed. Weak, bronchial, or blowing breathing is heard near the spine and over the compressed lung. Vocal fremitus is diminished or abolished. Vocal resonance is also abolished below the level of dulness, and it is bronchophonic or ægophonic towards its limits. Friction may or may not be present.

Difficulties may, however, occasionally arise from exceptional combinations of the phenomena presented by each of these diseases. In the rare instances when, in Pneumonia, there are found, together with dulness on percussion, a simple absence both of the respiratory murmur and of bronchophony and fremitus, the most accomplished observers have been led into the error of mistaking the condition for one of pleurisy with effusion.[1] The signs which best distinguish Pneumonia under such circumstances, are the absence of the enlargement of the side of the obliteration of the intercostal spaces, and of the displacement of the various viscera, which characterize extensive effusion. Variation of the percussion dulness with the position of the patient, may, if observable, serve as a further aid if pleurisy be present, though its absence cannot always be relied upon for the exclusion of this affection. As a sign of minor value, it may be stated that the percussion note is more absolutely toneless in pleurisy, and seldom, if ever, has the higher pitch of that observed in Pneumonia. The tubular or amphoric note over the upper part of the lung sometimes heard in pleurisy is not, as observed by Dr. Walshe, met with in the non-affected upper portions of the lung when Pneumonia exists at the base; the percussion here, though hyper-resonant, being commonly of a lower pitch, and sometimes tympanitic in quality. The invasion of the pyrexia is commonly less acute, and the temperature less elevated in uncomplicated pleurisy.[1] It is, however, an event of the extremest rarity that the absence of respiration and the diminution of the vocal fremitus and resonance are, as a matter of practice, found to coexist simultaneously over a pneumonic lung. In doubtful cases, the fremitus may aid in distinguishing the two diseases, being increased in most cases of Pneumonia and diminished in pleuritic effusion. In children, and when in adults and females the voice is weak, this sign may be comparatively indistinct.

In the cases of pleuritic effusion, where bronchial breathing and bronchophony persist, the diagnosis from Pneumonia may also commonly be made by the signs above enumerated. The fremitus may be a further guide,[2] and, as Dr. Walshe has remarked, the true tubular respiration of Pneumonia is wanting in pleurisy, and the indistinct bronchial breathing heard is most commonly met with near the spine.

Œdema of the lungs, which may be attended by the crepitant râle of Pneumonia, may commonly be distinguished from it by the absence of pyrexia, by the minor degree of dulness, by the respiration being simply weak, and by the concomitant affections in which it originates.

The diagnosis of simple Pneumonia from *acute phthisis* when the latter is only attended by the disseminated formation of miliary tubercles, is comparatively

[1] See Barthez and Rilliet, i. 589; also Wintrich, before quoted.

[1] This sign is of minor value in relation to cases of Pneumonia of moderate severity, and when the temperature does not rise above 102°. It should also be remembered that the Pneumonia may come under observation for the first time after the fever has subsided. Under these circumstances the diagnosis from pleuritic effusion may depend on the physical signs alone.

[2] Dr. Walshe, however, states that fremitus may be diminished in extensive hepatization, though not to the same extent as over an equal amount of effusion. He adds that he has often known fremitus feeble, and vocal resonance strong over effusion, but that he has never met with this combination in hepatization. The value of these signs in diagnosis depends on their combination, and but little reliance can be placed on either singly.

easy, owing to the absence of dulness in percussion in the latter affection. When, however, acute tuberculosis is attended by, or commences with, a rapid and extensive pneumonic infiltration, the diagnosis may be almost impossible during the early stages of the affection. This, however, is less common in acute phthisis than a more gradual extension of the pneumonic process, which usually takes place irregularly and through longer periods than are observed in primary Pneumonia. The pyrexia of acute phthisis is more irregular in its course; it has more marked remissions than those of Pneumonia, and the exacerbations often occur at very varying periods of the day, the maximum temperature being attained on one day in the morning and on another in the evening—a phenomenon of the extremest rarity in primary Pneumonia. Much depends on the time at which the case comes under observation. If at a later period than the first ten days, the protraction of the pyrexia may always be regarded as a suspicious circumstance. If the fever is very irregular in its course, and acute exacerbations with very marked remissions occur at uncertain intervals, the suspicion is still further strengthened, since in most cases, when the pyrexia of a simple Pneumonia is retarded in its final disappearance, the fever maintains as a whole a low standard, and subsequent elevations of temperature to 103° or 104° are very rare. Pneumonia of the apex, running such a course, is still more open to suspicion than that affecting the base of the lung.

If, in addition to these symptoms, signs of the formation of cavities become increasingly apparent, the grounds for an unfavorable opinion are still further strengthened, though doubts may still exist, owing to the possibility of the formation of abscesses in the hepatized tissue. Evidences of progressive disease in other parts of the lung at a late period of the case are still more serious symptoms; and the implication of the opposite side, particularly if general râles appear here accompanied by irregular spots of consolidation, and by signs of destruction of tissue, will, together with the conditions of pyrexia before alluded to, and in conjunction with rapid emaciation and other signs of hectic, render the diagnosis of tubercle almost a certainty. Louis believed that implication of the anterior and superior parts of the lung, without the invasion of the whole apex, was almost certainly an evidence of tuberculosis, but this statement, though affirmed by Barth and Roger, is disputed by Grisolle.[1]

The diagnosis from *Collapse of the lung* will be considered under the head of Broncho-pneumonia.

There are certain other diseases with which acute Pneumonia is occasionally confounded.

The sudden prostration, with severe headache and high degree of pyrexia at the outset, not unfrequently simulate *Typhus;* so much so that from the reports of the different fever hospitals it would appear that a certain number of cases of Pneumonia are annually sent to these institutions under this error. Even in the earlier periods the mistake both from typhus and typhoid may be avoided, as Dr. Grimshaw has remarked,[1] by the observation of the temperature, which rises suddenly in Pneumonia, but in the continued fevers rarely attains its maximum before the sixth or seventh day. At the later periods the physical signs of consolidation of the lung on the one hand, and on the other the appearance of the characteristic rash of the continued fevers, are sufficient to prevent mistakes. The existence of herpes may also serve as a clue to the nature of the affection, being very common in Pneumonia, while it is scarcely ever met with in the course of the continued fevers.

Pneumonia commencing with cerebral symptoms in children may be easily overlooked, particularly when it affects the apex of the lung.

Ziemssen has remarked that *tubercular meningitis* rarely gives at the outset the high temperature of acute Pneumonia. The remissions are also more marked. They are more variable in their extent, sometimes showing a range of temperature of 1·8°, 2°, or even 3° Fahr., and the pyrexia is less continuously maintained. Some differences also in the character of the nervous symptoms have been already alluded to.

The diagnosis of the *different forms of consolidation* rests upon no absolutely reliable signs.

The stages of *gray hepatization* and of *suppuration of the lung* cannot be determined absolutely by the duration of the disease.

The prune-juice diffluent sputa, which were thought at one time to be characteristic of the former, have been shown to be by no means pathognomonic of this state, though their appearance affords strong ground for suspecting its presence.

It may, however, be strongly suspected when the amount of sputa is much increased, and when, instead of being rusty and tenacious, they become profuse, diffluent and puriform, and still more so when they are fetid and offensive. Pro-

[1] Dr. Walshe also affirms that Pneumonia having this position is commonly, but not always, tuberculous (loc. cit. p. 497).

[1] See Dr. Grimshaw, Thermometric Observations on Pneumonia; Dublin Quart. Journ., May, 1869.

traction of the period of resolution, attended by coarse metallic râles in the chest, and by extreme prostration, pyrexia and delirium, afford, together with the signs derived from the sputa, additional evidence of this condition of the lung.

The diagnosis of *abscess* can only be made when the expectoration of puriform matter is sudden and copious. The detection of elastic fibres in the sputa affords a further proof of its existence. *Gangrene* can only be suspected when great prostration, together with extreme fetidity of the sputa, occur late in the disease; the only positive proof of its existence depends on the discovery of débris of the pulmonary tissue in the sputa, but in these, elastic fibres are seldom distinct.

THE PROGNOSIS of Pneumonia in relation to its general mortality has proved to be the same insoluble problem that Andral[1] pronounced it, when he drew attention to the fact that the death rate in different statistics varied from 33 to 2 per cent. The difficulty has, however, still further increased in later years by the varying and contradictory statistics of the result of the different methods of treatment adopted for the disease. The results attained by Dr. Bennett,[2] who in 129 cases, of which twenty-four were complicated, had the good fortune to see all recover except four which presented serious complications, are so singularly favorable that they might lead us to regard the disease as less dangerous than it sometimes proves to be. Even in young male subjects of previously good health, Pneumonia may, as I have seen, sometimes falsify the hopes entertained from the relatively small mortality of such cases; and my own hospital experience has yielded a much greater proportion of fatal results than Dr. Bennett's, though the general methods of treatment have been very similar to his. In fifty-five cases which I have observed or collected from the case-books of University College Hospital, and the North Staffordshire Infirmary,[3] I have met with eight deaths, but in all these the attendant circumstances of the disease were such as fully account for the mortality.

One was in an infant of five months, in whom the whole of one lung had passed by the fifth day into a state of gray hepatization.

One was in a young female, where the Pneumonia was apparently developed under the influence of some intense blood-poisoning, being complicated with parotitis occurring on the ninth day, and where there were also albuminuria, pericarditis, and constant vomiting, dysenteric diarrhœa, and a petechial eruption under the skin, which latter in some places passed into large vesicles filled with a dirty-looking blood-stained serum, and where also disseminated spots of a gangrenous character were found in both lungs.

In two other cases there were old-standing renal disease and recent pericarditis. In one, an old woman, the bronchi were calcified, and there was extensive pleuritic effusion on the opposite side and thrombosis in the pulmonary artery.

Two others also presented extensive double Pneumonia: in one, a man aged 60, there was also an adherent pericardium and a fatty heart. The other, a young man, had been a hard drinker, and was suffering from syphilis.

In the remaining case there was also general bronchitis with emphysema, and the whole of one lung was in a state of gray hepatization.

Pneumonia, when extensive, certainly carries with it conditions which may prove fatal whatever the treatment adopted. It may kill by the intensity of the cause in which it originates, or by the secondary lesions to which this may give rise, particularly in the pericardium and in the kidneys. It may prove fatal by asphyxia, especially when the affection is double, or is complicated by old-standing emphysema, by extensive general bronchitis, by œdema of the lung, or by pleuritic effusion of the opposite side; and finally, and particularly in elderly people of weak constitution, death may take place in spite of the most energetic restorative measures, and when no previous lowering treatment has been adopted, in the prostration following the crisis, which may pass into fatal collapse.

It must be remembered, also, as stated under the etiology of the disease, that the mortality varies greatly in different years under the same methods of treatment. This is seen markedly in Huss's statistics, where, under an "antiphlogistic" treatment, the relative numbers of 9·1 and 14·1 per cent. may be observed; and after this plan had been abandoned the mortality in different years may yet appear as 6·1 and 13·4 per cent. The returns from the Julius Hospital of Würzburg[1] for the triennial periods of 1854–7 and 1857–60 show a similar difference; the mortality in the former period being 11·2 per cent., and in

[1] Cours. de Path. Méd. 1836, i. 386.

[2] The Restorative Treatment of Pneumonia, 1866. A very similar result is recorded by Dr. Waters, Dis. of Lungs, p. 87, who in forty-four uncomplicated cases only met with one death.

[3] I do not present these as statistics of these hospitals, as I cannot feel sure that they embrace all the cases admitted.

[1] Bamberger, Wien Med. Woch. 1857, No. 5; Roth, Würzb. Med. Zeit. i. Nos. 3 and 4.

the latter 18·9 per cent., the conditions of treatment in both periods being very similar. Brandes,[1] in Copenhagen, found the mortality in two successive years vary to the degrees of 5·4 and 31·0 per cent. The same fact is borne out by the returns of the Registrar-General before alluded to.

The most important etiological conditions which influence the mortality of Pneumonia are the age of the patients, their previous health and habits of life, their sex, the extent of the disease, and, to a less extent, its seat and the existence of complications.

Age.—It was formerly thought that the Pneumonia of infancy and childhood was an excessively fatal disease,[2] but these statements rested probably in the first place on the confusion between Pneumonia and collapse of the lung, and in the second in no small measure on the severe antiphlogistic treatment then adopted. Strangely in contrast with this belief is the remark by Barthez and Rilliet, that the opportunities for post-mortem examination in the acute lobar Pneumonia of children are excessively rare.[3] The statistics of Ziemssen and Steffen bear out these assertions. The former, out of 201 cases of Pneumonia in children, only lost seven in the acute stage. In four others the recovery was imperfect, and two of these died, giving a total mortality of less than 4½ per cent. Steffen, in 94 cases, lost 13.[4] It would appear from his tables that the mortality is greatest in early childhood, since nine of these were under three years of age.

The period of dentition, though showing from the results of Steffen a greater mortality than the later years of childhood, does not in Ziemssen's opinion unfavorably influence the prognosis, if *all lowering treatment be withheld*. This state tends, however, to be accompanied by a higher degree of pyrexia and by more severe cerebral symptoms.

After the period of childhood the mortality remains comparatively low until the age of 30 is attained, but after this it rapidly advances with each succeeding decade;[1] so that Mark D'Espini's statement may be regarded as approximatively true, that in more than half the patients dying of Pneumonia the age exceeds 50;[2] while Prus showed that in 129 cases whose age exceeded 60, 77—or 59 per cent.—died,[3] and Hourmann and Dechambre[4] give a nearly equal proportion.

Sex.—Pneumonia is a more fatal disease to females than to males. Huss gives the relative mortality as, males 10 per cent., females 14 per cent. The returns from Vienna show the mortality to be as 2 to 3 in the male and female sexes, so that although Pneumonia is a less common disease in the female sex it is proportionately considerably more dangerous. The disease also in the female sex appears to be more protracted, showing an average duration of three days in excess of that observed in the male, in the cases which recovered. Females are also, according to Huss, more liable to double Pneumonia than males. His tables also appear to show that the mortality in the female sex is less influenced by age than in the male.

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[1] Virchow's Archiv, xv. 213. Brandes very properly solves part of this enormous difference by the explanation that the higher mortality was due in the latter instances to the patients with delirium tremens admitted under his care. The number of cases entered in the two years were respectively 55 and 87, and out of the 27 fatal cases in the latter period, 12 were instances of delirium tremens complicated with Pneumonia; five others were cases of typhoid fever with Pneumonia, and in five more, complications with "organic cardiac disease" were present.

[2] Thus Valleix (Mal. des Enf. nouveaux-nés, pp. 45, 47, 70) says, that of 128 cases collected by Vernois and himself, nearly all died.

[3] Mal. des Enfants, i. 515. Barthez and Rilliet (ib. p. 535) say that in hospital they lost one-seventh, and in town practice one-eighth of their patients, but this remark appears to apply to primary and secondary Pneumonias collectively. The previous health and earlier treatment of the patients in private practice would appear to be sufficient to explain the difference. They distinctly refer the deaths of some of their patients to "poisoning" (*sic*) by tartar emetic. Barthez (Bull. Akad. Med. 1862, vol. xxvii. p. 676) gives a further report on this subject, stating that among 212 children aged from 2 to 15, the subjects of Pneumonia, only two deaths occurred.

[4] Some of Steffen's cases were secondary to measles, scarlatina, and variola. Others were complicated with other diseases. Of his uncomplicated cases, 88 in number, he only lost 7.

[1] Among the mass of statistical evidence on this subject, the following table from Huss (p. 93) gives probably the most reliable data:—

Age.	No. of cases.	No. of deaths.	Percentage
5—10	9	1	11·11
10—20	229	14	6·11
20—30	1041	61	5·85
30—40	816	97	11·88
40—50	363	72	19·83
50—60	127	27	21·60
60—70	29	7	24·13
70—80	4	2	50

[2] Ann. d'Hygiène et Méd. Leg. 1840, xxii. p. 50.

[3] Mém. Acad. Méd. 1840, viii. 13.

[4] Arch. Gén. xii. 28.

Certain conditions peculiar to the female sex appear to add to the dangerous characters of Pneumonia in them, though these are scarcely sufficient to explain the whole of the relative difference.

The condition of pregnancy appears to render Pneumonia peculiarly dangerous. Eight out of 18 cases collected by Grisolle proved fatal, and this author remarks that abortion is more liable to occur in its course than in that of any other acute disease, with the exceptions of variola and cholera. Pneumonia occurring in the puerperal state has also an extreme gravity. Menstruation, according to Grisolle, increases the intensity of Pneumonia occurring during this period, though without necessarily adding to its mortality. The state of chlorosis, according to Huss, appears in some degree to afford a protection against Pneumonia, but imparts to it when present an element of additional danger.[1]

The extent of lung affected increases, *cæteris paribus*, the gravity of the affection in a manner which may be readily understood, though a limited area of inflammation may, when unfavorable complications exist, prove equally dangerous. Double Pneumonia must, *à fortiori*, be always regarded as a source of very serious danger from the extreme impediment to respiration involved by it, the mortality from this condition being by universal consent regarded as double that of the unilateral disease.[2]

Pneumonia of the apex was, especially by the authors of twenty years ago, regarded with peculiar distrust.[3] Grisolle states that the mortality in patients so affected, and under 40 years of age, is, when compared with that of the base, as 5 to 3. Louis[4] regarded it as one of the elements of the increased mortality in the aged. Barthez and Rilliet speak of it in children as being especially liable to be associated with dangerous cerebral symptoms. Ziemssen[5] also, and Gerhardt, although recognizing the comparative frequency of nervous disturbance attending Pneumonia of this site in children, do not regard it as being ultimately of unfavorable augury. Some doubt, however, still exists regarding its specially unfavorable character in adults.

The occurrence of gray hepatization is of very unfavorable significance. Huss states that one-third of the patients perished in whom its presence could be probably presumed. It usually, at least when occurring early, signifies a more rapid progress of the disease and a weaker resisting power of the individual. In the later stages it implies defect in the restorative powers which conduce to resolution.

Gangrene in the course of acute Pneumonia is of very serious augury. Of twelve cases occurring in Huss's practice only two recovered.

Sestier[1] and Briquet[2] both thought that Pneumonia was more dangerous in *cold seasons*. Grisolle disputes the validity of these data, and points out that in Briquet's cases a large proportion of the mortality was due to the advanced age of the patients, and concludes that season has but little influence in any other respect. Huss's statistics, however, show the remarkable fact that though Pneumonia is less frequent in the last half of the year, yet that the mortality during this period is by far the greatest, in the proportion of 17·6 per cent. for the later six months to 12 per cent. in the earlier, while the excess during the last half prevails during each individual month. The contrast is still greater for some months: April, which yielded 355 cases, showing only a mortality of 8·7 per cent.; while August, with only 113 cases, had a death-rate of 25·6 per cent. The cases occurring during the hot months also presented greater severity, a condition considered by Huss to be partly due to the liability during these to gastro-enteric catarrh, and also to a larger consumption of alcoholic fluids at this season.[3]

Previous attacks do not *per se* increase the danger of the disease. The more advanced ages at which later attacks may occur in adults, do, however, somewhat increase their risk.

It was at one time thought that Pneumonia was most dangerous in robust individuals; but Huss's statistics have most clearly disproved this, and show that the most dangerous forms of the affection, both clinically and pathologically, occur in patients of weakened constitutions.

Of all conditions, however, which, independently of other circumstances, impart

[1] Of twenty-five cases of this complication met with by Huss, five, or 20 per cent., died.

[2] See Grisolle, loc. cit. Huss (loc. cit.) gives as the collective mortality — double Pneumonia, 22 per cent.; right unilateral Pneumonia, 9 per cent.; left ditto, 7·9 per cent. Huss's tables show further, in respect to age, that while double Pneumonia is most common from 20 to 30, the mortality from it is greatest from 40 to 70. The mortality from double Pneumonia appears to be about equal for both sexes.

[3] Chomel, Dict. de Méd. xxv. 158. In 55 deaths he found 13 of the upper lobe, 11 of the lower, and 31 of an entire lung.

[4] Rech. sur les Effets de la Saignée, 42.

[5] Loc. cit., 211.

[1] Chomel, Léc. Clin. Méd., Pneumonie, p. 455.

[2] Arch. Gén. de Med. 3e Sér., 1840.

[3] The returns from the General Hospital of Vienna show that in some years the mortality is greatest in the winter months.

a special danger to the disease, *habitual drunkenness* must be regarded as one of the most serious. The mortality from Pneumonia under these conditions is nearly double that ordinarily observed, amounting to 20 or 25 per cent.[1]

The existence of *complications* forms the most serious element in the prognosis, and most largely influences the mortality of the disease. This is sufficiently apparent from Huss's collected results, where the mortality of the non-complicated cases was only 5·79 per cent., while that of the complicated cases amounted to 19·29 per cent. The relative danger of the various complications, as observed by him, will be best seen in the table before quoted (see p. 184). It is, therefore, only necessary here to remark that of the most ordinary of these the greatest mortality occurs in the presence of endocarditis (75 per cent.); pericarditis (54·5 per cent.); Bright's disease (50 per cent.); old valvular disease of the heart (30 per cent.); tubercle (33·3 per cent.); emphysema of the lung (23 per cent.); chlorosis (20 per cent.); and chronic alcoholism and drunkenness (25 and 20 per cent.). The danger is proportionately increased if more than one complication occur in the same patient. It may further be noticed that certain complications, such as rheumatism and erysipelas of the face, do not appear unfavorably to influence the general course of the disease, while both bronchitis and pleurisy do so to a less degree than might be *à priori* believed.

An extreme degree of pyrexia is considered by many an unfavorable sign. Wunderlich regards a temperature of 104° Fahr. as the limit of mild cases. It must, however, be remembered that cases may prove fatal in which this temperature is never attained.[2] Wunderlich regards a gradual rise taking place after the fourth day as a very unfavorable symptom.[3] The rapidity of the breathing has less influence on the prognosis than that of the pulse, but a very rapid respiration associated with a low temperature is pointed out by Wunderlich as indicative of danger. Irregular respiratory movements show a severe implication of the nervous system. Excessive dyspnœa amounting to orthopnœa, particularly when associated with cyanosis, are also indications of considerable gravity.

A pulse above 120 always indicates weakened cardiac powers, but its unfavorable augury is less in young children than in adults. In the latter, a pulse above 130 or 140 is a sign of great danger, and particularly when the temperature is not markedly high.[1] Grisolle says that all his cases died in whom the pulse exceeded 150. Extreme dicrotism of the pulse has a very similar import. It has been already stated that in some cases the pulse may be markedly retarded before a fatal issue. Irregularity and intermittence of the pulse except in elderly people, in whom these symptoms are not uncommon, must also be regarded as suspicious symptoms.

Few signs can be drawn from the sputa. Those of liquorice or prune-juice tint are the more serious, but do not necessarily indicate a fatal issue. The serious import of profuse hæmoptysis has been before alluded to. Diffluent puriform expectoration when profuse in the later stages of the disease, and when associated with great prostration and persistence of the physical signs, are also symptoms of considerable gravity. Suppression of the expectoration from weakness, together with increase of tracheal râles, is of very serious augury. The total absence of expectoration throughout the disease has no influence on the prognosis.

Marked disturbances of the nervous system are always indicative of the severity of the disease. A mild degree of delirium is not uncommon in children, and also in elderly people; but in the latter it is a serious symptom.[2] In adults, however, severe delirium is always dangerous, particularly when occurring late in the disease, or when habits of drinking have preceded the attack. Convulsions, repeated after the onset of the disease, are in children a sign of much danger.

Intense prostration with sunken and pallid features, and profuse sweating, are always suspicious, and have a gravity proportioned to their degree. In the more marked forms of so-called Typhoid Pneumonia, the prognosis must always be doubtful.

Total suppression of the chlorides in the urine indicates a greater severity of the disease than when these are present, but does not, independently of other circumstances, materially affect the prognosis.

Severe gastric disturbance and diarrhœa have a very similar import. Their effect is certainly unfavorable, but it can

[1] Huss, loc. cit.

[2] This is further confirmed by Griesinger. Of thirty fatal cases the temperature only reached or exceeded 104° in eight. A temperature of 105·2, occurring in only one instance, was the maximum attained among these fatal cases (Bleuler, loc. cit. p. 33.)

[3] Die Eigenwärme in Krankheiten.

[1] Bleuler, loc. cit. Of adults with a pulse above 120, one-third died whose age did not exceed 40; over ætat. 40 one-half died; over 60 all died.

[2] Bleuler (loc. cit.) observed a mortality of one-fourth of patients under ætat. 40 who exhibited marked delirium; over 40, three-fourths of these died.

only be judged of in relation to the general strength of the patient. Icterus does not necessarily increase the gravity of the prognosis.[1]

A protracted defervescence with a high pyrexia are also unfavorable. The liability to relapse in the early days succeeding the crisis should also impose caution against a premature confidence in the cessation of danger.

The terminations in a chronic state are so excessively rare that they hardly form an element in the consideration of ordinary forms of acute Pneumonia. The possibilities of a more protracted course are sufficiently shown in the previous account of the ordinary progress of the disease.

The occurrence of herpes appears from the researches of Griesinger[2] and Geisler[3] to have a favorable prognostic signification.

TREATMENT.—There is, perhaps, no subject in modern medicine which has been more earnestly discussed of late than the treatment of Pneumonia. It has been the *champ de bataille* between the advocates on the one side of "heroic" measures, and the supporters of a "rational" and of "expectant" treatment on the other; and since the first-named methods have been, to a large degree, shown to be worse than useless, the question has become further complicated by the assertion that a change of type has ensued, by which the nature and characters of inflammatory diseases in general have been, during recent years, materially modified.

When, however, the natural course and the various relations of this disease are attentively considered, it is apparent that no malady can well be chosen less suited to afford logical proof, by means of statistics, of the relative value and the curative effects of any system of treatment applied indiscriminately, though the reverse is more easily shown by the enormous excess of mortality which prevails when an "active" treatment is universally employed.

An acute disease with a natural tendency, under favorable circumstances, to terminate spontaneously by a sudden crisis occurring at periods varying from the 3d (or even the 2d) to the 7th or 11th days, presents the most singular elements of fallacy in reasoning from the beneficial effects of active medical interference. If to these we add the manner in which its mortality is affected by age, by constitution, by sex, by the presence or absence of complications, and by the other but unknown epidemic conditions which have no slight effect in the same direction, it would appear a task of the extremest difficulty to collect sufficient data in order to institute a logical comparison between patients under similar circumstances of the disease, but under different systems of treatment, so as to form any true conclusion as to the relative value of the methods to be adopted for its cure.

Looking to the evidence of statistics, and to the individual experience of careful observers, it must be admitted that medicinal interference and active treatment are, collectively speaking, of but little influence, either in shortening the duration in, or diminishing the mortality of, Pneumonia. Treatment, in its wider sense of nursing, diet, support, and remedies adapted to individual cases, is, however, the author believes, by no means inefficacious in aiding the tendency of nature to effect a cure.

The remedy which has been most extensively adopted, but which has been almost completely discarded of late in this country, is *venesection*.

Reintroduced by Sydenham[1] as applied both to pleurisy and pneumonia, with the statement "Hujus morbi curatio in repetita venæsectione fere tota est," and supported by Huxham and Cullen, the amount of blood taken by these authorities and their followers in the treatment of this and kindred disorders was enormous. Day by day, with the progress of the disease, fresh venesections were practised, and Dr. Gregory, after bleeding a young man into convulsions by the abstraction of between 4 and 5 lbs. of blood in three days, considered that he had thereby cured him of pleurisy.[2] Bouilland recommends a daily bleeding to the amount of 14 or 16 oz. until the disease is cured. Andral asserts that no period of the disease contraindicates venesection, provided the other symptoms appear to require it, and that age is no barrier to this treatment, citing in its favor at advanced ages the authority of Frank,[3] and that it is to be applied to children equally with adults: the slightest threatening of a relapse called in his opinion for further bleeding: it is not to be omitted without the greatest danger, even if menstruation be present: profuse sweating is no contraindication, nor is any amount of prostration to prevent it, if the respiration be seriously impeded:[4] it is to be equally

[1] This is the almost universally adopted opinion. Bleuler, however, records a mortality of 35 per cent. of cases in which icterus was observed.

[2] Arch der Heilk. 1860, vol. i.

[3] Ibid. 1861, vol. ii.

[1] Works, Syd. Soc. Ed. p. 352.

[2] Quoted by Dr. Alison.

[3] Grisolle similarly quotes Morgagni (Epist. xx.), who bled a man over 80 with "success."

[4] On this point he quotes Stott, who bled for the eighth time a patient covered with petechial eruption.

practised in the secondary pneumonias of measles, variola, and scarlatina, though with caution in typhoid fever: it is only contraindicated in the adynamic forms of the disease, and in some rare cases of special idiosyncrasy, and in the absence of expectoration. Grisolle, even for more moderate bleedings, recommends the abstraction of from 2 to 4 lbs. by repeated venesections, and still regards this plan as the most successful in the treatment of the disease.

The treatment thus indicated continued in use with more or less freedom in this country until attention was forcibly drawn by Dr. Balfour[1] to the lesser mortality of Pneumonia in Skoda's practice, and also in some of the homœopathic hospitals where bleeding had been for some time discontinued. Even before this period Becquerel[2] had shown the inutility of venesections in the pneumonia of children, and it is stated, on the authority of Legendre,[3] that Biett and Magendie had pursued an expectant treatment in Pneumonia with excellent results. Dr. Graves[4] had also asserted that the large bleedings practised by some physicians were unnecessary, and that repeated venesections were injurious in the disease; but the statistics of Skoda's practice showed for the first time the striking contrast between the "heroic" and the "expectant" methods; for while the mortality in 384 cases treated by him with small doses of *extractum graminis* and nitre was only 13·7 per cent., that of the Edinburgh Infirmary during a nearly corresponding period of five years was 35·9 per cent. Dietl's[5] comparative statistics, which appeared almost simultaneously with Dr. Balfour's papers, seemed still more forcibly to bring into contrast these systems of treatment, and may be said to have at once exercised an important influence on medical opinion both in this country and in Germany.

The arguments against bleeding have subsequently been most vigorously supported by Dr. Todd and Dr. Bennett, who have at least the merit of showing that the treatment by venesection is in most cases unnecessary, and that in a very large proportion it is positively injurious, and the same conclusions have been more or less completely adopted by the majority of the physicians of the present day.

The conclusion which has been practically arrived at by the medical profession with respect to the influence of venesection in Pneumonia may be, with approximative truth, expressed in the following terms:[1]—

(1) That indiscriminate bleeding immensely increases the mortality of the disease.

(2) That it is especially fatal in old people and in young children, in patients of exhausted constitutions, and in those suffering from chronic diseases, and particularly from Bright's disease.

(3) That it is absolutely unnecessary in the majority of cases of young adults and also of young children.[2]

(4) That in the vast majority of cases it has no influence whatever either in cutting short the disease,[3] or in lessening its duration, or diminishing the pyrexia, but that occasionally these results appear to follow from its use when practised early.

(5) That in the majority of cases it hinders the critical fall of temperature and delays convalescence.

(6) That in the majority of cases, as shown especially by Dr. Bennett's and Dietl's data, recovery is equally, if not more rapid, when it is not practised as when it is resorted to.

(7) That in a few cases a moderate venesection may be necessary in the early stages to avert immediate danger of death from asphyxia.

Individual cases where apparent success has followed venesection are really but little capable of proving its general utility. It is a treatment to which I have never but once resorted, and have very rarely seen practised, and I can certainly affirm that those cases which may occasionally offer the strongest temptation to the use of the lancet recover just as well when it is withheld. The mortality among the cases which I have mentioned as coming under my own observation, has certainly been in such as would not, with any modern knowledge, have been considered fit subjects for venesection. Even the relief of dyspnœa, which is thus effected, is proved by universal consent to be only temporary in its duration, for this symptom usually results more from attendant œdema of adjacent portions of the lung than from the actual obstruction to respi-

[1] Notes in the practice of Skoda, Edinburgh Medical and Surgical Journal, 1847, p. 397. Brit. and For. Med.-Chir. Rev. 1846, vol. xxii. p. 590.

[2] Sur l'Influence des Émissions sanguines et des Vésicatories, chez les Enfants, 1838.

[3] De l'Expectation dans la Pneumonie. A posthumous memoir; Arch. Gén. 1859, xiv. 283.

[4] Clinical Medicine, 1843, ii. 42.

[5] Der Aderlass in der Lungen-Entzündung.

[1] See Appendix F.

[2] This is especially seen in Ziemssen's treatment, and also in a memoir by Barthez, who, in 212 cases of young children with lobar Pneumonia, only met with two deaths. Barely one-sixth were subject to active treatment. (Arch. Gén. 1859.)

[3] This is most strongly affirmed by Louis and Andral, and also illustrated by the case by Zimmermann before quoted. Chomel (Dict. de Méd. xxv.) held that it might sometimes effect this.

ration in the part affected by the primary disease unless this be very extensive; and as the amount of fluid withdrawn by venesection is speedily replaced by the absorption of water, the pressure on the collateral circulation of the lung is thereby only very temporarily diminished. It was, however, to this cause that the repeated venesections of former times were probably due, a system whose impropriety it is scarcely needful to discuss further.

Its employment in severe pyrexia is also shown by Ziemssen to be unnecessary, for though he admits that he has occasionally resorted to its use when the temperature has appeared dangerously high, he yet states that other cases of a similar nature recover equally well without it. I have already stated that the fatal cases which have come under my own observation have not in any instance presented this phenomenon.

When we consider, therefore, that the most urgent symptoms of the disease—the dyspnœa and the pyrexia[1] are only temporarily diminished by venesection, and that they both tend in most cases to return after a few hours, the reasons for the adoption of this method of relief lose much of their validity.

It may be possible that cases of such extreme urgency may occasionally arise that venesection may be absolutely necessary to avert immediate death by apnœa. Such cases I must believe, however, judging from my own experience, to be excessively rare; though, in the event of their occurrence, this remedy is probably the best that could be adopted, and should not be shrunk from if the indications are urgent, but I believe that such a condition is the only one in which it can be regarded as absolutely necessary. The mortality from Pneumonia has appeared to me to depend much more on prostration in the later periods than on asphyxia in the earlier stages of the disorder; and the former result appears to be much more likely to occur when the strength of the patient is weakened by venesection. If, therefore, venesection appears to be positively required at an early period of the attack, the amount of blood withdrawn should be moderate, and should not exceed six or eight ounces.

With regard to the possible effect of this treatment in cutting short the disease, it may be stated that the chances in any given case are strongly against such a result. Looking at the general effects of this procedure, patients will, on the whole, be probably in a worse condition for passing through the later stages of disease when weakened by an artificial loss of blood than they are likely to be if their resources in this respect are husbanded: and though its dangers are the least in the case of young adults of good constitution who commonly "bear" bleeding comparatively well, this "tolerance" of the remedy by such subjects affords no proof of its general advantageous effects. [Tolerance, however, *plus* immediate relief of marked symptoms, and early recovery, affords the kind of evidence which, according to all rules of clinical experience, is wanted to establish the appropriateness of a remedy in practice. While an individual case (*e. g.*, the one in which, as above mentioned, Dr. W. Fox resorted to venesection) can prove but little, yet the aggregate of individual cases, carefully observed, furnishes a better basis than any *à priori* reasoning can do, for conclusions in inductive medicine. What is claimed by those who still advocate moderate venesection in a certain minority of cases of Acute Pneumonia, during the early stage, is, that having resorted to it, and seen it resorted to, in a large number of such cases, relief and early recovery followed, without any drawback of excessive weakness. Their legitimate inference is, that the unmitigated pulmonary inflammation would have produced greater debility than the timely withdrawal of a few ounces of blood. Nor does this conclusion, as a matter of fact, appear to be vitiated by the comparative effects of expectant or stimulant treatment, now so common, upon the mortality of the disease.—H.]

Most of the other methods of treatment directed immediately to the cure of Pneumonia afford nearly equal proofs of their inutility.

The comparative effect of large doses of *tartar emetic* is shown by Dietl's statistics,[1] while Rasori's[2] mortality from this method was 22 per cent., and Grisolle's 18 per cent.—or in those treated exclusively by this method, 13 per cent. Independently also of this considerable mortality, the poisonous effects of the remedy were very frequently observed. Laennec spoke very highly of tartar emetic in more moderate doses, and considered that it had reduced the mortality from Pneumonia in his practice to a minimum; but grave doubts have been thrown on the accuracy of Laennec's details[3] in respect to this method. Laennec asserted and Grisolle believes that it is more useful when preceded by bleeding. Louis[4] also and Trousseau[5] speak favorably of its results, but the data given by the former, complicated as his treatment was by venesection, afford but little proof of its efficacy.

[1] See Appendix E.

[1] See Appendix E.

[2] Ann. de Thérap. 1847, and in Archiv. Gén. 1824.

[3] See Grisolle [4] Rech. sur la Saignée.

[5] Dictionnaire de Médecine, art. "Antimoine."

Regarding the statements made, particularly by Grisolle, respecting its effects in Pneumonia, it cannot be denied that tartar emetic produces occasionally a feeling of relief to the patient, and in some cases lowers the frequency of the pulse, and apparently diminishes the pyrexia.[1] This effect, however, requires to be very carefully watched. It is a depressing agent both to the nervous system and to the circulation, and is liable to increase the dangers of the later collapse. As far as my own experience goes, I believe that it is a remedy which can only very rarely prove of essential utility, and certainly, to say the least, the vast majority of patients will recover as well, if not better, without its use; and it is absolutely inadmissible in the adynamic forms of the disease, and also in the Pneumonia occurring in old people, and in most cases in children. A very rapid pulse contra-indicates its use, and it is highly dangerous in most forms of the delirium accompanying the disease.

Calomel, with or without opium in combination, has also fallen into disuse, probably not without reason. Experience has gradually demonstrated the minor degree of power which it was at one time supposed to possess in aiding the absorption of exudations, and no valid proof has been afforded that the duration of Pneumonia has been shortened by its use. By most of its advocates it was only held to be efficacious after the previous employment of venesection and the administration of tartar emetic; and a remedy requiring such antecedents is one that may with advantage be abandoned. Even when resolution is delayed, the final termination of the disease is not, in most cases, less favorable; and I should not only feel extremely sceptical as to the value of mercurials in accelerating this process, but I should greatly hesitate to interfere with a remedy which often so materially impairs the general health and nutrition of the patient.[2]

Iodide of potassium has also appeared to me to exercise little or no influence in promoting resolution.

The methods of treatment by *alkalies*,[3] or by *acetate of lead*,[4] *copper*,[5] and *chloroform*,[6] introduced in more recent periods, only serve to show that Pneumonia is a disease little influenced by remedies; that the less "active" these are, the better for the patient. Chloroform inhalations may certainly relieve the cough and allay the discomfort of the patient, as Dr. Walsh has stated, but they appear to have no influence on the progress of the disease.

Digitalis, which was used by Rasori, has recently had an extensive trial, both by Thomas[7] and Ziemssen.[8] This remedy, from the researches of Traube[9] and Wunderlich,[10] seems to have a distinct efficacy in reducing the pyrexia in typhoid fever. It would appear, however, from Thomas's observations, that at periods antecedent to the crisis (except in a few cases, when a marked lowering of the temperature and of the frequency of the pulse is observed) this effect is much less distinct in Pneumonia, but when given in the later stages it tends to increase the post-critical fall to an abnormal degree.[11] Both in adults and children it produces at times intermittence of the pulse, which, however, Ziemssen regards as not intrinsically dangerous. Duclos and Hirtz,[12] who have also used it, give the alcoholic extract in divided doses, to the extent of 3, 6, or 10 grains daily. Ziemssen gives ʒj of an infusion made with gr. v. to the ounce of water every two hours (the infusion of the British Pharmacopœia is made with gr. iij to the ounce of water).

Veratria, introduced by Aran,[13] has been tested by several subsequent observers[14] with varying results. A more extensive trial of this remedy by Kocher[15] appears to show that in certain cases favorable results may attend its administration in diminishing both the pyrexia and also the frequency of the pulse. In some instances the temperature may be reduced by its use to the normal standard, though in many instances this effect is only temporary, but

[1] Accurate thermometrical observations on this point are wanting.

[2] Wittich has published a series of twenty-three cases thus treated, and without fatal results. (Canstatt's Jahresb. 1850.)

[3] Mascagni, quoted by Grisolle.

[4] Leudet, Bull Thérap. 1863, a mortality of 7 per cent.

[5] Kissel, Canstatt's Jahresb., 1852, a mortality of 4 per cent. All Kissel's cases do not appear to have been thus treated.

[6] Baumgartner, Wucherer, and Helbing, Canstatt's Jahresb. 1850; Varrentrapp, Henle and Pfeufer's Zeitsch. N. F., 1851, analyzed in Schmidt's Jahrbücher, lxxiii. 20. The treatment in some of these cases was mixed.

[7] Arch. der Heilk. 1865.

[8] Loc. cit.

[9] Annalen der Charité, i. 691.

[10] Arch. der Heilk. iii.

[11] The effect on the pulse also appears to be uncertain, and a marked lowering of the pulse may ensue without any fall of temperature, though the latter is never observed without the former. Occasionally the reverse effect is observed, and great acceleration of pulse may take place with or without a *rise* of temperature.

[12] Bull. Thérap. vols. li. and lxii.

[13] Ibid. xlv.

[14] Vogt, Schweitz. Monatsch. vi., and Bull. Thérap. 58; Fournier, Union Méd., 1855: Roth, Würzb. Med. Zeitsch. iii. 1863; Uhle, Arch. der Heilk. N. F., iii.

[15] Die Behandlung der Croüposen Pneumonie mit Veratrum Preparaten. Würzburg, 1866.

lasting in others for sixteen hours. In some, however, it appeared to accelerate the period of the crisis, and Kocher is of opinion that it also shortens the duration of the process of resolution; while in a few cases, when given early, it appeared to cut short the disease, and to prevent the occurrence of consolidation. The temperature is commonly affected before the pulse, but in a few cases these phenomena did not coincide; and either the pulse or the temperature may be affected singly and without any corresponding reduction in the other.

The remedy, however, appears in some cases to cause both vomiting and diarrhœa, and to produce, when given in the later stages, a dangerous amount of depression. For this reason Kocher recommends that its effect should be most closely watched, and it appears also desirable that it should only be given in the earliest periods of the disease. The veratria, as an alkaloid, can only be safely given in doses of one-twentieth of a grain, and should be administered in pill, the resin in doses of gr. $\frac{1}{6}$.[1] Kocher recommends that it should be given in frequent doses at intervals of from one to two hours, until a distinct effect has been produced upon the pulse and temperature.

In very severe cases he considers that its good effect is increased by venesection. Dessauer, however,[2] who has also used this remedy and speaks highly of its effects, regards venesection as unnecessary, and believes that veratria is a complete substitute for bleeding. He considers that no prejudicial effects attend the diarrhœa which it commonly produces, and he says that delirium usually disappears under its influence.

Aconite as a remedy does not appear to have been tested sufficiently to afford a proof of its effects in Pneumonia. In one or two cases in which I have given it I could not observe that any effect was produced by it on the temperature.

The treatment which has hitherto been shown to have the most marked effect on the pyrexia consists in *the external application of cold water to the body*. Tepid baths had been indeed, as Grisolle shows, recommended by Hippocrates, and used by others; and Grisolle himself speaks favorably of their effects in relieving pain and also the general distress of the patient. The use of cold water, though recommended by Currie in fevers, does not appear to have been employed by him in Pneumonia, but it has been largely used by the followers of Preissnitz.[1] It was further introduced into modern practice by Dr. F. Weber,[2] of Kiel, and has been highly praised by Ziemssen, both in the lobar and lobular Pneumonia of children, and by Niemeyer[3] in that of adults. Its effect during the pyrexial period only lasts during, or for a short time after, its employment, and it often requires a prolonged application to effect any marked lowering of the temperature. The reduction of the temperature also by this means appears from Ziemssen's observations to be rarely so marked as in the form of Broncho-pneumonia, and seldom appears to exceed $1\frac{1}{2}^{\circ}$ or 2° Fahr. It appears, however, simultaneously to reduce the frequency of the pulse and of the respiration; and though often unpleasant at first, it seldom fails to afford great relief to the patient, and to produce quiet sleep. The method adopted by Niemeyer is that recommended by Weber, of applying compresses wrung out of cold water, and changed every five minutes, to the chest, and especially to the affected side. Ziemssen recommends the employment of Esmarch's ice-bag,[4] covered with linen, for the same purpose.

In a few cases in children this treatment appears, as also in the form of Broncho-pneumonia, to produce a depressing effect, and it therefore requires to be carefully watched, but it does not appear to be attended with any other risk, either of exciting bronchitis or of setting up secondary complications.[5] It does not ap-

[1] Kocher has found that the tincture of the veratrum viride contains very variable amounts of the alkaloid veratria.

[2] Oesterreich. Zeit. Prakt. Heilk. and Schmidt's Jahresb. 1866, cxxxii.

[1] Schedel, quoted by Grisolle.

[2] Beiträge zur Path. Anat. der Neugebörenen, ii. 63. Weber says that this method was first recommended to him in 1837 by Dr. Niessen, of Altona. Grisolle, p. 678, says that it was also recommended by Dr. Campagnano, of Naples, who revived patients *in extremis* by cold baths. Grisolle states that Campagnano also employed bleeding and antimony "*avec une vigueur presque barbare.*"

[3] Spec. Path. Therap. i. 182. Niemeyer states that the treatment has been most extensively used in Prague, with good results. He says that under this treatment cases of Pneumonia rarely last beyond the seventh day; that in an extraordinary number the disease terminates on the third day.

[4] Langenbeck's Archiv für Chirurgie, ii. 275.

[5] I have employed this treatment experimentally in only one mild case in a child for a few hours. The continuous application of cold cloths to the chest lowered the temperature half a degree Fahrenheit. It rose again with the ensuing exacerbation to the same height as on the previous evening (103°), after they were discontinued by the nurse, on account of the dislike of the patient to the treatment.

pear to shorten the duration of the disease, but only to act beneficially by diminishing the pyrexia.

Blisters in the earlier stages of Pneumonia are to be considered as both useless and as greatly increasing the distress of the patient. When resolution is progressing favorably, they also appear to be quite unnecessary. In a few cases when resolution is delayed, or when there is evidence of a small amount of pleuritic effusion, they may, I believe, in adults be occasionally employed with apparent advantage. In children they are almost invariably inapplicable. Warm fomentations or poultices to the side often give great relief to the pain. I have by no means satisfied myself that any advantage accrues during the acuter stages from any more irritant applications, whether of mustard or turpentine, though in cases of threatening collapse, or when dyspnœa is severe, they have occasionally appeared to afford relief.

It may, however, be desirable that after the foregoing analysis some account should be given of the treatment of Pneumonia which is most in accordance with the result of modern observation.

The author, in commencing this branch of the subject, feels it right to express his conviction that a large number of the milder cases occurring in young adults require no more medicinal interference than similar cases of other acute febrile disorders, and that neither depletory measures nor alcoholic stimulants are necessary to bring such cases to a satisfactory termination.

Rest in bed; a free supply of fresh, but not too cold, air; attention to the evacuations, and the administration of a sufficient amount of liquid, nutritious, and easily digestible food—indications abundantly fulfilled by milk and beef-tea—are often all that is requisite. [Cold and fresh air will be better for the patient than that which is warm and impure. In two very severe cases I have met with a craving for air from open windows, when the weather was very cold. One of these patients was a man about thirty-five years of age. On being called to see him in the midst of his attack, I found him lying with his two windows wide open near his bed, the thermometer indicating 17° Fahr. When I attempted to close one of the windows, he made known at once his distress for want of air. Continuing, with reluctance, this aerial refrigeration of his lungs, his recovery suggested the thought, that, to the inflamed pulmonary tissue, possibly a direct "apyretic" influence may have been thus extended, similar to that of cold applications to a superficial inflammation. Almost precisely the same observation occurred to me afterwards in the case of an old lady more than eighty years of age; who manifested a craving for the admission of cold winter air through her windows. She also recovered, under that exposure, from a very severe attack of broncho-pneumonia.—H.] Pain may be assuaged if severe by a few leeches to the side, by linseed poultices, and more effectually by the hypodermic injection of morphia. Sleep also may be procured by the same means, or by moderate doses of opiates, or probably by the hydrate of chloral.[1] When cough is distressing, and opium is not contra-indicated by cyanosis, this remedy in small doses has appeared to me to give much relief, and to have no injurious effects. Neutral salines also favor the action of the skin, and thus reduce the discomfort from the pyrexia, and probably aid in the elimination of effete matters by the urine. If any extensive bronchitis be present ammonia may with advantage be combined with these, and small doses of ipecacuanha have also under these circumstances appeared to me to be useful. When convalescence is established, solid food and a moderate use of stimulants adapted to the strength and habits of the patient, are frequently all that is necessary to promote a rapid cure. Iron and quinine or strychnia are, however, to be given if there be anæmia or much weakness remaining.

In severe cases of Pneumonia, threatening to invade a large tract of lung, and coming under observation within the first forty-eight or seventy-two hours of the disease, and if the dyspnœa threatens asphyxia, and the distension of the superficial veins indicates overfilling of the right side of the heart, a cautious bleeding may probably be practised with advantage to the extent of six or eight ounces, particularly if the patient be young and vigorous, and of previously temperate habits.[2]

[1] "A close, narrow, stifling room is exceedingly incommodious to any person sick of a fever, but much more so to those ill of a peripneumony, as I have many times observed, especially among the lower part of tradesmen when two or three families perhaps live in a house together. Celsus's advice is never more proper, nay necessary, in any kind of fever than in a peripneumonia, *in amplo conclavi tenendus æger*. If such close rooms cannot be avoided, they certainly should be frequently but prudently aired." (Huxham on Fevers, 1757, 199.)

[1] I have not had a full opportunity of experimenting with this remedy in Pneumonia.

[2] Huss lays down the following rules:—Venesection may be practised when the pulse is full, tense, or depressed. The large full pulse sinks at first, but venesection is to be continued until it rises again. In patients with a "tense" pulse venesection is to be continued until it becomes soft. If the pulse is depressed, venesection is to be continued until it becomes full. The indications for

Under these circumstances also, if the fever be high, tartar emetic may be given in doses of gr. ½ to gr. j or gr. iss, combined with salines and small doses of paregoric, every hour or two hours until some relief is experienced—a relief which may be further aided by the application of leeches or cupping to the side. I think it right, however, to add here, that although I have not hitherto adopted the application of cold water in such cases, I should, after the testimony adduced in its favor by the authors before quoted, feel strongly disposed to make a trial of its effects.

Under all circumstances food must be given in suitable quantities, for it is important to husband the resources of the patient as much as possible.

Cases such as these now under consideration vary much in their later manifestations, and it is in these that judgment and decision are most required.

One complication which may be regarded as most indicative of danger is *delirium*, and it is to this symptom especially that I now refer.

By many of Dr. Todd's pupils the occurrence of delirium in Pneumonia has been regarded as a certain indication for the administration of stimulants, and I believe that in the majority of instances the practice is both well founded and successful. Cases do, however, occasionally occur when acute delirium associated with a considerable degree of pyrexia is not benefited by this treatment, and though comparatively rare, they belong to a class which requires separate consideration.

We have unfortunately but little exact knowledge of the state of the brain during delirium to serve as a pathological guide for its treatment. It is now pretty generally admitted that delirium in many cases is by no means an expression of hyperæmia or inflammatory irritation of the brain, and it is only clinical experience which has led us to the discrimination of these conditions in the various diseases associated with this symptom.

In Pneumonia the evidences, as before stated, of meningeal or cerebral hyperæmia associated with delirium are very rarely met with *post mortem;* but I believe that we may with advantage discriminate two conditions under which delirium occurs in this disease. In one the state is that of weakness, for which we have no more precise pathological expression; in the other it is the expression of a blood-poisoning by the products of the pyrexial disturbance, though not, I believe, as some are disposed to think, depending on the direct effects of overheated blood on the nervous centres. It is probable also that in many cases both these conditions are more or less combined in various degrees.

In conditions of pure weakness the reasons for giving stimulants are abundantly clear, but in delirium from blood-poisoning this is more doubtful. It is, however, by no means easy to apply any certain clinical test to distinguish these two states. Delirium with high pyrexia should always induce doubt as to its nature, and this doubt is increased when it has been preceded by severe cephalalgia. I do not think that the special characters of the delirium always afford a certain guide; at least its violence is no proof of the sthenic or asthenic character of the primary disease, though a low muttering delirium almost invariably belongs to the latter class. A correct opinion on this point must depend on the practitioner's judgment as to the state of the patient's strength; and if indications of asthenia exist, it is better to depend on this as a guide, rather than on any theoretical reasoning respecting the origin of the symptom.

The state of the pulse is, I believe, the surest indication which we at present possess. An extremely rapid pulse, *i. e.*, one above 120 or 130, generally calls for the employment of stimulants. When the pulse presents the characters of dicrotism to any distinct degree, they are almost invariably necessary, and under both these conditions the use of bleeding or tartar emetic is absolutely contra-indicated. Tremors and subsultus rarely coexist with violent delirium; when they are present, they also strongly require the remedies under discussion.

In doubtful cases it is safer to make a cautious trial of stimulants than to omit their use: when beneficial, their good effect is usually seen early.

Huss recommends the use of tartar emetic in doses of gr. j to gr. ij every hour in the delirium of drunkards, when this sets in early, accompanied by high fever and by a flushed face and tense pulse. He considers bleeding in these cases to be entirely inadmissible, and the tartar emetic is to be discontinued directly

venesection to be drawn from the pulse were repeated by nearly every writer of the early part of the present century. How little these were to be relied upon, even by those in the habit of testing their practice by this means, is apparent from the following observations of Hourmann and Dechambre, who may at least be supposed to have been conversant with the fallacy of "fulness" in the pulse of old people to whom these remarks refer: "Nous avons vu des malades chez qui le pouls *invitait* la saignée, cesser de rendre leurs crachats immédiatement après que celle-ci avait été pratiquée et mourir en moins de douze à quinze heures." (Arch. Gén. de Méd., 2e Sér. xii. 190.) Intense severity of dyspnœa appears to me to be the only positive indication for this remedy. A very high amount of pyrexia in the early stages is also so, but to a less degree.

the pulse falls in volume, or if diarrhœa or vomiting should occur. The use of all lowering remedies directed solely to the delirium is, however, only to be pursued with the greatest caution, for the diagnosis of the pathological state present is often doubtful, and their danger, when inappropriately used, can hardly be overrated.

Opium in these forms of delirium can only be used with caution. Full doses often increase the prostration, and fail to procure sleep. Huss regards the condition of the pupil as affording a valuable indication for the treatment to be pursued. If this be contracted, opium is contra-indicated, but belladonna, in doses of gr. $\frac{1}{8}$ of the extract, given three or four times daily, may induce a quieter condition, ending in sleep.

I believe that in such cases as these the value of cold applications in lessening pyrexia will be found to be very considerable when properly used, and may aid in solving the difficulty which has hitherto attended some of these cases. Digitalis or veratria,[1] when the pulse is rapid, are remedies that appear to me to be deserving of a further trial than I have yet had opportunities for making of their efficacy.

The class of cases which have now been considered are fortunately comparatively rare. In the majority the discrimination is more simple, and in the severer cases of Pneumonia the administration of stimulants in the later stages is almost invariably both useful and necessary. They are, indeed, often required almost from the outset in cases marked by debility, at whatever age, but particularly in patients of bad constitution, in those who have indulged freely in alcohol, and in old people; and under all these circumstances attention must be paid to the previous habits of the patient in regulating the amount given.

In such cases as these I believe that all depletion and the use of tartar emetic are in the highest degree injurious, though simple salines may usually be given with apparent advantage.

In the majority of cases the amount of stimulants given during the pyrexial period may be very moderate. It is, indeed, always best to begin with a minimum dose, and to increase the quantity as required; and under all circumstances it is desirable, as far as possible, to husband resources of this nature. For infants, brandy, which is the best form of alcoholic stimulant for these purposes, may be given in doses of five to ten drops, increased to thirty drops, or ʒj every two, three, or four hours. For adults, from one to three drachms may be given at similar intervals, and in a large number of cases it is seldom necessary to give more than six or eight ounces of brandy in this manner in the twenty-four hours. The indications for the amount and frequency of these doses are best gained from the pulse and from the general signs of asthenia. As long as these are distinct, stimulants must be persevered with; and though always to be used with caution, they must in some cases, especially in patients addicted to habits of intoxication, be given both unflinchingly and unsparingly when the need arises. I have in one or two instances given 36 ounces of brandy daily for several days consecutively, in doses of six drachms every half hour, with a successful result, in cases of Pneumonia in drunkards; every attempt to diminish the dose being immediately marked by dangerously increasing signs of asthenia; and it was only when the more marked evidences of prostration diminished, that any symptoms of alcoholic intoxication were observable.

Such cases are, however, rare, and, as before observed, much smaller amounts of alcohol are usually sufficient.

The period immediately following the crisis is that in which moderate doses of alcohol appear to be most called for; and in many cases which have not previously presented marked signs of asthenia, very considerable prostration, which in old people may prove fatal, may occur at this time. Indeed I believe that one of the chief duties of the practitioner in most cases of Pneumonia is to watch carefully for symptoms indicating the employment of stimulants, and to regulate by frequent observations the amount necessary to maintain the strength.[1]

[1] The lowering of the pulse by veratria is often very considerable. I have known it reduced in acute rheumatism from 100 to 54 in the minute within eight hours by the tincture of the veratrum viride, given in doses of ♏v every two hours. The influence of this remedy on the temperature (104°) in this case was much less perceptible. It fell half a degree, and the ensuing exacerbation did not take place. The pulse regained its former frequency within twelve hours after the remedy was discontinued.

[1] It is due to the memory of the late Dr. Todd to point out that a great part of the reform in medical practice with respect to the administration of stimulants in acute diseases is due to him. It is possible that he may have pushed this method at times to an extreme, but of their general utility and of the advantage of administering them in repeated doses, as recommended by him, there can now be but little question. It is beyond the scope of this article to enter upon the rather wide discussion to which this practice has given rise respecting the mode of action of this class of remedies. The chemical side of the ques-

In cases of extreme prostration with a very rapid pulse, and attended by profuse sweating, I believe from what I have seen of the effects of digitalis in the analogous condition of delirium tremens, that this remedy may probably be tried with advantage.[1]

If in the later stages of the disease expectoration becomes profuse and copious, and abundant fine râles in the lung show the presence of œdema, and if resolution be proceeding but slowly, expectorants may be used with advantage. The muriate of ammonia and senega appear to be the best of these, and carbonate of ammonia may be beneficially combined with them.

Counter-irritation may at this stage often prove useful.

The maintenance of the general strength is, however, of paramount importance; and bark, quinine, the mineral acids, or preparations of iron, will often promote recovery more rapidly than remedies devoted to the special condition of the lung. Strychnia is useful in cases where much nervous prostration is present. The use of cod-liver oil is also often beneficial at this stage.

It remains to treat briefly of some of the attendant circumstances and complications of the disease.

Severe gastric catarrh, with a loaded and furred tongue, and whether attended or not by vomiting, is in adults often benefited by one or two purgative doses of calomel (gr. j to gr. iij), followed by a saline aperient, and this remedy is recommended by most authors for the "bilious" form of the disorder. Mustard poultices may also be applied to the epigastrium if vomiting is troublesome. In children, however, this symptom may depend on cerebral disturbance.

If *diarrhœa* be present, a few grains of Dover's powder may be combined with the calomel, and the saline should then be omitted. Severe diarrhœa may, however, require the use of astringents, though, as far as I have observed, this symptom is seldom sufficiently intense to call for their employment. Huss recommends cold compresses to the abdomen, or leeching to the colon, in the dysenteric diarrhœa which accompanies Pneumonia in hot seasons.

If gastric catarrh continues in the later stages, simple alkaline remedies, the bicarbonate of soda combined with bismuth, have appeared to me the most useful. Huss and other German authorities recommend the muriate of ammonia for this symptom.

Hæmoptysis, if profuse, may be met by the internal administration of styptics. The most efficacious of these will probably be found to be gallic acid, acetate of lead, and ergot. The latter is especially recommended by Huss when the pulse is quick, small, and weak. Venesection has been recommended for this symptom, but its true efficacy may be considered as doubtful. It must be remembered that large hæmoptysis is most commonly a symptom of attendant tubercles, and that any reducing measures are, in such a case, specially contra-indicated.

For the condition of *gray hepatization*, Huss and Grisolle recommend the use of camphor, musk, and turpentine. It must be remembered, however, that the full employment of stimulants does not appear to have been practised by these authors. Their administration appears to me to be likely to be better than that of the remedies in question; though these, of which however I have no experience, may at times be useful. Huss recommends the oil of turpentine in doses of five to ten drops every two hours, and says that it is particularly valuable in the Pneumonia occurring in the course of typhoid fever. He remarks that it seldom disagrees even when the tongue is dry and coated, but that if it causes vomiting it may be combined with hydrocyanic acid. He recommends camphor when delirium is present. This remedy, however, appears occasionally to produce redness of the face and dryness of the skin, and under these circumstances it is to be replaced by ammonia.

For the complication of *abscess of the lung*, Huss recommends acetate of lead in doses of gr. ij repeated every four or six hours, as long as the sputa continue offensive and copious. In the later stages bark or quinine with the mineral acids (Huss considers the phosphoric acid to be the best) are the most suitable remedies.

Gangrene of the lung appears to be but little open to remedial treatment. The employment of inhalations of turpentine,

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tion will be found discussed in the researches of Lallemand, Perrin, and Duroy, who maintained that the alcohol so given was excreted by the kidneys; while Strauch (De demonstratione spiritus vini in corpore ingesti, Diss. Dorpat. 1862), Schulinus (Arch. der Heilk. 1866), Dr. Hall Smith's "Experiments on the Chromic Acid Test for Alcohol" (Brit. and For. Rev., 1861), and Dr. Anstie (Lect. Roy. Coll. Phys., Lancet, 1867, vol. ii.), have shown that this only takes place to a very limited degree. The latest researches on this subject are by Dr. Parkes and Count Wollowicz (Proc. Roy. Soc. xviii. 1870).

[1] I have known it under these circumstances, when combined with the administration of alcohol (though the remedy had previously been freely given) markedly reduce the frequency of the pulse and increase its power, while the sweating ceased within a few hours after it had been commenced. The digitalis was given in doses of ʒj of the tincture every two hours.

recommended by Skoda, or of chloroform, has proved useless in Huss's experience. Two cases recovered in his hands; one under the internal administration of creasote in doses of one drop given every two hours, and another with pyroxylic acid in doses of ten drops, combined with five drops of tinct. opii every two hours, but the same remedies proved ineffectual in other cases. More reliance must probably be placed, both in this and in the last-named condition, on the maintenance of the strength of the patient by abundant support, and by bark and ammonia or the mineral acids.

Pneumonia complicated by *intermittent fever* requires the use of quinine. Huss recommends that eight grains should be given during the rigor, and repeated in the sweating stage.

The complication with *pre-existing Bright's disease* also calls in Huss's opinion for the use of turpentine. I have no experience of this method of treatment. It might, however, prove valuable if alcohol appeared inadmissible in such cases. Huss does not appear to regard this remedy as productive of injurious effects on the condition of the kidneys.

For the complication with *pericarditis*, local cupping or leeching and the internal administration of mercurials have been recommended. The utility of all these measures is, however, I believe, in the highest degree doubtful. Deaths from Pneumonia complicated with pericarditis have always appeared to me to present the most marked symptoms of asthenia. The advisability of small local bleedings must, however, be considered in relation to the general strength of the patient.

For *œdema of a limb* remaining after the disappearance of the disease, friction, shampooing, and an elastic bandage are the most appropriate remedies. (Walshe.)

SECONDARY AND INTERCURRENT PNEUMONIAS.

Pneumonia, when appearing as secondary to other diseases, presents in some cases both the anatomical and the clinical features of the acute primary form. In other instances the disease appears in spots of variable size irregularly scattered through the lungs, when it has received the name of Lobular Pneumonia, though it is seldom so strictly limited to individual lobules as this name would imply.

The features of the disease, when of the latter class, and particularly when occurring in children, differ so widely from the Lobar form as to require a separate description.

A short account will also be given of the principal variations in the characters of Pneumonia when appearing as a complication of other disorders.

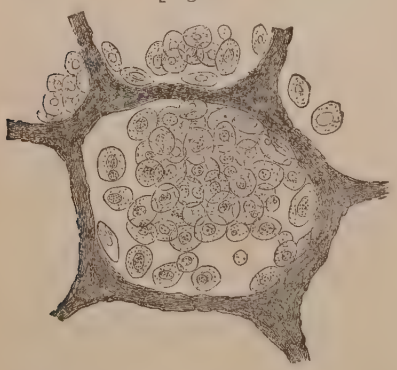
"Catarrhal Pneumonia" is a variety of Pneumonia whose characters are in some respects clinically, and in others pathologically, only imperfectly defined from those of the acute primary form.

Until recently it has been considered to be almost exclusively a disease of childhood, originating either in primary bronchitis or in the bronchitis secondary to measles, hooping-cough, and influenza, and in some cases of diphtheria. It is probable, however, that some forms of the pneumonia of old age may belong more truly to this category; and some recent German authorities have been disposed from pathological considerations—which appear, however, to the author to rest on insufficient foundations—to regard many other cases, hitherto classed with the primary disease, as belonging to this variety. This form of Pneumonia is almost constantly characterized by being preceded by catarrh of the bronchial mucous membrane; and it is a not uncommon complication of bronchial dilatation. The inflammation of the vesicular structure of the lungs is in such cases the result either of direct extension of the inflammatory process, or it is induced through the intervention of collapse of portions of lung, owing to obstruction of the bronchi communicating with them, in a manner which requires a separate and fuller description hereafter. It does not, however, appear to me to be correct to regard *all* cases of Pneumonia which are preceded by bronchial catarrh as forming a separate class. In many of these the bronchitis can only be regarded as one of the prodromata of a pneumonia induced by the same cause, but preceding the true invasion by a period of from twenty-four to seventy-two hours. In others the pneumonia is an accidental complication of pre-existing bronchitis, which possibly may have predisposed to its occurrence, but which, without the intervention of other causes, would not have led to the inflammation of the pulmonary tissue. In both these classes of cases the invasion of the pneumonia is sudden—it runs a typical course, and terminates by a crisis within the usual period.[1]

[1] Out of fifty-three cases I found thirteen to have been preceded by catarrh. In four of these the cough preceded the rigor from twenty-four to seventy-two hours; in one, a chill had taken place a week before the rigor. In three there had been cough for a week before the sudden invasion of the Pneumonia, which commenced either with rigors or vomiting. In three others there was a history of chronic bronchitis. In all these the invasion of the Pneumonia was sudden: two of these cases

In a third class, however, which may truly be termed Broncho-Pneumonia, the invasion is gradual; it is preceded by bronchitis of some standing or intensity, and the implication of the pulmonary tissue is only marked by an increased pyrexia, or by a slight sense of chilliness, usually without rigors, and by prostration with a quick and small pulse and a tendency to sub-delirium, sometimes attended by, but at others without, distinct changes in the characters of the cough and sputa. The latter are usually bronchitic throughout, or they may be puriform, and in a certain proportion of cases rusty sputa are observed. The course of the disease in these cases is protracted and indefinite, either ending fatally, or by a slow lysis and very gradual resolution. In fatal cases the lung is very commonly found in a state of gray hepatization. In a few cases again the invasion may be insidious and gradual, attended by cough and by increasing weakness, but the symptoms may be of such slight comparative severity that patients so affected may continue during some weeks, although with difficulty, their usual occupations. Cases of this class, which bear a strong resemblance to the variety described as "Latent Pneumonia," tend to pass into chronic forms of the disease; and, though occasionally occurring without the complication of tubercles, they have appeared to me, in most instances, to be more or less closely associated with this diathesis.

[Fig. 33.



Catarrhal Pneumonia. — From a case of acute phthisis. Showing the large epithelial cells which fill the alveoli. × 200. (Green.)]

This form of Pneumonia is, however, common during epidemics of influenza, but it may occur without the direct effect of this specific poison. Huss met with it in 140 out of 2616 cases, or in a proportion to all forms of Pneumonia of about $\frac{1}{10}$. The mortality is, however, greater than that of the acute primary form, amounting to 14·28 per cent. It is also very common in tuberculosis, of which it forms a most dangerous complication, and markedly hastens the fatal issue. This association and the clinical phenomena attending it belong, however, more properly to the subject of Phthisis, and will not therefore be considered here.

BRONCHO-PNEUMONIA;[1] LOBULAR,[2] DISSEMINATED, OR VESICULAR PNEUMONIA.

The Broncho-Pneumonia of childhood was by earlier writers largely confounded with collapse of the lung, which was considered a result of inflammation before Legendre and Bailly demonstrated its true character. The publication of their observations led indeed to an almost equally strong reaction in the opposite direction, and it has been thought by many that no true infantile Pneumonia ever accompanies bronchial catarrh, but that all the changes in the lung attending this state are due to collapse alone. This opinion, however, is almost equally erroneous with that which it has displaced, since both pathologically and clinically, inflammation affecting the pulmonary tissue has, under these circumstances, certain well-marked features which it is important to recognize.

The peculiarity of this form of disease consists, as before stated, in its origin in pre-existing bronchial catarrh, either extending from the upper air-passages or commencing as capillary bronchitis. It is not, however, always easy to decide the precise period at which the extension of the disease from the bronchi to the air-vesicles takes place, since this is usually gradual, and in scattered points; and hence in some cases, in children, the condition of Broncho-Pneumonia represents a variable combination of bronchitis and of Vesicular Pneumonia, the symptoms of which are also in part due to attendant collapse.

For the proper understanding of its clinical features and physical signs it is necessary, however, to anticipate so far the description to be hereafter given of its morbid anatomy by stating that the mode of implication of the pulmonary tis-

died on the seventh and eighth days. Of these the affected lung was in one in a state of typical red hepatization; in the other, in a state of gray hepatization. In one case there was a history of previous catarrh of indefinite duration; the invasion was sudden, but the case was protracted. In one only was the invasion gradual. It was, however, a distinct case of Acute Pneumonia.

[1] The term first used by Seiffert (Die Broncho-Pneumonie der Neugeborenen und Säuglinge, 1837).

[2] Burnet; Journ. Hebdomadaire, 1833.

sue ordinarily differs from that found in the Acute Primary or Lobar Pneumonia, and that the nodules of pneumonic consolidation are usually scattered through tracts of air-containing tissue, which is often emphysematous; that these nodules may vary in size from the dimensions of a poppy-seed to those of a walnut, and that they may coalesce until larger tracts are invaded; and further, that the inflammatory changes often commence in portions of collapsed lung; and finally that both lungs are very frequently and simultaneously affected.

ETIOLOGY.—The frequency of this form of Pneumonia in children is variously stated. Ziemssen[1] observed 98 cases as contrasted with 186 of the primary form. Steffen,[2] for 94 of the primary, has met with 72 of the catarrhal or lobular form.

It is most common and most fatal in the earlier periods of life. Of 72 cases observed by Steffen, 52 occurred before four years of age. The age thus specially prone to it corresponds, therefore, with the period of the first dentition; but whether any increased liability to the disease is induced by this process appears to be doubtful, since it is almost constantly a secondary effect of bronchitis, or of diseases of which bronchitis is a common complication in early life.[3] The causes of bronchitis in children are therefore in some degree also causes of Broncho-Pneumonia, and hence it is most common in cold seasons. It is also said to occur at times epidemically, but this is probably due in great measure to the prevalence of influenza or of other zymotic diseases associated with bronchial catarrh.

There appears to be little doubt that a previous condition of bad nutrition markedly predisposes to this form of Pneumonia. The influence of bad air has also been strongly insisted on by Bartels as a more or less direct cause of its occurrence in cases of measles. It is probable also that all causes which diminish the respiratory muscular force of children operate in the same direction particularly when it is remembered that the occurrence of partial or general collapse is frequently the immediate precursor of the inflammatory changes, and that a long-maintained recumbent position, by causing congestion of the posterior portions of the lungs, favors the pneumonic process. Constitutional predisposition to bronchitis at early ages also favors the occurrence of this disease, and thus it is prone to recur in the same individual.

SYMPTOMS.—The signs of pneumonic inflammation are usually developed more acutely in the course of capillary bronchitis and of measles,[1] in which the bronchial inflammation is more intense, than in hooping-cough, in which latter disease the invasion is more gradual, and is almost invariably preceded by pulmonary collapse.

The period of its accession varies also in different diseases. In measles it most commonly occurs during the decline of the eruption, and it may be deferred until the second or even to the third week after the subsidence of the pyrexia and of the exanthem; occasionally, however, it has been noticed to precede the eruption by a period of nearly a week.[2] In diphtheria the pulmonary complications usually occur within the first five or six days; but

[1] Loc. cit.

[2] Klinik der Kinderkrankheiten.

[3] Steiner (Prager Vierteljahresch. 1862, vol. lxxv.) gives the following table of conditions coincident with Lobular Pneumonia:—

	Cases.	Boys.	Girls.
Rickets	26	15	11
Rickets and tubercle combined	11	8	3
Atrophy	16	10	6
Tubercle of glands	15	9	6
Measles	10	7	3
Scarlatina	3	2	1
Smallpox	2	1	1
Dysentery	7	5	2
Noma	2	2	0
Heart-disease	1	1	0
Meningitis	1	1	0
Burns	1	0	1

Steffen, out of fifty-two cases, found thirty-eight arising from bronchitis, eight from hooping-cough, and six from measles.

Ziemssen, in ninety-eight cases, found thirty-two associated with bronchitis or chronic bronchitis, twenty-three with hooping-cough, and forty-three with measles.

Bartels (Virchow's Archiv, xxi. p. 75) found in an epidemic of measles that 12 per cent. of those attacked were affected with Broncho-Pneumonia.

Peter (Gaz. Hebdom. 1863, p. 689) found the disease very frequent in diphtheria. He states that in 100 cases of diphtheria he found sixty-seven of confirmed Pneumonia and twelve of engorgement. Peter's data would appear, however, somewhat to overrate this frequency, for in some of the cases which he cites as instances of "hepatization," the portions affected floated in water; and he also speaks of collapse as a stage of Pneumonia.

Dr. Wilks (Guy's Hosp. Rep. 3d Ser. vi. 146) finds that burns of the skin are very frequently followed by this variety of Pneumonia. In some instances, however, it assumed a more extensive and lobar form.

[1] It has, however, been before stated in respect to measles, that some forms of Pneumonia occurring in this disease approximate very closely in their characters to the true primary form, both in the rapidity with which a large tract of lung is invaded, and also in their anatomical characters.

[2] Steffen, loc. cit.

in hooping-cough they seldom appear until the disease has been considerably protracted, and both the general nutrition and the muscular power of the patient have been impaired. In acute bronchitis Pneumonia may occur early, when the affection is severe, or when other causes predisposing to pulmonary collapse are present; and among these, early infancy, rickets, or previously defective nutrition are prominent. In chronic bronchitis the supervention of Pneumonia is commonly a late phenomenon.

The extension of the inflammation from the bronchial mucous membrane to the tissue of the lungs is rarely attended by the phenomena of rigors, or vomiting, or by the cerebral symptoms which mark the invasion of the primary form of the disease, though the latter may occasionally be observed.[1]

The pulmonary complication is usually first evidenced by an increase of dyspnœa, together with the supervention of fever, if this has not been previously present, or by an aggravation of that already existing.

The dyspnœa, except in very mild cases, when it may be comparatively slight in degree, is commonly both objective and subjective, and gives the patient much distress. It tends at times, and particularly in rickets and when collapse of lung is also present, to occur in suffocative paroxysms, and, even when these are less marked, it varies in intensity at different times of the day, and is usually most felt in the morning and evening.

Great acceleration of the respiration is the rule where the disease is of any considerable intensity; it may then equal the extreme degrees of frequency observed in the primary forms of Pneumonia, and may sometimes attain to 107 respirations in the minute.[2] Commonly the frequency of the respiration is, as in the primary disease, disproportionately greater than that of the pulse, the ratio of 1 to 1·5 being sometimes observed; but when cerebral congestion is also present, the pulse may be rapid and the respiration even slower than natural (Bednär). The respiration is not unfrequently retarded when a fatal termination is approaching. Irregularity in its rhythm is not uncommon, and this may amount to so complete a cessation of the respiratory movements during some minutes as even to simulate death[3].

The thoracic movements are shallow, with great elevation and little expansion. Inspiration is imperfect and short; expiration is often forcible, prolonged, and noisy. The action of the accessory muscles is violent. The chest is raised by the elevatory muscles, but the lower portions are drawn in by the diaphragm. The anterior superior portions appear distended when emphysema is also present, but may yet be comparatively motionless. The action of the alæ nasi is also greatly exaggerated. The cough varies in character. It is sometimes paroxysmal, but this character, even when previously present, as in hooping-cough, may disappear on the supervention of Pneumonia, and the cough may become short and dry; and this change in its character often forms, together with the pyrexia, one of the earliest signs of the implication of the pulmonary tissue in this disease. The cough also often becomes painful, eliciting cries from the patient, a symptom which is also sometimes a valuable indication of the pneumonic change. I believe, however, from some cases in which I have observed this in adults, that such pain may be extra-thoracic and myalgic in its nature, and that it is partly caused by the sinking of the inferior parts of the thoracic walls due to collapse of the lung. In some cases the pain complained of may be in the epigastric and in the upper abdominal regions.[1]

The secretion from the bronchi, if previously free, as shown by the looseness of the cough, is often diminished. Expectoration is rarely seen in young children. When brought up by vomiting, it is commonly bronchitic and tenacious, sometimes streaked with blood, but rarely if ever rusty. The same characters are observed in the sputa of adults attacked by Pneumonia during the prevalence of influenza.

The physical signs in the earlier stages are often obscure. They commonly affect both lungs simultaneously, though rarely in an equal degree. The immobility of the thorax and the sinking of the lower ribs, with deepening of the diaphragmatic depression, occur in cases of simple bronchitis, with attendant emphysema and collapse; but when the lung becomes extensively infiltrated, the depression of the ribs may partially disappear.

The percussion results may be uncertain: usually the upper parts of the chest are hyper-resonant, and they may be quasi-tympanitic when much attendant emphysema is present. When the spots of collapse or of pneumonic infiltration are disseminated through healthy pulmonary tissue, the sound, though less resonant than natural, is rarely dull, and

[1] Dr. West (Dis. of Infancy and Childhood, p. 326). Steffen has once seen spurious hydrencephalic symptoms precede the outbreak of the Pneumonia, and cease on its appearance (loc. cit. 302).

[2] Bednär, Lehrbuch der Kinderkrankheiten, 268.

[3] Barthez and Rilliet, i. 264.

[1] Steffen.

only loses the pulmonary tone when these have coalesced into more extensive tracts. The dulness of Pneumonia does not differ markedly from that of collapse, though the latter may occasionally acquire a tympanitic tone (Ziemssen); but it is usually more intense. The site of collapse is, however, peculiar. It tends to occur at the free border of the left lung overlapping the heart, and also at both bases posteriorly, when, instead of extending uniformly, it passes upwards in an elongated and quasi-pyramidal form along the lines of the intervertebral grooves, and it may, maintaining this peculiarity, extend nearly to the apices of the lungs.[1] As, however, collapse often constitutes the first stage of the pneumonic process, this form of dulness may be maintained after the latter has set in.

The respiratory sounds over collapsed portions are commonly weak or inaudible. In lobular pneumonic consolidation they usually acquire a bronchial, but never a tubular character (Walshe). This quality of respiration, though occasionally, is only very rarely met with in simple collapse.[2]

The respiration in other portions of the chest is usually exaggerated and attended by râles. Generally disseminated dry or moist bronchitic râles indicate only the bronchial catarrh. When Pneumonia supervenes, they, however, often become finer, and may thus be heard in limited spots of the pulmonary tissue, and they frequently change in site from day to day;[3] but they seldom present the typical characteristics of the crepitation heard in the Acute Lobar Pneumonia. In some cases, however, when the finer bronchi are dilated, the râles heard may be coarse, and they may acquire a quasi-metallic character if consolidation surrounds these dilatations. Râles are seldom heard directly over collapsed parts, unless they be conducted from adjacent bronchi.

Vocal fremitus is commonly exaggerated over pneumonic infiltration more than over collapsed portions of lung. The differences of degree observable in this respect are, however, very variable.

Vocal resonance, as heard when a child cries, is usually much increased by pneumonic consolidation of any extent, and frequently under these circumstances it acquires a bronchophonic tone. These characters may, however, be absent when the bronchi are extensively obstructed.

[1] Ziemssen, loc. cit.

[2] Barthez and Rilliet; also Ziemssen and Gerhardt.

[3] When masked by other râles it is desirable to follow the advice given by Barthez and Rilliet, and to repeat auscultation after the act of coughing.

The pulse is rapid. It rarely, even in the early stages, presents the fulness or strength of the primary disease. At more advanced periods it becomes excessively frequent, small, and feeble, so as scarcely to be felt. Irregularity of its rhythm is also occasionally observed. Fulness of the superficial veins, extending even to those of the hands (Trousseau), is also observed, and œdema of the extremities has sometimes been noted (Steffen).

Simultaneously with these symptoms there is a great restlessness: the eyes are sunken, and the face assumes an anxious expression, which is painfully distinct in young children. Strength fails rapidly; as the disease progresses somnolence and a semi-comatose condition supervene, in which the child lies passive, but starting up from time to time into an erect or semi-erect posture, with jactitation and movements of distress, when attacks of cough and dyspnœa return.

The skin is hotter than natural, though not commonly presenting the pungency of heat which characterizes the acute lobar form, and it is often bathed in profuse perspiration, which occasionally alternates with a dry heat. The perspiration may be general, or in rickety patients may appear chiefly about the head. The surface is generally pallid with the exception of the cheeks, which present a flushed or violet tinge, which is sometimes transitory and alternates with a cyanotic pallor. Cyanosis of the lips and finger-nails increases with the progress of the disease, and is especially distinct in the pneumonia succeeding to hooping-cough, and when collapse forms a prominent feature.

Vomiting, unless caused by the cough, is less common in this form of Pneumonia than in the acute primary disease. Diarrhœa, on the other hand, is not unfrequent, particularly in the Broncho-pneumonia attending measles, and if not originally present it is very easily excited by medicinal remedies, especially by tartar emetic. The tongue, at first moist, becomes dry in the later stages, and sordes form on the teeth or on the angles of the lips, which are also dry and cracked: aphthous stomatitis may occur when the course is protracted. The appetite is completely lost, but thirst is marked; infants at the breast suck it eagerly, but the power of continued sucking is lost, owing to the difficulty of breathing. A slight degree of delirium is occasionally observed, particularly in older children, as an exaggeration of the restlessness which tends to increase towards night. Convulsions are, however, much less frequent than in the acute disease; when they do occur, they form a very unfavorable feature. A semi-comatose state is more common; it passes later into deeper un-

consciousness when the signs of mal-oxygenation of the blood become more apparent. In some cases, however, a hydrencephalic condition with restlessness and cries has been observed.[1]

The urine, owing to the early period of life in which the disease usually occurs, has not been made the subject of exact observation. Bednär says that the chlorides are present. In some cases the presence of a small amount of albumen has been noticed.

Emaciation and loss of strength progress with marked rapidity; there is great loss of weight, the eyes are sunken, the muscles are wasted and the skin is flaccid. These appearances may, in severe cases, become very distinct within a few days from the outset: if the disease runs a more protracted course, and particularly in the Pneumonia succeeding to hooping-cough, the wasting of the tissues may attain an extreme degree of marasmus—proportioned, however, in most cases to the age of the patient and to the severity of the disease. Ecthymatous pustules often form, which lead to painful sores. Excoriations of the nose and angles of the mouth are also observed, and bedsores form on the prominent parts of the emaciated limbs. The patient often dies completely exhausted, or sinks suddenly during a paroxysm of cough, or with the increasing cyanosis may pass into a state of final somnolence and coma. When death occurs in the early periods of the disease, it is usually due to the combined asphyxiating effects of capillary bronchitis and collapse. The course of the disease after Pneumonia has set in is usually more protracted.

Many of the above symptoms, and especially the increasing intensity of the dyspnœa, may occur in severe cases of bronchitis accompanied by extensive collapse of the lung, and uncomplicated by Pneumonia. The most characteristic feature of the latter is constituted by the pyrexia, the presence of which is almost essential to its recognition. Acute bronchitis in children is, indeed, not unfrequently attended by fever, but when uncomplicated by Pneumonia the temperature seldom rises in it above 101° or 102°. The fever also when present is not continuous, the temperature in the morning being often nearly at the normal standard, or perhaps falling to 99° or 99·5°. The invasion of Pneumonia is marked by accession of fever if the disease has been previously apyrexial, or by an increased temperature if fever has already existed, and this, in the pneumonia of measles, may speedily attain the degrees of 103, 104, or 105. The lower standard of 102° may, however, not be surpassed in the whole course of the case.[1] Sometimes a rise of temperature may be observed to *follow* the accession of dulness on percussion, both at the outset and during the subsequent exacerbations, showing that collapse has preceded the inflammatory changes. This, however, is not always to be observed, and the rise of temperature may be comparatively sudden and rapid, and may either proceed *pari passu* with the loss of resonance on percussion, or may precede this by some hours or days—the diminution of pulmonary resonance only becoming distinct when the islets of Lobular Pneumonia, by becoming confluent, affect tracts of tissue sufficiently extensive to give rise to this physical sign.

In some cases again, when Pneumonia succeeds to measles, the invasion both of the physical signs and also of the pyrexia may present a great resemblance to the phenomena of the acute primary disease—the temperature rising rapidly, and maintaining a tolerably uniform elevation with comparatively slight morning remissions, but in its later periods, running a protracted course resembling the catarrhal type.[2] In its subsequent course, however, the pyrexia as measured by the temperature presents certain characteristics which aid considerably in the recognition of this form of Pneumonia. The chief among these are the irregular course of the fever, the extent of the remissions and exacerbations, and the absence of critical phenomena—the fever being usually protracted, and ending only by a slow and gradual decline, which is often interrupted by renewed exacerbations.

The remissions may be as great as from 1·8° to 2·5° Fahr. They occur at irregular times during the day, differing in this respect from the ordinary course of the acute primary disease. It has been already stated that in some cases of this form, the maximum temperature may be observed in the morning instead of at night; but this condition, which is exceptional in Primary Pneumonia, is much more common in Broncho-pneumonia, when it may be noticed to occur irregularly in the course of a single case—a peculiarity which is probably due to the indefinite course and irregular extensions of the pulmonary inflammation. The termination of the fever is also protracted. Ziemssen regards a case terminating

[1] Barthez and Rilliet (i. 467) attribute this to frontal neuralgia.

[1] In some fatal cases the temperature may rise shortly before death to upwards of 107° (Ziemssen), but on the other hand a rapid fall of temperature, due probably to defective aëration of the blood and to extension of the collapse, may immediately precede the fatal issue.

[2] Ziemssen and Krabler, Klin. Bericht ueber die Masern und ihre Complicationen, p. 169.

within seven days as a very exceptional one; and the pyrexia may last for weeks,[1] in the Broncho-pneumonia both of measles and hooping-cough, presenting in its irregular exacerbations and remissions a striking resemblance to the course of tuberculosis, which, however, according to the observations of Bartels and Ziemssen, is a much less common sequela of these diseases than is usually believed. The defervescence rarely, if ever, presents the abrupt critical fall so commonly observed in the acute primary form: or if this commences, it is usually followed by subsequent elevations of temperature: the decline of the fever is only gradually effected, and rarely extends over a shorter period than three or four days, and it is often interrupted by irregular secondary exacerbations. In some cases the temperature often finally sinks during some hours or days below the normal standard.

The range of temperature is commonly lower, and both the course of the disease and the duration of the pyrexia are more protracted in the Pneumonia succeeding to subacute bronchitis and to hooping-cough than in that which follows measles. In hooping-cough the degree of pyrexia may be very slight, and the morning remissions may attain almost to the normal standard,[2] but exceptional cases occur in which Pneumonia complicating this disease appears in an acute form and runs a rapid course to a fatal termination.

With the gradual decline of temperature other signs of improvement become evident. There is rarely any appearance of critical sweating, but perspiration appears from time to time, and often seems to afford relief. The dyspnœa and the cyanotic aspect diminish; the pulse and respiration fall in frequency; the cough becomes looser and less hard, and it may again acquire a paroxysmal character, if this has, as in hooping-cough, been previously present; diarrhœa, if present, ceases; the appetite gradually returns, and thirst disappears or diminishes in intensity.

Recovery is, however, almost always slow and protracted; cough persists long; and the duration of the physical signs of consolidation, and especially of bronchitic râles, may continue during many weeks. Some acceleration of the respiration and of the pulse may also continue after the fever has subsided.[3] Slight returns of the pyrexial symptoms may also be observed during this period. The restoration of the digestive powers and of the nutrition is only very gradually effected, and the patient may be for months liable to a renewal of catarrh, attended with slight degrees of feverishness.

In some cases, after a long continuance of the pyrexia and of the physical signs, the former may subside, but the latter may change their character and present those of chronic Pneumonia or of bronchiectasis, or sometimes of the latter alone.[1]

Complications. — Independently of the diseases which exert a direct or predisposing influence on its production, the liability of Broncho-pneumonia to other complications is comparatively slight. Some of these will be further alluded to among attendant pathological phenomena. One of the most important is intestinal catarrh, which has been attributed by M. Beau to the swallowing of unhealthy sputa. It appears, however, to be more directly due to the venous congestion of the intestinal canal, and to the tendency of catarrh in children to affect the whole gastro-pulmonary tract of mucous membranes. True dysentery was observed by Steiner in seven out of 110 cases. Pleurisy is less frequently observed than in the acute primary disease. Steffen in seventy-two cases only found six of extensive pleuritic effusion. Various degrees of the affection, in the form of adhesions, are by no means uncommon.

Tubercle is not uncommonly associated with the chronic form, but whether as to cause or effect it is not in all cases easy to distinguish. It is not improbable that repeated relapses may give rise to this tendency, and a statement of Steiner's, which will be alluded to hereafter, would seem to show that even collapse may suffice to set up tubercular formations in predisposed subjects; and Bartels has remarked that when present it is specially prone to occur in the condensed portions. On the other hand, the statements of Bartels appear to show that tuberculosis is a much less common sequela of the Broncho-pneumonia of measles than has been commonly believed.[2]

PATHOLOGY AND PATHOGENESIS.— It has been already stated that this form of Pneumonia may be produced by the mechanism of two distinct processes.

[1] Eight weeks. (Bartels.)

[2] See a case by Steffen, p. 313. This case was the more remarkable, as it was one of hooping-cough complicated by tuberculosis, which had progressed to the stage of excavation.

[3] Steffen.

[1] This subject will be again referred to under the head of Chronic Pneumonia.

[2] Bartels only found tubercle four times in twenty-one post-mortem examinations, and in two of these the affection was meningeal. Ziemssen only observed cheesy changes twice in sixty-three cases of death. Legendre and Bailly found five cases of tubercle in twenty-seven of catarrhal Pneumonia. Ziemssen and Krabler remark, however, that acute tuberculosis is not uncommon *after* measles.

The pulmonary alveoli may suffer by the direct extension of the inflammatory action from the bronchi, or the inflammatory changes may only take place in portions which have already become the subjects of collapse. The pathological appearances in the inflamed lung present under these conditions several points of difference. Under both sets of circumstances the changes in the mucous membrane of the bronchi are very similar, exhibiting various degrees of inflammatory congestion, together with swelling and softening of the mucous membrane, which are accompanied by slight superficial ulcerations. These changes may appear throughout the whole course of the air-passages when the catarrh has commenced in the larynx and trachea and has travelled downwards. In some cases, however, they may be limited to the smaller bronchi, and the upper air-passages may present a comparative immunity. The walls of the finer bronchi are often so much thickened as to cause them to stand out rigidly in sections of the pulmonary tissue.

Dilatations of the bronchi are very common; they are generally in the cylindrical form, but are sometimes globular, and they may be so universal throughout the lung as to give it a cribriform aspect on section, or even to present the appearance of a number of small cavities. The contents of the bronchial tubes are usually a creamy pus, or a denser exudation constituting a form of false membrane, or sometimes a clear tenacious mucus containing nucleated cells in which a large proportion of desquamated ciliated epithelium may be found. In other cases again little or no mucus can be found in them.

The changes in the lung tissue, when the inflammation has proceeded directly from the bronchi, present the appearance of a number of small whitish-yellow spots with indistinct margins fading insensibly into the surrounding tissue. They are not very prominent,[1] and do not stand out sharply defined like the gray granulations or the small softer spots of more acutely produced tubercle. They are often very thickly scattered through the pulmonary tissue, and the portions of lung intervening between them are softer, more vascular, and more œdematous than natural. They are slightly but not markedly granular, and usually, on scraping or pressure, a turbid milky fluid can be expressed from them. As they increase in age they become firmer and drier, but still have little of the granular character. That they undergo any cheesy metamorphosis independently of pre-existing or superadded tubercular changes is in my opinion very doubtful. In addition to these spots others are found, varying in size from a pin's point to a hemp-seed, of a brighter yellow, consisting of dilated air-vesicles, or sometimes of groups of air-vesicles whose walls have broken down, or of the terminal extremities of dilated bronchi. In whichever manner originating, they form little collections of puriform fluid, which escapes when they are pricked, but which also may become more inspissated, and which when evacuated leave irregular cavities. They are sometimes found under the pleura forming little sub-pleural abscesses, and in some cases may by their rupture give rise to pneumo-thorax. It is doubted by some writers whether these originate in a true inflammation of the air-vesicles of the lungs, or whether they are not merely collections of pus which have gravitated or have been drawn by inspiration into the extremities of the dilated bronchi and infundibula. I believe from my own experiments on animals in which, especially in dogs, both these sets of appearances can be produced easily by injecting ammonia into the trachea,[1] that they are to be regarded as truly inflammatory in their nature; though the latter mode of origin is however possible, since, as Ziemssen (who agrees with the view previously expressed by Fauvel of their nature) remarks, they are most commonly to be found after the prolonged paroxysms of hooping-cough. The purulent yellow spots before described are perhaps more commonly found in the Pneumonia succeeding to measles than in other conditions.

I have before stated that the Pneumonia which is secondary to diphtheria may present in some cases all the anatomical characteristics of the acute primary form. In other cases it takes place by the intervention of collapse; but in a third class the appearances observed correspond with those which have just been described. When the bronchial ramifications are opened it will be found, in proportion as these diminish in size, that the firmness of the exudation layer diminishes, and that the finer bronchi are only filled with a soft puriform fluid. In some instances this process does not extend beyond bronchi of from two to four lines in diameter; but, in other cases, patches of a yellowish color and soft consistence, varying in size from that of a hemp-seed to a horse-bean, and only rarely attaining the dimensions of a hazel-nut, are found in the pulmonary tissue. These are impervious to air, and are only slightly granular. They fade insensibly into the surrounding tissue by an ill-defined margin. They are friable, and on pressure

[1] These spots are often described as "prominent," but the author believes that as a question of degree the distinction here drawn will be found to be correct.

[1] See p. 159.

they allow a thin yellowish fluid to exude. On microscopic examination the air-vesicles are found, for the most part, occupied by an amorphous exudation, in which are seen a few puriform and granular cells, together with proliferating epithelium from the air-vesicles. I agree with Sir William Jenner,[1] that in most cases these spots are formed by the fluid formed in the bronchi being drawn by inspiration into the air-vesicles of the lungs.

In the Pneumonia which occurs consecutively to collapse of the lung,[2] various stages may be observed in the inflammatory process. It requires, however, to be stated, that in many fatal cases of bronchitis, and particularly in infants, the condition of extensive collapse, unattended by a trace of inflammation, may be the only morbid change present.

The mechanism of the production of collapse will be more fully treated of in the section devoted to this subject. It is therefore only necessary in this place to refer to the inflammatory changes ensuing in parts which have already undergone this change.

In collapse pure and simple, the parts affected are sunk below the level of the surrounding tissue. They are most usually found at the bases of the lungs and at the free borders of the lower inferior lobe, and they commonly affect both lungs simultaneously, though rarely in an equal degree. They are often pyramidal in shape, with the base at the periphery of the lung corresponding to the distribution of the terminal bronchi leading to the affected parts. They, sometimes, however, occur in the more central portions. They are irregularly distributed, and vary in size from that of a hemp-seed when they arise from the collapse of small groups of pulmonary lobules, to spots of one or two inches in diameter. In severe cases, both in infants and in adults, a whole lobe may be affected, though the pyramidal form is that most commonly observed.[1] On section the nodules are of a bluish-purple tint, which is uniform except when traversed by bronchi, blood-vessels, or interlobular septa; they are smooth on section, allowing only a small amount of blood-stained serosity to escape on pressure or by scraping; they are often, however, attended with scattered ecchymoses. They are resistant, and do not break down readily under pressure, and they are airless and sink in water, but can be restored to their normal condition of expansion by inflation, which process, however, leaves the affected parts of a brighter red than the surrounding tissue. This latter is, however, more or less congested and œdematous, and not unfrequently emphysematous. When the condition has lasted longer, the ability to insufflate the lung diminishes, and may even be partly lost, and the parts thus affected may finally undergo either a simple atrophy, or may become the seat of fibroid metamorphosis or of calcareous degeneration.[2] These changes belong, however, rather to the pathological history of bronchitis than to that of Pneumonia, with which they have no necessary connection, though under the names of CARNIFICATION and CARNIZATION[3] they have often been confounded with it.

The process of inflammation in the collapsed parts is effected through two sets of changes, which, however, differ chiefly through the degree in which they are complicated by passive congestion. In some cases this is extensive, and affects large tracts which have previously become collapsed, and extends also to the surrounding tissue. The cause of this congestion appears to be the increased impedi-

[1] Oral communication.

[2] Hasse (Path. Anat., Syd. Soc. Ed., p. 251) says that Pneumonia is not necessarily frequently associated with atelectasis. This may be true of primary atelectasis in the recently born infant, but I believe that acquired collapse frequently forms the starting-point for secondary Pneumonia. Hasse says that he has seen spots of collapse in the midst of hepatized lung, but not participating in the inflammatory changes. There is no doubt but that Pneumonia and collapse may not unfrequently be found coexisting, but I believe that it is quite as common to find Pneumonia commencing in the midst of collapsed portions, as it is to observe the condition described by Hasse. The elucidation of the origin and nature of collapse, and the recognition of the distinction between this condition and Pneumonia, appear to have led many writers since the publication of the researches of Legendre and Bailly to deny too exclusively the existence of Lobular Pneumonia in children, and to attribute all the appearances of consolidation found in their lungs to the condition of collapse. There is no doubt as to the comparative frequency and the important pathological and clinical significance of this condition, but I believe that these have been to some degree exaggerated, and the importance of pneumonic changes has been in consequence underrated.

[1] In some cases of collapse from pneumothorax a whole lung may thus become affected by secondary Pneumonia (Steffen, loc. cit. p. 24).

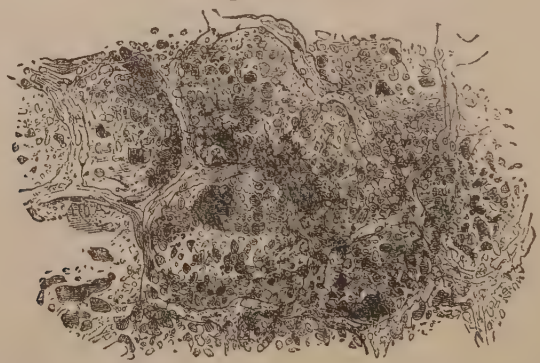
[2] Hasse (Path. Anat., 250); Gairdner (Brit. and For. Rev., April 1853, p. 467; Path. Anat. of Bronchitis, p. 68).

[3] It appears best to confine the significance of these terms to simple "collapse," which presents an appearance which they fully describe. The application of them to other conditions only involves confusion. MM. Isambert and Robin (Gaz. Méd. 1855) have applied the title of "Carnification congestive" to a form of induration of the lung secondary to heart disease.

ment to the circulation arising from defective aeration of the blood, and also from the absence of the alternate expansions and contractions of the pulmonary tissue, which, under normal conditions, largely favor the passage of blood through the capillaries of the lung. To this passive congestion œdema quickly succeeds. The tissue then loses its bluish tint and becomes of a darker purple; much serosity exudes on section; and this, in the parts not previously collapsed, may be frothy from the admixture of air. The tissue also becomes more swollen, and the tough resistant character of simple collapse being lost, it is also rendered more friable and breaks down under pressure. To this state the term SPLENIZATION has been applied, and it is sometimes erroneously confounded with the "Pneumonie des agonisés" of Piorry, though the changes now described are not, in their essential nature, of an inflammatory character. Congestion of this kind may affect large tracts of tissue surrounding collapsed portions, and hence it has been observed that these parts may be crepitant, or that the collapsed and congested portions may be insufflated; and from this fact has arisen the statement that insufflation is possible in the early stages of Lobular Pneumonia. When inflammatory changes occur in such parts they appear generally in scattered nodules which are solid and granular, but very friable, and in which the interlobular septa have disappeared and the ordinary character of collapsed tissue is destroyed. These nodules are whiter in color than the dark purple tissue surrounding them, but into which they fade insensibly at their margins. They depend on the accumulation in the interior of the air-vesicles, of enlarged epithelial cells, mucoid cells, and pyoid cells, which fill and distend them; and hence, in addition to being solid, the parts thus affected become prominent above the level of the surrounding tissue. Collapsed portions which have become congested sink in water without pressure. Congested parts surrounding these usually float imperfectly, but sink after pressure. The pneumonic nodules sink without pressure. On scraping the latter a milky fluid exudes, airless, and presenting under the microscope cells of the same character as those found in the pulmonary alveoli. As the process extends, the whole of the collapsed and congested parts may gradually become infiltrated until the greater part is solidified, but usually the nodular form is preserved for some time with congested and excessively œdematous tissue intervening between the pneumonic portions. This form of Pneumonia readily passes into the condition of gray hepatization. The congestion disappears from the infiltrated parts, and the gray appearance is produced by the rapid progressive fatty degeneration and the liquefaction of the inflammatory products, aided by the coexistence of œdema.

Pneumonic changes may, however, occur in collapsed portions, without being preceded by such marked evidences of congestion and œdema as those last described. Under these circumstances, the

[Fig. 34.



Broncho-Pneumonia.—From a child, aged four, with capillary bronchitis. A section of one of the patches of consolidation. Showing the stuffing of the alveoli with what appears in the main to be inhaled bronchial secretion. × 200. (Green.)]

appearances presented offer a considerable resemblance to the first form of Vesicular Pneumonia, and it may be questioned whether in some cases the existence of collapse is not an accidental complication superadded to the pneumonic process, rather than an essential factor in its production. In the collapsed portions nodules are found of a grayish-yellow, contrasting strongly with the surrounding purplish tint of the adjacent tissue. They are also prominent about its level. They are solid, and usually granular, and yield a milky or yellowish creamy fluid on scraping or pressure. In them, however, dilated bronchi filled with a yellowish puriform fluid are often observed, and these may even give the appearance of small abscesses

scattered through the nodules; when evacuated they leave small cavities. Dilated bronchi with similar characters may sometimes be found in parts affected by simple collapse;[1] but unless true Pneumonia be superadded, the granular character of the adjacent pulmonary tissue is absent.

These appearances may occur in collapsed portions of lung, however originating. I have found pneumonic nodules identical with those last described in the base of the lung of an adult dying of uræmic poisoning, and who had presented during life no signs of bronchitis, but in whom dulness on percussion, unattended with pyrexia, had appeared at the base during the later days of life. The whole of the lower lobe of one lung was collapsed, and in this part were spots of Pneumonia surrounding small dilated bronchi, which were filled with a yellowish pus. It has been recognized since the observations of Dr. Baly,[2] that in the exhausting diseases of adults collapse may easily occur, probably from muscular weakness, though this change is comparatively less frequent in them than in children, owing to the more yielding character of the chest-walls and the smaller calibre of the bronchial tubes in the latter. Collapse is thus in many cases the first stage in the production of hypostatic Pneumonia, so frequently found in post-mortem observations. Hourmann and Dechambre had previously described appearances identical with these in the lungs of the aged, as consisting of scattered yellow or whitish spots, from which a yellow fluid escaped, leaving behind small vesicles situated in the midst of congested tissue,[3] and passing at times into larger nodules of a granular appearance; and Durand-Fardel[4] has confirmed these observations.

It is not uncommon to find the processes of collapse, congestion, and Lobular Pneumonia intermingled in the same lung, and every stage may be occasionally observed between them. The characters of the pneumonic change also vary, and in some parts nodules may be found resembling in all their essential features spots of red hepatization, as observed in the acute primary form, while in others the pneumonic portions may be white or gray, or almost diffluent, as if softening into abscesses.

In all the forms now described, and in fact in nearly all cases where nodules of Pneumonia reach the surface of the lung, the pleura participates in the inflammatory changes, and a layer of lymph is found on the surface, which is congested and roughened. These characters may sometimes aid in the distinction between Pneumonia and simple collapse, for in the latter, though sub-pleural ecchymoses are sometimes observed, a true inflammation of the pleura is rarely found. Extensive pleural effusion is, however, very unfrequent in Lobular Pneumonia.

Scattered nodules of red hepatization, occurring apparently without the intervention of collapse, and also without any appearance of direct extension from the bronchi, are found in other blood diseases. I have seen them in smallpox[1] and diphtheria, and they also occasionally occur in scarlatina and typhoid fever. It is probable also that some forms of the Pneumonia occurring in the course of typhus originate in a similar manner. These cases certainly militate against the view expressed by some recent writers, that the acute primary or croupous Pneumonia is a specific disease with special anatomical characters.

All these disseminated varieties of Pneumonia, including the true vesicular or broncho-pneumonia of measles, may occasionally coalesce and occupy large tracts of the lung,[2] and under these circumstances also the anatomical distinctions from the acute primary disease become very uncertain, though the consolidated tissue is generally paler and whiter, and presents a greater number of puriform spots arising from the accumulation of pus in previously dilated bronchi, than are commonly observed in the acute primary form. In the rarer cases, when a Lobar Pneumonia appears to be produced in adults by a gradual extension from the bronchi, there are few, if any, exact anatomical observations respecting the condition in the earlier stages. Most of these, except in old people, occur in tubercular subjects. A few pass into the condition of Chronic Pneumonia, hereafter to be described. The leading characteristic of such cases, when non-tubercular, appears to be a tendency to pass early into a state of gray hepatization.

There is also in the typical forms of catarrhal Pneumonia a greater amount of proliferation of epithelial cells, and a smaller number of pus corpuscles, at least in the earlier stages, than is observed in the primary disease. A spontaneously coagulable fibrinous exudation is rarely present, and the material in which the cells are imbedded is semi-fluid

[1] Dr. Graily Hewitt, "Pathology of Hooping Cough."

[2] Communicated to and cited by Dr. West (Dis. of Infancy and Childhood, p. 291).

[3] Arch. Gén., xii. 2e Sér. 274.

[4] Maladies des Vieillards, 475.

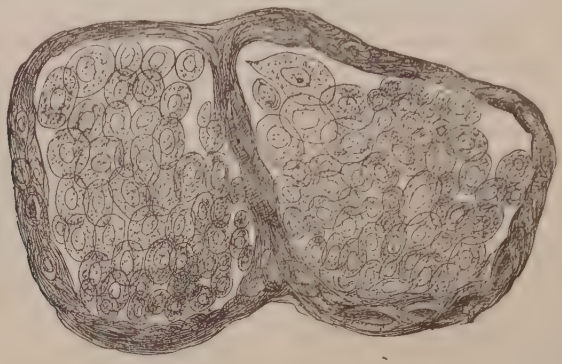
[1] Steffen says that yellow spots corresponding to puriform collections in dilated bronchi may occasionally be found in the secondary Pneumonia of variola (loc. cit. p. 294).

[2] The "Pneumonie Lobulaire Généralisée" of Barthez and Rilliet.

and allied to mucus, approaching in this respect the characters of the gelatinous-looking exudation which fills the air-cells in the pneumonia attending many cases of tuberculosis; I believe, however, that all gradations can be observed between these products.[1] In some cases also of the most distinct hypostatic Pneumonia I have found the air-vesicles filled almost entirely with pyoid cells, resembling those seen in the later stages of the acute primary disease. (See Fig. 31, p. 193.)

In the further progress of Broncho-pneumonia there is little doubt but that in many cases a perfect *restitutio ad integrum* may occur, and that the lung may

Fig. 35.



Alveoli in Broncho-pneumonia.

regain its normal condition. In other instances, however, dilated bronchi may persist long with some condensation of the pulmonary tissue surrounding them, but may gradually return to the normal state, as far as may be judged of from the physical signs. Abscesses also may occasionally form, though usually they are small in size, and gangrene is also sometimes observed in the affected portions.[2] In rarer instances, general consolidation of the lung may remain in a chronic form, the characters of which will be described under the head of Chronic Pneumonia. In some instances the pneumonic nodules, particularly in scrofulous and rickety children, may pass into cheesy changes with destruction of tissue, and may run the subsequent course of tubercle. Ziemssen describes these as a true tuberculization. Barthez and Rilliet[3] also describe them as surrounded by a zone of gray induration, precisely resembling in this respect the changes found around tubercles in the adult. Bartels, as already stated, has found tubercle to be a less common complication of the pneumonia of measles than is commonly believed; but I am convinced, from my own microscopic observations, that tubercle may occur in these spots of pneumonic change, although it may be masked, and may be undiscoverable by mere ocular inspection.[1] The pneumonic changes are always limited to an accumulation of cells in the interior of the air-vesicles and terminal bronchi, and, except in very chronic forms, when interstitial thickening occurs, the walls of the alveoli themselves are not the seat of nuclear growth.

The associated pathology of Broncho-pneumonia presents but few special features independently of those of the diseases of which it constitutes a complication. The dilatation of the right side of the heart, resulting from obstruction to the pulmonary circulation, may lead to permanence of the openings of the foramen ovale and ductus arteriosus.[2] Throm-

[1] See Appendix D.

[2] This is, however, rare. Steiner only observed two cases. Steffen also reports two such. It is rather more common in the indurations surrounding chronic bronchial dilatations.

[3] Loc. cit. i. p. 436.

[1] Legendre and Bailly only found five cases of tubercle in twenty-seven of catarrhal Pneumonia (loc. cit. 215). Steiner (loc. cit.) describes accumulations of nuclei as occurring in the interstitial tissue of parts affected by collapse, proceeding to such an extent as to fill the cavity of the alveoli. Other observers describe in the chronic forms of collapse a thickening of the interstitial tissue. I cannot but believe that such accumulations of nuclear growth as Steiner describes are of a tubercular nature. Steiner speaks of miliary granulations of tubercle in the pleura as not uncommon. I am disposed to believe, in spite of the recent statements of some high authorities, and particularly of Niemeyer, that cheesy changes in the lung are a very rare event when not caused by or associated with tubercle.

[2] The patency of the foramen ovale with attendant cyanosis was observed, as early as by Jörg, to be a frequent complication of collapse (Diss. de Pulmonum vitio Organico, p.

bosis of the pulmonary artery is occasionally observed. Pericarditis is also an occasional complication. The bronchial glands are swollen and medullary-looking. Sometimes they are distinctly hyperæmic, but when the swelling is extreme they may be pale. In a few cases they are unaffected. Sometimes cheesy spots or calcified nodules are found in them, but these usually accompany tubercles in the lung. Suppuration of the post-tracheal glands leading to an ulcerative opening into the trachea was once observed by Steiner.

The appearances observed in other organs are for the most part the result of venous congestion. Œdema and congestion of the brain is common in fatal cases. Meningitis of the base is a rare complication. The liver is congested, and hyperæmia and catarrh of the stomach and intestines are also common. In the large intestines the catarrhal congestion may even give rise to dysenteric changes. The kidneys are also congested, and concretions of urates are often found in the straight tubules of the pyramids. General dropsy is an occasional complication. Amyloid changes in the liver, spleen, and kidneys, which are sometimes present, must be regarded as pre-existing and accidental conditions rather than as results of the pulmonary disease.

Diagnosis.—The diagnosis of Broncho-pneumonia may be occasionally difficult, as may be inferred from the preceding description of its symptoms and physical signs. The chief points requiring to be alluded to here are the actual existence of Pneumonia in some cases of bronchitis, and its distinction from collapse in others.

Under all circumstances, the indications afforded by the thermometer are most valuable, and often aid in the interpretation of the physical signs.

The latter are often obscure ; but when sufficient lung-tissue is consolidated to alter the qualities of the percussion-resonance and of the respiratory murmurs, the following characteristics may aid in the diagnosis of Pneumonia from *collapse*, though, in the intermediate stages by which the latter passes into the former, many of them are but little available.

The loss of resonance is more absolute in Pneumonia, and the note, though sometimes tubular, seldom possesses any of the tympanitic quality occasionally observed in collapse.

The side is more retracted, and the sinking in of the ribs and elevation of the diaphragm are more distinct in collapse than in Pneumonia. Indeed, when the latter is extensive, falling in of the lower ribs is not observed.

In collapse the respiratory murmur is weak or *nil*. In Pneumonia it is bronchial or blowing, but seldom tubular, in the form of Broncho-pneumonia. Exceptions, however, occur on this point, and pure collapse may give a metallic tone to the respiration.[1] Râles are not heard as a rule in collapsed lung (though the possibility of their presence when dilated bronchi traverse the collapsed portion can hardly be disputed). In Broncho-pneumonia, râles more or less approaching the crepitant or sub-crepitant type are tolerably constant, though not invariably present. Vocal resonance and vocal fremitus are increased in Pneumonia, and the former may acquire a bronchophonic tone. They are both, as a rule, diminished over collapsed portions, without alteration of the quality of the vocal resonance.

Many of these points may, however, fail, and the distinction by the physical signs alone is not always easy. Under such circumstances, observations on the temperature materially assist the diagnosis.

Whether the condition of the patient be pyrexial or not, collapse alone will neither give nor increase fever ; and the presence of consolidation, together with the supervention or increase of fever, is at least an indication that some portions of this are due to true pneumonic changes.

Among other indications, the following, taken in conjunction with the pyrexia, are also of value in the recognition of the pulmonary disease : The change in the characters of the cough from a paroxysmal and painless to a short, dry, and painful one ; the acceleration of the respiration and of the pulse, and the increased distress and restlessness of the patient, may also serve to point attention to the true nature of the disease.

When Pneumonia occurs in the lobular form in the course of bronchitis or measles, without the intervention of collapse, the diagnosis may be much more difficult. In both diseases the increase of the pyrexia, which rarely attains in the simple bronchitis of children a temperature exceeding 100° Fahr.,[2] or its continuance in measles beyond the period of the specific fever, should, even in the absence of distinct pneumonic dulness, afford grounds for a strong suspicion of pulmonary mischief, and particularly when moist bronchitic râles are also present.

The distinction of Broncho-pneumonia

16), and also by Weber (Path. der Neugeborenen, ii. 39).

[1] Ziemssen, loc. cit. 335.

[2] I have recently seen two cases in adults where a temperature of 105° was attained in the course of apparently uncomplicated bronchitis.

from the lobar form of the acute primary disease may in measles be occasionally difficult when the patient is seen for the first time after a large tract of lung has been invaded. If the affection be purely unilateral, the difficulty is further increased. In most forms of Broncho-pneumonia the affection exists in both lungs simultaneously, though seldom to an equal degree.[1] In some cases, however, when collapse has preceded the Pneumonia, the peculiar pyramidal form of the dulness may aid the diagnosis. Moist râles in the opposite lung, or in other portions of that affected, are a further clue. The characters of the pyrexia in the two affections are in most cases a valuable guide, but it must be borne in mind that in the fever of the acute primary form a crisis may occasionally be wanting, and that its subsequent course also may be sometimes protracted.

The diagnosis from pleurisy, under these circumstances, rests on the same grounds as that of the acute primary disease.

The diagnosis of Lobular Pneumonia from acute tuberculization, or the recognition of tuberculosis as a complication of the Pneumonia, may at times be very difficult. This is especially the case when the disease originates in simple bronchitis; and when the general dissemination of moist râles, accompanied by pyrexia, may often closely resemble the phenomena observed in disseminated miliary tubercle. In measles and hooping-cough, a febrile state associated with pulmonary symptoms developed in the course of these affections would raise a presumption of its acutely pneumonic character.

The rapid development of signs of consolidation, with or without antecedent collapse, will, under all circumstances, favor the diagnosis of Pneumonia, but especially so in the two latter affections.

As points of minor value may also be noted the fixity of the râles in cases of tuberculosis, while the dyspnœa is often more distinctly disproportioned to the physical signs than it is in Pneumonia. Usually also the strength is less markedly or suddenly prostrated in tuberculosis than in Pneumonia.

Even in the later stages the difficulties may increase rather than diminish, since the progressive dilatation of the bronchi may closely simulate the formation of cavities from softening tubercle. The emaciation also and loss of strength in protracted cases of Broncho-pneumonia may bear a great resemblance to the phenomena observed in the later stages of tuberculization.

[1] Ziemssen and Krabler remark, however, that a double pneumonia may occasionally occur in measles so acutely as to be undistinguishable from the primary "croupous" form (loc. cit. 169).

Under such circumstances the observer must often remain in doubt, though his opinion may be influenced by the previous health of the patients, by their constitutional or hereditary predisposition, and by the evidence or not of the existence of tubercle in the lymphatic glands.

The Prognosis of Broncho-pneumonia is of much greater gravity than that of the acute primary disease. Ziemssen records thirty-six deaths in ninety-eight cases in children, and in nine more various sequelæ remained. The mortality of Steffen's cases, also in children, amounted to forty-one out of seventy-two cases; in six of these, however, the lung affection was complicated by heart disease, noma of the external genitals, tubercle, pleurisy, and meningitis. Steiner records a mortality of one in three cases. The results of different authors vary as to the relative mortality of the disorder in the different diseases of which it is a complication;[1] but, as a rule, the acute forms appear to be less dangerous than those running a more protracted and chronic course.

The age of the patients affected has, however, a marked influence on the mortality. Bartels says that Broncho-pneumonia after measles was fatal in all the children who had not completed their first year. From ætat. one to five the mortality was 39 per cent., and after the age of five years it was 37 per cent. The collective mortality of all the cases enumerated by Ziemssen under one year old was as 1 : 1, occurring in equal proportions in measles, bronchitis, and hooping-cough:[2] of the whole number of Steffen's cases the mortality before two years of age amounted to 54 per cent.

The condition that most unfavorably influences the prognosis is the weakness of the patient, not only as affecting the direct possibility of recovery, but also as predisposing to further collapse. This statement explains the mortality at very

[1] Ziemssen's data give the following percentage of mortality:—Measles, 25 per cent.; bronchitis and chronic bronchitis, 43 per cent.; and hooping-cough, 51 per cent. Steffen found the mortality from measles the greatest, amounting to five-sixths, but his cases of this disease were few in number, and affected children of very early ages. His mortality from the pneumonia of hooping-cough was as 8 : 10, and in bronchitis and chronic bronchitis, as 14 : 41 cases. Bartels' mortality in pneumonia from measles was 42 per cent., and he states that 80 per cent. of all the deaths in 573 cases of measles were due to the lungs.

[2] There appears to be some omission in Ziemssen's subsequent tables (p. 329), as the totals of those affected at different ages do not correspond to the whole numbers of his cases.

early ages, and is specially applicable to hooping-cough, where collapse, which is itself due in great measure to exhaustion of the muscular powers, is frequently the direct agent in the production of pneumonic changes; and hence the forms of Pneumonia occurring at late periods of the disease are not only indicative of loss of strength, but also predispose, by still further increasing the already existing weakness, to induce an extension of the pulmonary changes in which they originate.

For the same reasons a higher degree of fever, when of short duration, may be regarded as less unfavorable than a lower range of pyrexia, but protracted over a longer period. A temperature exceeding 105° Fahr. must, however, be considered as being of very serious augury. Among other symptoms which are to be regarded as unfavorable must be enumerated a great extension of the bronchitis over both lungs, signs of extensive collapse, increasing cyanosis, and diminished power of cough and expectoration, as shown by râles in the trachea and larger bronchi. To these must be added extreme frequency and particularly great feebleness of the pulse.[1] Somnolence and coma, indicative of mal-oxygenation of the blood, are also serious symptoms, not only in their direct indications, but also through the injurious influence produced on the respiratory muscles and on the heart by the impaired conditions of innervation of which they are the evidence. Convulsions in the later stages of the disease are a most unfavorable sign.

TREATMENT. — In the treatment of Broncho-pneumonia it must be constantly borne in mind that in the vast majority of cases this is a secondary disorder, and one whose very existence and mode of origin are very commonly indicative of weakness. This statement, which is applicable to all the forms of the disease originating in collapse of the lung, is hardly less true of the cases when, as in measles and diphtheria, the pneumonia originates by direct extension of the inflammatory process from the bronchial mucous membrane.

When this fact is remembered it is scarcely necessary to mention all measures of treatment calculated to depress the powers of the patients, such as abstraction of blood by bleeding, by leeches or by cupping, tartar emetic, calomel, and mercurials in general, except to state that their employment in such cases is only to be regarded with the strongest reprobation, since there is no doubt but that they tend to increase the mortality of the disease. Even when there is the temptation to abstract blood in order to relieve urgent dyspnœa, it must be remembered that the subsequent duration of the disease is long, that its tendency is to produce death by exhaustion, that all depletory measures diminish the muscular powers, and that by increasing the difficulty of expectoration they favor an increase of the collapse of the lung, which under such circumstances may speedily prove fatal.

In the earlier periods of the Bronchopneumonia of measles, unless the fever is severe, an expectant treatment, with the administration of nutritious food and the employment of salines, is all that is absolutely necessary. When, however, expectoration is difficult, and when the râles in the chest are abundant, and dyspnœa is marked, the occasional employment of emetics is productive of considerable relief. Of these, ipecacuanha in emetic doses is the most serviceable, since its administration tends, simultaneously with the evacuation of the contents of the bronchi, to favor a freer and looser secretion, and thus to ward off the tendency to collapse. The beneficial effects of emetics are seen in the relief of dyspnœa and in the diminution of cyanosis, and the temporary depression which they sometimes occasion is speedily recovered from in consequence of the relief which they afford to the breathing. If the cough is very troublesome and frequent, opiates may be cautiously used, particularly as the continued expiratory efforts, when prolonged and forcible, are among the chief agencies by which collapse is produced. This statement, though applicable to all forms of the disease, is especially true of those originating in hooping-cough; and in this disease other agents capable of diminishing the violence of the spasmodic cough, such as opium, belladonna, alum, zinc, or the bromide of ammonium, may be also employed with advantage. In the administration of opiates, however, narcotism is to be carefully avoided; and it must be remembered that this effect is easily produced in all diseases in which the aëration of the blood is impeded. Such an effect is also doubly dangerous through the diminished muscular power which it entails, and the doses given must therefore be in the minimum amount sufficient to allay the cough.

In cases where the expectoration continues difficult and the pulse is weak, sedatives may with advantage be combined with the carbonate and muriate of ammonia,[1] together with small doses of the

[1] Barthez and Rilliet record a case where the pulse could not be felt during many days.

[1] The preparations of ammonia are best administered to infants in milk. Mr. Squire has recently, at my request, made a number of experiments in order to discover the best method of disguising the unpalatable flavor of the muriate of ammonia, and has done so

vinum ipecacuanhæ (♏iij to ♏v), and with preparations of benzoic acid and of senega.

When prostration is more marked, wine or brandy should be given in doses proportioned to the age of the patient. For infants it is best to commence with brandy in doses of from 5 to 10 drops every two or three hours, gradually increasing both the quantity and the frequency of the dose, until a decided effect is produced on the pulse and on the respiratory movements. It may occasionally be necessary to give as much as ʒss or even ʒj every hour, to infants of a few months old, though it is very rarely that such an amount is required during long periods. Under its influence, however, both the pulse and the respiratory movements become slower, and the latter deeper and fuller; the convulsive movements are arrested; the prostration and semi-coma sometimes observed are diminished or disappear, and the pallor mingled with cyanosis gives place to a more natural color. It is most important that the employment of these agents should not be too long delayed; and when dyspnœa and prostration are extreme, the action of emetics may often be assisted by their administration. When from the intensity of dyspnœa deglutition has become difficult, it is occasionally advisable to administer stimulants combined with beef-tea, or with Liebig's extract of meat, or with egg or milk, by the rectum, until the patient has rallied, and the state of depression is alleviated. Under these circumstances also, quinine and the preparations of bark may often with advantage be combined with the expectorant remedies. If diarrhœa exists, it must be carefully combated by astringents, and by bismuth combined with these and with small doses of opium.

Stimulating liniments or the application of mustard poultices to the chest are advisable in all these conditions, but the employment of blisters is highly undesirable, since they weaken the patient, and in children are liable to cause a dangerous sloughing of the subcutaneous cellular tissue.

The method of treatment by cold compresses applied to the chest has received the strongest encomiums both from Bartels and Ziemssen, and from their statements and observations it appears to be one of the most valuable of our remedial agents, particularly when the fever is high; nor does any danger appear to arise during its employment from any extension of the pulmonary disease. It appears to operate favorably in two directions, both by increasing the strength and depth and by lessening the frequency of the respirations, and also by the reduction which it effects in the temperature—a result which appears unattainable by any other agent, at least in an equal degree.[1]

Bartels particularly insists on the benefit derived from the first deep inspirations excited by the application of the cold compress to the thorax, in promoting the expansion of the lung, and warding off the threatened danger of increasing collapse.

The reduction of temperature which follows their application is also very remarkable. In eight hours Bartels witnessed a fall from 105·25 to 96·8° Fahr., or of more than 8° Fahr.;[2] and Ziemssen, within seven and a half hours, observed in the rectum a fall of 5·8° Fahr. This effect is not, however, permanent, for after a few hours' intermission of the treatment the temperature again rises, and the application of cold requires therefore in some instances to be continued during some days before the temperature is permanently reduced to the normal standard.

This treatment needs to be carefully watched, since when it is prolonged without intermission, a dangerous degree of depression may ensue. It is, however, rarely observed until after several hours' application of cold; but in a case recorded by Ziemssen it was noticed within half an hour after the treatment had commenced. The face under such circumstances becomes pallid, the eyes sunken, the skin cold, and the pulse small and almost imperceptible. This alarming state is said by Ziemssen speedily to disappear on the temporary intermission of the cold applications, and neither in five cases of this nature observed by him, nor in a similar one occurring in an infant aged only thirteen months, recorded by Bartels, did any further injurious or fatal consequences ensue, and the treatment was repeated with favorable results after the depression thus excited had passed off.

Although the first application of the cold compresses is often disagreeable to the patients, a remarkable improvement usually appears speedily in their general state. Both the pulse and the respirations fall in frequency, and the former becomes fuller and the latter deeper. The pulse may fall from 170 to 130, and the respirations from 80 to 34 in the minute. The appearances of cyanosis simultaneously diminish, and the patient, previously restless, often sinks into a sound sleep while

very successfully by combining it with the tinct. limonis and sp. chloroformyl.

[1] Digitalis has little or no effect on the temperature of Broncho-pneumonia, and only very rarely has it any influence in lessening the frequency of the pulse.

[2] This result was obtained in the axilla, and is therefore less trustworthy than Ziemssen's as regards the general effect on the temperature of the body.

still enveloped in the cold wet cloth. With the intermission of the applications the pulse and respiration again increase in frequency, simultaneously with the rise of temperature which is then observed.[1] It is probable, as Bartels has remarked, that these favorable effects are not attributable solely to the artificial abstraction of heat, but that they are also due in part to a diminished destruction of tissue throughout the body, and that thus the production of an excess of carbonic acid, for the elimination of which the diseased lungs are incompetent, is also favorably held in check.

The favorable effects of this system are strikingly shown by Bartels' results; for whereas under other methods of treatment he lost, in the pneumonia following measles, seventeen out of twenty-six cases, or 65 per cent., the mortality after its adoption, and when no other remedies were employed, amounted to only thirteen out of forty-two cases, or little more than 30 per cent. In some cases, even when it was employed, the duration of the pneumonic consolidation was, however, very protracted, extending in one instance to eight weeks.

Under all the circumstances of the disease, the hygienic treatment of the patient requires to be carefully attended to. Fresh warm air, and the avoidance of draughts, are most important points to be insisted upon, and flannel should be worn next to the skin.

During convalescence the same precautions are to be observed, and the liability to relapse must be constantly remembered. It may be necessary during some months to enforce the extremest precautions against causes of catarrh, and during the winter months a confinement within the house, but in well-ventilated rooms, which should have a southern aspect, may be absolutely necessary in the case of children of delicate constitutions, or in those whose strength has been much reduced by the disease. Careful attention to diet, and the maintenance of the nutrition of the patient, are also most important. The administration of cod-liver oil and of preparations of iron, and small quantities of wine sometimes given two or three times daily, are often necessary to complete restoration; while in many instances a change of air, particularly in children brought up in large towns, is the most effective remedy that can be employed.

When the consolidation becomes more chronic, and is attended with profuse secretion from the bronchial mucous membranes, and particularly when dilatation of the bronchi exists, as shown by coarse râles in the chest, the same method of treatment must be sedulously followed. The administration of stimulants must, however, be pursued with caution when any tendency to pyrexia persists; and under these circumstances, when the weakness of the patient appears to require their employment (a condition frequently observed), it is well to administer them during apyrexial periods of the day, which must be carefully ascertained and subsequently watched by the aid of the thermometer.

The use of inhalations, and particularly of turpentine, has under these circumstances been tried by Ziemssen, and with some favorable results.

[1] Even in the most advanced and seemingly hopeless cases, and when the eyes appeared insensible to the stimulus of light, Bartels observed a gradual return of power, and a finally favorable result, after the adoption of this method.

Other Forms of Secondary Pneumonia.

Pneumonia occurring in the course of Bright's Disease may in some cases present no special variations from the characters observed in ordinary acute Pneumonia. In others it may begin, as before stated, in collapsed portions, resembling more or less in its course and characters the Lobular Pneumonia of children. Even when this is not the case, the characters of the primary disease are modified by this complication. The pyrexia is usually moderate, but the sputa tend to be thin and watery, and there is a considerable liability to œdema of the lungs and to consecutive gray hepatization. The tendency to pericarditis is also, I believe from my own experience, increased by this complication.

Rosenstein[1] has observed that when Pneumonia supervenes in Bright's disease, the total quantity of the urine is diminished, but that, in contradistinction to what is observed in other conditions, the amount of urea and the specific gravity

[1] Path. Therap. Nieren. Krank. The following are the results of Rosenstein's analyses:—

Day of pneum.	Amount cc.	Density	Nacl. gram.	Urea gram.	Album. gram.
1st	600	1013	2·4	3·6	1·8
2d	650	1013	2·47	4·37	3·25
3d	600	1012	2·10	4·5	2·4
4th	700	1012	2·24	5·85	2·8
5th	700	1012	2·8	....	2·1
6th	580	1012	2·32	4·98	1·7
7th	190	1013	0·76	1·04	0·95

Death on the seventh day from suppuration and œdema of the lung.

still remain below the normal standard. M. Jaccoud has drawn attention to the fact that a low specific gravity with diminished water and a minor amount of urea may aid in the diagnosis between chronic Bright's disease complicated by Pneumonia, and the cases where albuminuria occurs as a complication of the primary disease, but in which, nevertheless, an excess of urea is commonly present and the urine retains a high specific gravity.

The dangers of this complication have been already alluded to.

Rayer[1] remarks that Pneumonia occurring as a complication of diseases of the urinary organs, associated with alkaline urine, has the tendency to render this secretion acid, and his statement is confirmed by Grisolle.

The Pneumonia occurring in the course of the Acute Febrile Diseases has its features materially modified by the special symptoms of these, and presents in consequence so many variations that no general description will embrace the whole of the phenomena observed.

In *typhoid fever* it usually commences during the later stages. Its invasion is rarely marked by rigors, but commonly by a rise of temperature above the standard previously maintained. Fuller data are wanting on the subject of its further course. In cases which I have observed, the phenomena of crisis were absent, and when improvement has taken place it has been by a gradual fall of temperature, which may only occur after the pyrexia of the primary disease has subsided; the resolution of the infiltration also is often slow and protracted. Greatly increased prostration and asthenia attend this complication. The pulse and respiration are accelerated and their ratio is perverted, and the increase in the rapidity of breathing, together with that of the pyrexia, may be the first indication of the changes in the lung. Cough may be almost entirely wanting, and the rusty sputa are, as observed by Louis, comparatively rare. The insidious mode of invasion of Pneumonia in these cases renders a frequent examination of the chest necessary in all cases of continued fever. The Pneumonia commonly assumes the anatomical characters of red hepatization, but the tissue is softer and more gorged with blood than in acute primary form. Various stages of transition to gray hepatization are also found.

The Pneumonia arising from *diseased heart* presents also in many cases the features which are most characteristic of catarrhal or broncho-Pneumonia. This is especially evident in cases of marked disease of the mitral valve. The congestion thus produced in the bases of the lungs may be so extreme as to give rise to dulness on percussion, but the respiratory murmur at this stage is blowing rather than tubular. There is almost always chronic cough, with frequent exacerbations, until finally a more acute attack supervenes, attended by œdema of the lungs, and accompanied by coarse râles. The sputa are bronchitic, clear, watery, or frothy, sometimes blood-stained, but rarely distinctly rusty or tenacious. The dulness gradually progresses, and the breathing becomes more bronchial in character; and these physical signs, accompanied by increased vocal fremitus and by intensified though rarely by bronchophonic vocal resonance, often appear in scattered patches, which may vary in site from day to day. Rigors are hardly ever observed as the pneumonic changes progress: the invasion is gradual, and the temperature is often scarcely elevated even when the pneumonic consolidation is considerably advanced.

Portions of lung thus affected are found intensely œdematous, but airless; the section is smooth or indistinctly granular, and the pneumonic consolidation usually begins in patches of variable size, in which all gradations of the inflammatory changes may be observed. They finally, however, tend to coalesce and to pass into the condition of gray hepatization, yielding, from the œdema present, an excessive amount of turbid fluid on pressure.

The frequency with which Pneumonia occurs in the course of *other chronic and exhausting diseases* has been already referred to. It is usually of the hypostatic form, and tends to appear in scattered nodules in the midst of congested tissue; and, as before stated, it is not improbable that in many cases it is produced through the intermediate mechanism of collapse. The nodules are very soft and friable, often whitish, and distinct, and break down easily into a pulpy débris.

Its invasion is rarely preceded by rigors; cough and sputa may be alike absent, and the only evidence of the disease until detected by the physical signs, are the supervention of pyrexia—commonly moderate in amount—and some acceleration of respiration. Pneumonia is under these circumstances, very frequently the direct agency by which a fatal termination is induced.

The Pneumonia from *pyæmic conditions*, or Metastatic Pneumonia, has been already described in this work under the head of Pyæmia. (See Vol. I.)

The treatment of these forms of Secondary Pneumonia rests upon the same principles as have been described as applicable to all the adynamic forms of the acute primary disease. Briefly, it may be described as consisting almost exclu-

[1] Maladies des Reins, i. 573.

sively in the administration of sufficient quantities of stimulants and support. In the Pneumonia of the continued fevers, these indications are especially called for, and considerable amounts of alcoholic fluids must sometimes be given in order to sustain the patient's strength.

In Pneumonia complicating heart disease, digitalis may often be given with advantage in moderate doses, when the pulse is rapid and small; but the administration of stimulants is by no means to be omitted. The carbonate and muriate of ammonia may also be used with benefit under these circumstances.

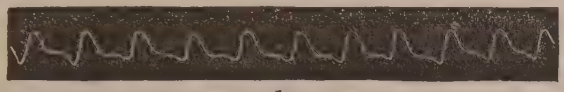
APPENDICES TO ARTICLE ON ACUTE PNEUMONIA.

Appendix A.

ON THE PULSE IN ACUTE PNEUMONIA.

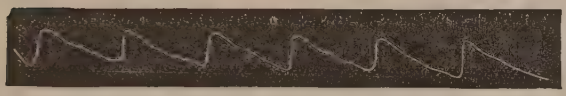
The accompanying wood-cuts represent the chief forms assumed by the pulse in various stages and in different degrees of severity of the disease.

The first three were taken from a man, aged thirty, with consolidation of the



1.

2.



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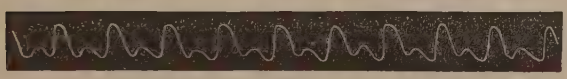

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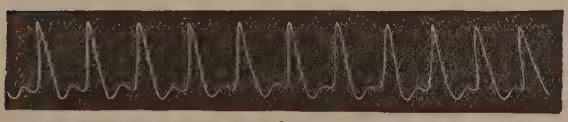

6.

lower two-thirds of the right lung, and they depict the gradual improvement following the crisis and during the administration of stimulants.

No. 1 was taken on the eighth day, when the temperature was 104·8, and when great prostration was present. The frequency of the pulse was 110. It was distinctly jerking and excessively compressible. The number of the respirations was forty.

The tracing shows a slight tendency to dicrotism. The recoil is rapid, and the curve with the convexity downwards, corresponding to the normal condition of arterial tension, is almost entirely absent.

On the evening of that day the first marked remission took place by a fall to 102·8, and by the ensuing evening the temperature had fallen to 98·4, above which no further rise ensued.

No. 2 represents the pulse on the ninth day of the disease and after the temperature had fallen to normal, and the patient

had taken during twenty-four hours rather more than three ounces of brandy, in doses of ʒij every two hours, with carbonate of ammonia gr. iij every four hours.

The arterial tension and also the cardiac power are shown to be greatly increased, by the prolongation of the recoil, and by the even, gradual, downward curve of the descending line. The tendency to dicrotism has also almost disappeared.

On the eleventh day, as seen in No. 3, the pulse had nearly regained the normal standard. The same treatment had been persisted in throughout this period, though the brandy was given at the longer intervals of from three to four hours. During this period the pulse had fallen to 72 and the respirations to 30 in the minute, and on the following morning the normal proportion of 84 to 20 was regained in their ratios.

No. 4 is a tracing taken on the fifteenth day of the disease, from a man of dissipated habits, who was accustomed almost daily to drink excessive quantities both of beer and of spirits. The Pneumonia involved the lower two-thirds of the right lung. The disease in this case ran a protracted course. An imperfect crisis took place on the ninth day, but the fever returned, and only subsided on the twenty-second day, with occasional slight subsequent exacerbations occurring until the thirtieth; on the fifteenth day, when this tracing was taken, the temperature was 100°, the pulse 92, and the respirations 32. Puriform sputa, indicative of gray hepatization, appeared early; and constant delirium with intense prostration, and profuse perspirations, were prominent symptoms throughout the case. Large quantities of brandy were necessary from the outset, and from the eleventh to the twentieth days (including therefore the time when this tracing was taken) brandy was administered in doses of an ounce and a half every hour continuously. The pulse was very weak throughout, and was frequently intermittent. The tracings show great deficiency in cardiac power and arterial tension, but dicrotism is not observed here.

Tracings 5 and 6 are those taken on the sixth and seventh days in a case ending fatally on the tenth day. The Pneumonia was double, affecting nearly the whole of both lungs. Pericarditis was also present. The pulse tracings show an extreme degree of dicrotism, and in No. 6 "hyperdicrotism" (Anstie) is seen in the line of the recoil falling below the level of the rest of the tracing. The temperature on these days was respectively 104° and 104·8°. The pulse was 120, and the respirations 60 in the minute. Brandy was given freely in this case, but not in the same amount as in the last instance.

Appendix B.

On the Retention of Chloride of Sodium in the System, and its Presence in the Sputa in Pneumonia.

A diminution of the quantity of chloride of sodium in the urine is common to a great number of febrile diseases. It is not, however, constant in them, nor is total suppression invariably observed in cases of Pneumonia. It would appear, therefore, that it is governed by some of the general laws of pyrexia; and although in Pneumonia the sputa and also the inflamed pulmonary tissue are found to present a considerable amount of chloride of sodium, as pointed out by Dr. Beale, yet this is hardly sufficient to account for the deficiency below the normal average (177 grains, Parkes). Dr. Beale found in one case that while the urine was absolutely deficient in chloride of sodium, the amount contained in the sputa was 10 per cent. of the solid matters. Dr. Beale thought that the chlorides were attracted to the inflamed lung as a consequence of the rapid cell-formation taking place there. When the chlorides were reappearing in excess in the urine, a similar excess was found in the serum of a blister, amounting to 8·93 of the solids, which Dr. Beale attributes to re-absorption from the lung during the period of resolution. (It must be remembered, however, that the serum of a blister is also an inflammatory product.) In a case ending fatally, the following proportions of chloride of sodium were found by Dr. Beale in different parts:—

Chloride of Sodium.	Per cent. of solids.
Urine	0
Blood from heart	0·68
Hepatized lung	2·59
Healthy lung	1·43

It would appear desirable that some analyses of the blood should be made during the period of absence of the chlorides from the urine.

I subjoin analyses of the urine and sputa in a young adult man, the subject of double Pneumonia, in whom, however, the temperature did not rise to any marked height. The analyses of the urine were conducted for me by my friend and then clinical assistant, Dr. Poore; those of the sputa were conducted by Dr. Meusel, assistant to Prof. Williamson, in the Birkbeck Laboratory in University College. The case is so far complicated that until the tenth day the patient took daily 40 grains of hydrochlorate of ammonia, which would probably increase the amount of chlorides both in the urine and sputa. It will be seen, however, that in

the early days the amount excreted in the sputa by no means complemented the deficient excretion by the urine. No cause could be assigned for the diminution of the urea on the eleventh day. A similar decline will also be noticed in the urinary chlorides from the eleventh to the fourteenth days. The patient perspired freely during this period, and possibly a considerable amount of chlorides may have been thus eliminated by the skin, though the amount of the water of the urine was less affected than the chlorides during this period.

Day of disease.	Temp. max.	Urine, amount in c c.	Sp. gr.	Urea. Grains.	Chlorides. Grains.	Sputa Chlorides. Grains. Total.	Total excreted chlorides in urine and sputa. Grains.
3d day	102·8	.....	.....	.....	.....	17·6	.....
4th "	103	.....	.....	.....	.....		.....
5th "	101·2	970	1020	598·68	11·19	15·7	19·7 (est.)
6th "	101·4	870	1020	535·72	16·786		24·586 "
7th "	101·5	1450	1019	792·71	50·204	5·37	55·574
8th "	100	1210	1020	670·82	64·142	6·0	70·742
9th "	99	1560	1020	792·79	95·896	6·34	102·236
10th "	100	1830	1016	605·86	119·658	3·803	123·461
11th "	99·8	1510	1015	488·33	36·806	2·78	39·586
12th "	99	1370	1015	506·35	71·610	.....	.....
14th "	98	1860	1015	657·20	48·756	.....	.....
15th "	97 (?)	1380	1013	382·33	147·955	.....	.....
16th "	98	2135	1012	509·58	50·880	.....	.....
17th "	98·5	1800	1011	388·20	157·850	.....	.....
18th "	98·5	2050	1010	473·5	115·808	.....	.....

The amount of urea subsequently varied between 385 and 462 grains during the succeeding five days. On admission the right base alone was affected, the left became implicated on the fourth day. Free perspiration commenced on the seventh day and continued on subsequent days. The sputa, which at first were copious, on the ninth day had lost their rusty tint, and had become bronchitic in character; on the tenth day they were much diminished in amount, but on the eleventh some rusty tint remained. The physical signs had only completely disappeared on the thirtieth day.

Appendix C.

The cause of the granular appearance of a pneumonic lung has been a subject of much dispute. The question will be found discussed at length in the works of most writers on the subject of Pneumonia, particularly by Laennec, and also by Andral, "Clin. Médicale;" Chomel, "Lec. Clin. Méd., Pneumonie;" Dr. Williams, art. "Pneumonia," Cycl. Pract. Med.; Addison, "Works," Syd. Soc. Ed.; Hodgkin, "Morb. Anat. of the Mucous and Serous Membranes," vol. ii. It was by these writers, in varying degrees, attributed to swelling of the walls of the air-vesicles, to interstitial exudation in the walls, and to the filling of the air-vesicles themselves with exudation materials. The merit of having first distinctly asserted in this country that the exudation took place into the interior of the air-vesicles, is claimed for Dr. Addison, and Dr. Hodgkin admitted that his views on the question had been changed by Dr. Addison. Dr. Addison's statements on this subject are, however, somewhat contradictory, as it would appear from his writings that he regarded the solidification of the lung during the stage of red hepatization as due to the swelling of the walls of the air-vesicles (loc. cit. pp. 8, 21), and that even gray hepatization was attended with a similar change (loc. cit. p. 22), and that at a later stage the softening of the walls admitted "of an albuminous material being poured into their cavities." In another passage, however, he states (loc. cit. p. 18) that "Pneumonia has its original and essential seat in the air-cells of the lungs, and that the ordinary pneumonic products are poured into these cells." Dr. Addison's editors and former pupils asserted that he distinguished red hepatization from gray hepatization by the fact that the former consists in the swelling and gumming together of the walls of the air-cells without effusion into them, and that the latter consists of an albuminous effusion into the cells. (Editor's preface, loc. cit. p. 25.) This distinction cannot now be regarded as tenable; for in the first stages of Pneumonia the inflammatory products accumulate in the interior of the vesicles, and the walls are unaffected except by vascular hyperæmia. In the recognition of the intra-alveolar exudation, Addison was, however, according to Virchow (Ges. Abhand. p. 725), preceded by Lobstein (Arch. Méd. de Strasbourg, 1835, No. 1).

Virchow (loc. cit.) states that the intensity of the granular appearance depends on the solidity of the exudation, and that it is less marked in the lungs of children, of old people, and also in dogs, because the exudation in them is commonly more fluid in its consistence. The granular appearance may, however, be distinct in the lungs of children, though on a finer scale than in adults. It may be still a question whether this appearance may not be in part due to the post-mortem coagulation of fibrinous and other materials, which during life are semi-fluid; and whether the exudation matter in the lungs may not undergo changes similar to those which ensue after death in other organs, such as the liver, the spleen, and the muscles (as shown by Kuhne's researches), through which they acquire increased firmness after life has ceased. Toulmouche (Gaz. Méd. x. 489) found in pneumonic lungs examined very shortly after death, that a quantity of fluid blood escaped from the cut surface.

Appendix D.

THE ORIGIN OF EXUDATION AND OF CELL-PRODUCTS IN INFLAMMATION.

The account of Cohnheim's researches may be found in Virchow's Archiv, vol. xl. 1867. It is due to earlier observers to state that although Cohnheim has by means of woorara found an admirable method of observing the escape of the white corpuscles through the walls of the blood-vessels, and has reduced it to a true demonstration, he was, however, anticipated in his observation by Dr. Addison of Brighton (Exp. and Pract. Researches on Inflammation, 1843; Healthy and Diseased Structure, 1849), by Dr. Waller in 1846 (Philosoph. Mag. vol. xxix. pp. 271–338), (I am indebted to Prof. Sharpey for this fact), by Zimmermann (Prager Vierteljahresch. 1852, vol. xxxv. p. 145), and still more recently by Dr. Lionel Beale (Microscop. Journ. xii. 1864). Dr. Beale describes the so-called white cells of the blood as multiplying *in loco* from the germinal matter of the nuclei of the capillaries, and he states that portions of this germinal matter pass through the capillary walls, and grow externally into cell-forms in the exudation. If I rightly interpret Dr. Beale's view expressed in other places on this point, he regards the solidified parts of the exudation as the "formed material" produced by "germinal matter"—an opinion corresponding in some respects with Virchow's that fibrinous exudations are the product of tissues in an excessive state of nutritive activity. Virchow, indeed, believes that in most instances the connective tissue, from the close relation which it bears to the lymphatic structures, is the origin of fibrinous exudation (Gesch. Abhand. p. 137). Buhl, however (Sitzungsbericht der Akad. der Wissensch. zu München, 1863, vol. ii. p. 59), has argued that, when this exudation occurs on mucous surfaces, the material so produced may be the result of transformations effected in the blood-plasma by the agency of epithelial structures. Virchow (loc. cit., and also in Archiv, vol. iv. p. 310, and in Spec. Path. Therap. vol. i. art. "Entzündung") has pointed out that the exudative processes of inflammation have a close analogy to secretions, and that the fibrinous exudations are at times more or less interchangeable with those in which a material resembling mucin is formed; and further, that all degrees of transition, in respect to the qualities of the exudation, may be observed between catarrhal and "croupous" inflammations. These statements have a great interest and an important bearing in the processes observed in Pneumonia, where these transitions in the nature of the exudation may be observed in its different stages. They serve also to show that the boundary-line between the so-called croupous and catarrhal forms, on which some recent authors have especially insisted, is by no means so sharply defined as is now sometimes believed; while in the latter, as noticed by MM. Herard and Cornil (Phthisie Pulmonaire, p. 135), a true fibrinous exudation may be occasionally observed.

Appendix E.

ON THE TREATMENT OF PNEUMONIA BY VENESECTION.

As it is still at least theoretically maintained by some that the statistics of cases of Pneumonia treated by venesection show a superiority for this procedure over other methods, it appears desirable to give a short sketch of the principal data which are accessible on the subject. All statistics on this subject are more or less beset with fallacies, but the final conclusions to be drawn from them appear to me to be those which I have stated.

The arguments in favor of venesection rest chiefly on the data given by Louis[1] and Grisolle,[2] with whom also may be ranked Wunderlich,[3] who has recently supported the same view.

The weight of Louis's and Grisolle's argument goes to show that cases bled early, within the first four days, have a more speedy recovery than those bled at

[1] Rech. sur la Saignée.
[2] Loc. cit.
[3] Arch. Phys. Heilk. 1856, xv.

later periods. Louis stated that cases bled within the first period had an average duration of seventeen days, those within the second, an average duration of twenty days; and in a second series he contrasted the duration under the same circumstances, as being in the first instance between twelve and fourteen days, and in the second between fifteen and eighteen days. Grisolle states that in patients bled in the "first stage," convalescence began on the tenth day and was completed within three weeks; while in those bled in the second stage, convalescence began on an average on the twelfth day, and they were discharged on an average on the twenty-second day. Both Louis and Grisolle date convalescence from a day or two after the cessation of the fever. If, however, these data are compared with the periods at which it is shown that the fever naturally tends to decline without active interference, it would appear not unjustifiable to infer that, regarded from the "positive" side, this evidence has no bearing on the absolute value of early bleeding, though demonstrating the relatively injurious effect of late bleeding in the disease.

Looking at the general results of these test cases, we find, however, that the mortality under Louis[1] amounted to 32 out of 107 cases, or 30·8 per cent., while that of Grisolle in 233 cases was 15·8 per cent., or 10 per cent. for the earlier bleedings, and 17·5 per cent. for the later; a mortality which in Louis's cases is vastly in excess of the average results of an expectant treatment, and in Grisolle's is only so to a less degree.

For an absolute comparison of the results of the bleeding and non-bleeding plan by the same individual, the most authentic data are those of Grisolle, Wunderlich, Huss, Dietl, and Dr. Todd. The two former have attempted to show that a number of cases treated by venesection have on the whole a shorter duration and a more favorable course than those in which no abstraction of blood is practised. Grisolle's data only rest on a comparison of eleven mild cases left to absolute expectancy (*including a rigorous French diète*), and thirteen of the same type treated by bleeding. In the former, he states that the convalescence only began on the tenth day, and the disappearance of the physical signs was protracted to the twenty-second or thirtieth, while in the latter the fever disappeared on the average on the seventh, and the physical signs on the twelfth days. These data have a certain incontestable value, but the number of cases is too small to weigh largely in the balance of evidence derived from the natural history of the disease.

Wunderlich's evidence is also in favor of bleeding. He gives a total of 190 cases, with an average mortality of 11·57 per cent.; 76 were treated without bleeding, with a mortality of 17·10 per cent., and 47 with bleeding, with a mortality of 6·38 per cent. The data as to sex, age, and complications are, however, here also very imperfect.[1] In contradistinction, however, to the almost universal evidence of other authorities, he considers that in eighteen cases of those bled on the first or second day, there was an almost immediate cessation of the pneumonic process in ten (*i. e.* crisis on the second,[2] third, and fourth days), and in five more a diminution of the pyrexia.

Traube (Ueber Krisen und kritische Tagen) had before asserted that as the natural tendency of Pneumonia is to a crisis in the uneven days, active therapeutic interference by emetics or bleeding was likely to induce the crisis at these periods.

Thomas,[3] however, as the result of his researches, made eight years later than Wunderlich's, but in the same hospital, asserts that bleeding has little or no influence in the reduction of temperature, and that the course of Pneumonia is identically the same, both under "active" and indifferent treatment; and the same result had been before arrived at by von Baerensprung,[4] one of the earliest observers on this subject. Thomas further points out that the effect of bleeding shortly before the crisis is in some cases to pro-

[1] Louis's own data afford one of the best evidences of the fallacies inherent in this class of statistics, for the percentage of mortality in his first series is greater in those bled during the first four days than in those bled later, in the proportions of 42·8 to 25 per cent.; a fallacy which Louis himself pointed out and rightly attributed to the comparatively advanced ages of the patients who constituted the former class.

[1] Wunderlich gives collectively 114 cases in whom local bleeding (58) (?), general bleeding (47,) and spontaneous hemorrhage (9) occurred. The evidence deduced from the latter is, however, almost valueless; in seven of these epistaxis occurred simultaneously with the crisis; in two females menstruation took place at an early period of the disease, and in one of these the crisis followed its cessation. Wunderlich gives no collective data, but only selects typical numbers; *e. g.* he only analyzes thirty-two of the seventy-six cases where no bleeding was practised.

[2] This result is certainly remarkable when compared with the average frequency of the crisis on the third day. The amount of blood drawn was from seven to sixteen ounces.

[3] Arch. Phys. Heilk. 1864. It may be worth remarking that Thomas's data respecting critical days, before quoted, do not *include* these cases of Wunderlich's, though made in the same hospital. Thomas states that his observations date from 1860. Wunderlich's paper was published in 1856.

[4] Müller's Archiv, 1851, p. 174.

duce an unnaturally low temperature after this has occurred. As such extreme depressions of temperature are almost always associated with marked prostration, this result of venesection can by no means be considered a desirable one.

Dietl's observations have been before alluded to, but they afford very strong arguments against the utility of venesection. His first observations[1] on 380 cases gave the following results :—

	Venesection.	Tartar emetic, large doses.	Expectant.
No. of Cases	68	106	189
Mortality per cent.	20	20·7	7·4

Dietl's statistics, in exact opposition to Grisolle's, show that cases left to a purely expectant treatment have a shorter duration of the pyrexial period, and a more rapid convalescence than those treated either by bleeding or tartar emetic, while the age of the patients and the complications present appear to have been nearly equivalent under all the systems tried. In a later publication[2] he gives the statistics of 750 cases (412 males and 338 females), all treated without venesection in the four years 1847–50, with a mortality of 9·2 per cent. Many of these were complicated (389 cases), including all the fatal cases; a large proportion (515 cases) suffered from "severe" dyspnœa. In the majority of those who recovered, the pyrexia lasted only from five to eight days, and the convalescence in most, from seven to fourteen days.

Huss's observations, extending over a period of sixteen years, brought him to the conclusion that during the time in which bleeding was indiscriminately practised in the hospitals of Stockholm, the mortality was greater than after it had been discontinued. During the former period of eight years, from 1840 to 1847, when venesection was generally employed, the mortality was 11·54 per cent., and during the succeeding eight years, from 1848 to 1855, when it was rarely resorted to, it was 10·21 per cent.; or the mortality of the former period exceeded that of the latter by 1·33 per cent. The average duration of the disease in those who recovered during the latter period was also shorter, being 20·9 days in the former, and 18·12 days in the latter.

The contrast of the effects of the two systems of treatment on the duration of the disease was markedly greater in the females than in the males; the average duration in the former being 7·6 days shorter in those not bled, while in the latter the difference was only 1·83 days.[3]

Huss asserts that there was no difference in the character of the pneumonias admitted during these periods, and he concludes that the difference is to be attributed solely to the influence of treatment.[1] Huss further adds that, in his

that the corresponding numbers of the two sexes during these periods were as follows:—

1840 to 1847.	1848 to 1855.
Males . 773 cases.	Males . 1195 cases.
Females 147 "	Females 220 "

[1] I have analyzed Huss's tables, to see if any difference in age of the patients treated could have had any influence on these results, but the subjoined table, constructed from his, would appear to negative the possibility :—

Age	5—10.		10—20.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847 Bleeding	3	33·3	104	7·6
1848 to 1855 Non-bleeding	6	0	125	4·8

Age	20—30.		30—40.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847 Bleeding	430	6	321	10·5
1848 to 1855 Non-bleeding	611	5·2	495	10·8

Age	40—50.		50—60.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847 Bleeding	125	22·4	49	18·3
1848 to 1855 Non-bleeding	238	16·7	76	23·7

Age	60—70.		70—80.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847 Bleeding	7	14·2	1	0
Non-bleeding 1848 to 1855	22	27·2	3	66.6

The totals of those above and below æt. 40 may be represented thus :—

	Under 40 Years.		Over 40 Years.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847	858	9·5	182	20·8
1848 to 1855	1201	7·4	341	19·3

It will thus be seen that a minor degree of mortality exists both for cases below and

[1] Der Aderlass in der Lungen Entzündung.

[2] Wien. Med. Woch. 1852. Schmidt's Jahresb. 1852, lxxvi. p. 30.

[3] An analysis of Huss's statistics shows

opinion, treatment by bleeding disturbs the natural tendency of the disease to crisis, a result before pointed out by Baglivi and adopted by Grisolle.

If we look at the effect of large bleedings indiscriminately practised, we see an enormous excess of mortality attending the treatment. Many of the data of these are very contradictory, but some are all but conclusive.[1]

Andral's mortality amounted to more than half his cases, or thirty-six out of sixty-five. Of the uncomplicated cases, nine were bled in the first stage of congestion, and two died; of thirteen bled in the second stage, five died; of seven bled in the third stage, *all* died; and of thirty-six complicated cases, twenty-two died.[2] The mortality of Bouillaud, according to his own report, was only 11 per cent., according to Pellatan's 12 per cent. Louis's mortality we have already seen. That of Chomel, according to Louis's report, was 32 per cent. Rasori's—a treatment complicated with enormous doses of tartar emetic—gave a mortality of 22 per cent. The mortality of Broussais is given at 68 per cent.

Many will contrast with these the results of Dr. Bennett before alluded to, which even if considered exceptionally favorable, demonstrate that during a period extending over sixteen years a very large number of cases of Pneumonia, taken indiscriminately, *may* recover perfectly without venesection,[1] in the absence of serous complications, although presenting in some instances the most marked forms of dyspnœa and lividity of face, associated with double Pneumonia, or an extensive affection of one side involving in fifteen cases the whole of one lung. They further show that the period of convalescence and the duration of the disease do not exceed, and in many cases fall very short of, those observed when venesection is practised. Nor can we refuse to admit the conclusive evidence of his facts adduced from the same field of observation, that while the mortality from Pneumonia in the Royal Infirmary of Edinburgh, prior to 1848, and when large bleedings were practised, was 36 per cent., this diminished during eight years when bleeding was less employed to 21 per cent., and in the following nine years to 11 per cent., while in Dr. Bennett's own practice it has only amounted to 3 per cent.

Dr. Todd[2] also pointed out that while the mortality from Pneumonia treated by bleeding combined with the use of tartar emetic amounted to one in six cases, this under a treatment by salines, nourishment, and support was only one in nine.

The argument that Pneumonia has changed its type and has acquired of late years a more asthenic character than it formerly possessed, is one on which exact data are necessarily wanting.[3]

above 40 in the second half of this period of sixteen years, though the number of cases in both instances is larger than in the preceding period, but that in the later decennial epochs of life, after ætat. 50, the mortality, as shown by the first table (in the second period of non-bleeding), absolutely *appears* to be greater. This is, however, probably fallacious, owing to the smaller number of cases on which they are calculated, since, in the face of the positive evidence to the contrary, it would be absurd to believe that venesection is relatively less dangerous at advanced ages. The data in Huss's tables give no means of forming further accurate comparisons on the influence of sex as compared with age, or of the complications present. The former, as far as I can gather from his tables, appears to be immaterial—the latter remains unanswered; but in dealing with such large numbers the probable influence of this cause of fallacy in the comparison is reduced to a minimum. The fluctuations in the mortality in different years appear, as already pointed out, to have been almost as great in the latter as in the former period.

[1] How uncertain such data may be appears from a communication made by Skoda to Dr. Balfour, that in 1840 he treated sixty-four females by large bleedings and tartar emetic, with only *one* death, but that in the same year the deaths among the males brought this average mortality to 12·5 per cent. (Brit. and For. Med. Chir. Rev. 1846, xxii. p. 590.)

[2] Analysis by Dr. Markham.

[1] Nine of Dr. Bennett's cases were bled before he saw them, and to an extent varying from twelve to thirty ounces; sixteen more were subjected to limited bleeding by leeches or cupping: the amount so lost is calculated by him as varying from one and a half to eight ounces. These cases had not a more favorable course than those not so treated.

[2] Clinical Lectures, p. 310.

[3] It is impossible to give more than a very superficial sketch of the able and elaborate arguments which have been advanced in this controversy. They will be found for the most part contained in the Edinburgh Medical Journal for the years 1856–59, in papers by Drs. Alison and Christison, Sir Thomas Watson, Dr. Bennett, Dr. Gairdner, Dr. Balfour, and Dr. Mitchell. An admirable summary of them is contained in an article attributed by Dr. Bennett to Dr. Sibson, "The Blood-letting Controversy," in Brit. and For. Med. Chir. Rev. 1858. The question of the theory of the "change of type" in acute inflammation is fully considered and negatived by Dr. Markham in the Gulstonian Lectures for 1864, "Bleeding and Change of Type in Diseases," and also by Dr. Balfour, "Hæmatophobia, an historical sketch," Edin. Med. Journ. 1858. To the latter the author is indebted for much of the earlier history of the schools of opinion on this subject. To Dr.

Cullen's description of high fever and of a full bounding pulse applies to pleurisy as well as to Pneumonia, and instances of this class of symptoms in both diseases in young adults are not now, I believe, so very rare as they are sometimes stated to be. Such cases of Pneumonia, however, are those most likely to recover under any circumstances, and the statement that they "bear" bleeding better than the more adynamic forms of the disease is no proof of the utility of the treatment, but only of the minor degree of danger attending it under these circumstances, while there is abundant evidence that in the majority of such cases it is, to say the least, superfluous. Further, the soft and yielding pulse, which is the most common in Pneumonia, has been shown by Dr. Balfour, from Dr. Gregory's own records, to have been prevalent in his time, as now, but that it certainly formed no obstacle to his course of venesection. The argument of Drs. Balfour and Markham, that this asserted change of type was not recognized by some of the most acute observers then practising for nearly twenty years[1] after it was said to have begun with the epidemics of cholera and influenza in 1830 and 1832, and that bleeding was only discontinued after the experiment of an expectant treatment had proved its inutility, appears also a very forcible one. Bleeding was instituted and practised on the theoretical ground of humoralism, or on the mechanical (or "hydraulic," Balfour) ground of relieving the congested lung. It was held to be the almost universal remedy for fever and inflammation, irrespective of age or sex, and that at a period antecedent to the more perfect recognition of Pneumonia by means of physical diagnosis, which has been supposed to have extended the practice; but the final proof of experiment necessary to an inductive science was not applied by its advocates, and when thus applied the inutility of the treatment was immediately demonstrated. The analogy also of a change of type in fevers is a most doubtful one, since there is the strongest reason to believe that those referred to by Sydenham and others were not different manifestations of one disease, but were in reality the different forms of typhus, typhoid, and relapsing fever, whose specifically diverse nature was not recognized until the writings of Sir W. Jenner. Dr. Balfour's historical researches have proved that this question is by no means a new one, but that it has descended to us from the followers of Pythagoras as opposed to those of Galen, and that even in the last century the same argument was advanced when the opponents of venesection had demonstrated its inutility in acute disease; Dr. Markham has also shown that it was supported by no less an authority than John Hunter. The opinion that such a change of type has taken place within more recent periods is further controverted by contemporary though indirect evidence. Laennec stated that the success of Dumangier in the treatment of Pneumonia without bleeding was equal to that of Corvisart, who bled freely; and Dr. Balfour observes that at the very time Dr. Gregory was practising his enormous bleedings, Laennec asserted that the treatment of Pneumonia by tartar emetic alone had reduced its mortality to 3 per cent. The argument also involves this remarkable paradox, that a disease in its asthenic form is, in the abstract, vastly less dangerous than when presenting a sthenic type; a paradox utterly confuted by our daily experience, not only of this, but of all other inflammatory diseases. This paradox appears in some of the ablest arguments advanced in support of the theory of a change of type in acute disease, since one of its most eminent advocates hails with satisfaction some signs of a return of the sthenic character.

The history of the origin of the change of treatment from venesection to a milder system also militates strongly against this view. Skoda and Dietl commenced their investigations on the results of expectant treatment on purely experimental grounds, and the former to the present day denies[1] any recognizable change of type in the forms of Pneumonia observed by him.[2]

As a final conclusion of the argument, it must, the author believes, be admitted on the evidence brought forward, that at no period since A. D. 1700 has blood-letting in Pneumonia been shown to be a general necessity in the disease; and that although on more than one occasion since this date a change in the vital characteristics of the disease has been asserted, in order to explain the recovery of patients suffering from it, on whom no venesection was practised, yet that no valid proof has been afforded that such a change has really at

Bennett's work on Pneumonia, and to Dr. Sibson's article, the author is indebted for many valuable statistical contributions.

[1] Dr. Balfour cites Dr. Alison as writing in 1844 ("Pathology"), that bleeding was the most important remedy for Pneumonia.

[1] Allg. Wien. Med. Zeit. viii. 1863. Schmidt's Jahrb. cxx. 34.

[2] A denial also maintained by Bouillaud, who is stated by Dr. Bennett to pursue his system of venesection *coup sur coup* with unabated energy, and with the fullest belief in its success; while Grisolle, on the other hand, though still holding venesection to be the best treatment, asserts his belief that "la constitution médicale est moins inflammatoire qu'il y a vingt ans."

any time taken place. [To indicate that I am not alone in maintaining that the use of venesection is not obsolete (but rather has of late partially revived) in American practice, I may quote some remarks made recently, in a discussion in the Philadelphia County Medical Society,[1] by Prof. Wm. Pepper: "If a patient is seen early, before hepatization has occurred, and while, although the central part of the affected area is probably so seriously damaged that fully developed inflammation will there occur, there is a zone surrounding this where the vessels are merely extremely congested, and where, if a prompt relief of this engorgement can be effected, proliferation and diapedesis (*i. e.*, inflammatory exudation) may be prevented. Now, at this stage I feel sure that prompt venesection will favor such a good result, and thus may possibly abort or, at all events, limit the extent of the inflammatory process. This same effect may be secured in a less degree, but with more safety, in cases where any doubt exists as to the propriety of general venesection, by leeching or wet cupping; but later, when the local disease is fully developed, venesection seems to me of doubtful propriety. The only advantage to be hoped for would be the relief of a laboring and over-loaded right heart, and this relief would necessarily be transient, since the mechanical cause would remain. It seems, therefore, that in most cases, after full development of hepatization, failure of the right heart from over-loading may be treated more successfully by other means than venesection."]

INTERLOBULAR PNEUMONIA. — INFLAMMATION OF THE INTERLOBULAR TISSUE OF THE LUNG.

This is the acute form, and in the human subject is a disease of the extremest rarity. Dr. Hodgkin[2] alludes to it, and it has been figured by Sir R. Carswell.[3] Dr. Stokes[4] also describes a case where "the substance of the lower lobe was completely dissected from its pleura by the suppurative inflammation of the subserous mucous membrane. This process also was found to have invaded extensively the interlobular and intervesicular cellular tissue, so as to cause this part of the lung to resemble nearly the structure of a bunch of grapes. All these nearly isolated lobules were surrounded by puriform matter, in which they hung from their bronchial pedicles." The exact condition of the vesicular texture is not described by Dr. Stokes, but his description would lead to the inference that it was in a state of hepatization. Rokitansky[1] has also described the disease in a form very similar to that met by Dr. Stokes.

In Dr. Stokes's case death took place on the twelfth day from the first symptoms of the disease. Large râles were heard over the site of the change, and the characters of the respiration led Dr. Stokes to suspect the existence of a cavity. Renewed rigors and copious sweating occurred on the seventh day, and were repeated up to the time of the patient's death.

I have seen one instance of this change in the interlobular tissue, caused by the direct extension of a post-pharyngeal abscess along the posterior mediastinum to the roots of the bronchi. There was effusion with recent lymph in both pleuræ. The interlobular septa of the lower lobe of one lung were greatly thickened and of a yellowish color, and were found to be the seat of a purulent infiltration. The lung tissue intervening between them, was condensed, but was otherwise healthy, with the exception of several pyæmic abscesses scattered through its tissue. Thrombi were, however, found in several branches of the pulmonary artery. In this case also there were considerable pyrexia and frequent rigors followed by sweating. Dulness on percussion existed at the base for nearly a fortnight, and was attended by weak bronchial breathing and by fine crepitation, mingled with fremitus in these situations. The physical signs present, however, cannot be referred in this case exclusively to the condition of the interlobular septa, since other complications were present.

It may be noticed as worthy of remark, that this implication of the interlobular septa, though so rare in man, is the ordinary appearance of the pleuro-pneumonia of the bovine species. A full description of its characteristics has been given by Professor F. Weber of Kiel.[2]

There is no evidence at present existing that such a condition precedes those thickenings of the interlobular septa which are occasionally observed to follow inflammation of the pleura, but it is by no means improbable that the occurrence of this process in a modified form may be the origin of such appearances to which further allusion will be made.

[1] Feb. 19, 1879; reported in Phila. Medical Times, April 26, 1879.]

[2] Mucous and Serous Membranes, ii. 149.

[3] Museum Univ. Coll. c.b. 573. In his manuscript account of this drawing, Sir R. Carswell states that the patient was a man aged 60, who died of disease of the bladder without pulmonary symptoms.

[4] Diseases of Chest, 144.

[1] Anat. Path. 1861, iii. 72.

[2] Virchow's Archiv, vi.

CHRONIC PNEUMONIA.

By Wilson Fox, M.D., F.R.C.P.

Synonyms. — Cirrhosis (?)[1]; Interstitial Pneumonia;[2] Lungen-Induration (Heschl), *German;* Sclerosis of Lung (Jaccoud); Fibroid Phthisis; Phthisie avec Melanose (Bayle); Scirrhus of Lung (Avenbrugger and older writers).

Definition.—A chronic induration of the pulmonary tissue, depending on a thickening of the walls of the alveoli by a fibrous growth, which causes a gradual obliteration of the cavity of the air-vesicles. This condition leads finally to contraction of the lung. It is commonly unilateral; it is frequently associated with dilatation of the bronchi; and it tends, either through ulcerations proceeding from these, or from secondary inflammation of the indurated tissue, to give rise to cavities in, or gangrene of, the lung. It is associated with dyspnœa, with cough, occasionally with fetid expectoration, and with hæmoptysis. The course of the disease is protracted, but it tends to a fatal issue after considerable periods, through impairment of sanguification, dropsy, diarrhœa, and gradual marasmus, or through acute intercurrent diseases affecting the opposite lung.

History.—The condition of the lung included under this title is one whose nature and pathological relations are as yet only imperfectly defined.

The views expressed by some recent pathologists respecting the inflammatory nature of the changes in the lung in many instances of phthisis would, if correct, necessarily involve the inclusion under this title of a very large proportion of cases hitherto regarded as tubercular, and indeed the estimate of the frequency of Chronic Pneumonia formed by different authors has varied largely with their opinions respecting the nature of tubercular changes. This division of opinion dates at least from the period of modern pathological research. By some authorities, and in particular by Broussais,[1] Cruveilhier,[2] Reinhardt,[3] and more recently

[1] Dr. Walshe, for whose opinion I entertain the most profound respect, and to whom as a former teacher I cannot sufficiently express my obligations, regards Chronic Pneumonia and Cirrhosis as independent diseases. The habits of inquiry which he taught his pupils will, I trust, serve as an excuse for one of them expressing an opinion on this point which differs in some respects from his own. The illustrations of the final effects of a pneumonia which has lapsed into a chronic state, appear to me to show that the result of the changes thus induced differs in no essential particulars from those which are met with in "cirrhosis" of the lung, in regard both to the induration of the pulmonary tissue and the dilatations of the bronchi, which so commonly are found in this state. M. Charcot is indeed disposed to make the existence of such dilatations a ground of distinction between the two diseases, but there is evidence enough to show that such dilatations are found in cases where induration has succeeded to an attack of Acute Pneumonia. They are not indeed so evident in the early as in the later stages of such cases, and induration found in the latter is only a progressive change; but it appears to be an inevitable consequence of the disease if sufficiently protracted. The question is in one sense a purely pathological one, but as far as clinical diagnosis rests on a pathological basis it is not without its significance. There is abundant proof that thickening of the walls of the air-vesicles, resulting in the complete obliteration of their cavities, is a final result of Chronic Pneumonia, and it is this condition which is described in all (the few) authentic cases of "cirrhosis." I have discussed at some length the possibility of its origin in idiopathic changes independently of such inflammatory action. In the light in which I regard this state, and with this explanation, I have ventured to use Dr. Walshe's recorded case of this disease, which is the most perfect extant, and also his no less admirable commentary, as an illustration of chronic pulmonary induration.

[2] The term Interstitial Pneumonia also appears to me etymologically to express only very imperfectly the real character of this affection. The most important secondary effect of chronic inflammatory action on the tissue of the lungs is the thickening of the walls of the alveoli, and not of the interstitial tissue. It is indeed a question how far the latter is implicated, at least primarily, in this process.

[1] See especially Examen, vol. i. Aph. 161 to 171; Hist. des Phleg. i. Proleg. p. liv. v. vi. ib. p. 3; Examen, iv. 245, 402; Hist. des Phleg. ii. 385. Broussais recognized a pulmonary non-tubercular phthisis, but he regarded tubercles as the result of inflammation or irritation of the lymphatic tissues.

[2] Anat. Path. Gén. vol. iv. 1862.

[3] Annalen der Charité, vol. i.

by Lebert,[1] all tubercular changes have been regarded as essentially inflammatory in their nature.

Others, with Andral,[2] who recognized only the softer and more opaque granulations as tubercular, have regarded the gray granulation of Bayle, which many now consider the type of "true tubercle," as the result of a Chronic Vesicular or Lobular Pneumonia. A third series of observers—among whom may be named Gendrin,[3] the late Dr. Addison,[4] and, more recently, Niemeyer[5] and Colberg[6]—maintain an opinion precisely the reverse of Andral's, and assert that the greater part of the softer "tubercles," and nearly all caseous changes found in the lung, are due to a Pneumonia which some of these authors have termed "cheesy" or "scrofulous." This view has also been in part supported by Virchow,[7] but it has been generalized by some recent writers to a wider degree than has been done by him.

It is undesirable in this place to enter further into the discussion of these widely diverse views.

They have however largely influenced, and particularly of late, the opinions expressed respecting some forms of induration of the lung classed under the head of Chronic Pneumonia, and even the descriptions given of this condition, and they appear to have caused not a little discrepancy of statement respecting its relative frequency.

Thus authors who, like Hasse, Grisolle, and Chomel, maintain the doctrines of Laennec respecting tubercle, assert that Simple Chronic Pneumonia is a disease of extreme rarity, and that it is hardly ever met with except when complicated with tubercles.[1] Grisolle[2] states that he has only met with six cases in twenty-five years, and only four where the acute disease passed into a chronic state; and Chomel[3] writes that in sixteen years, during which he performed nearly three thousand post-mortem examinations, he only met with two examples. Andral,[4] however, regarding the subject from a different pathological point, stated that he had met with the disease much more frequently than Chomel. Dr. Stokes[5] says that in his experience Chronic Pneumonia is a very rare affection, but that it is "difficult to define the meaning of the words Chronic Pneumonia, or to draw the line of distinction between it and that low irritation of the lung which is followed by tubercular infiltration." In the succeeding pages the author proposes to treat only of such forms of chronic induration of the lung as may be reasonably presumed to have been caused by processes in which tubercular changes have had no share. In this sense the disease is of great rarity, and examples of it can only be found in isolated cases scattered in different journals and in monographs on diseases of the lungs. The author's own experience would almost confirm the statement of Hasse, that it seldom occurs except in the presence of tubercles; for out of five apparent examples of the disease which have come under his own observation, in one only were the lungs found on microscopic observation to be free from tubercles. In the analysis of

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[1] Gaz. Méd. de Par. 1867. Sur la Pneumonie disséminée chronique.

[2] Prec. Anat. Path. ii. 518 et seq.; Empis, De la Granulie. See also Reynaud, Mal. des Bronches, Dict. de Méd., vol. vi.

[3] Hist. Anat. des Infl. ii. 334.

[4] Works, Syd. Soc. Ed. Dr. Addison's statements on this subject are somewhat conflicting, and some passages in his writings would almost lead to the conviction that he held tubercle to be an inflammatory product; *e. g.* loc. cit. p. 33: "Unless the simple transparent tubercle already alluded to can be considered as a separate and distinct body, there is not one of the varied morbid conditions coming under the denomination of tubercle which has not appeared to result from changes in or on the natural tissue. . . . These morbid changes have appeared to me perfectly identical with those of inflammation." "The immediate morbid changes produced by ordinary pneumonia and by phthisical disease are the same, with the exception of the albumen, ... being much more susceptible of organization, and consequently more likely to become permanent in the former than in the latter" (ib. p. 34). "If called upon to give an expressive name to tubercular phthisis, I should venture to designate the disease Scrofulous Pneumonia." In other places (*e. g.* p. 30), however, he treats of the gray granulation as occurring independently of inflammatory change; and at p. 49 he states, "However analogous and closely allied the abnormal condition which produces tubercle may be to that which constitutes inflammation, we cannot in the present state of our knowledge admit their identity." In another passage, however, he distinguishes two kinds of tubercle, a firm transparent, and a soft opaque form (loc. cit. pp. 49, 50).

[5] Lehrb. Spec. Path. Therap. Ed. 1868, ii. 233–5. Klinische Vorträge über die Lungen Schwindsucht, *passim*.

[6] Deutsch. Arch. Klin. Med. ii.

[7] Wien. Med. Woch. 1856. Die Krankhaften Geschwülste, vol. ii. pp. 600 et seq.

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[1] Hasse, Path. Anat., Syd. Soc. Ed., p. 225. This is admitted to a great extent by Prof. Niemeyer, but he explains the concurrence of cheesy products with tubercle by the theory that the tubercles are secondary to Pneumonia.

[2] Loc. cit. pp. 82, 338.

[3] Dict. de Méd. xvii. 223.

[4] Clin. Méd. iii. 491.

[5] Loc. cit. 353.

cases by other authors those cases will be spoken of as tubercular which present granulations—gray, or soft, or cheesy—in the lungs or other organs.[1]

Chronic Pneumonia, in the restricted sense in which it appears to the author desirable to employ this term, is found principally in the forms described by Andral,[1] of red, gray, yellow, and black induration.[2] The two former are almost invariably a direct consequence of a prolongation of the acute disease. The last-named is often found under circumstances which leave considerable doubt respecting its mode of origin, though in not a few instances it can also be referred to past inflammatory conditions. To these, perhaps, may be added the induration of lung occurring in connection with heart disease, and designated by Virchow[3] as the brown or pigmentary induration of the lung, syphilitic disease of the lung, and also certain rare conditions associated with non-tubercular ulceration.

[1] The author feels considerable diffidence in thus somewhat dogmatically criticising cases by other observers, and he is aware that exception may be taken to the view here expressed, which differs from the opinions entertained by many advanced pathologists of the present day, but which has only been arrived at by him after a prolonged and careful investigation of this subject. The question of the nature of tubercle underlies the whole of this question, and he can only shortly state here the opinion which he entertains, that tubercle as a growth is not only liable to "cheesy" degeneration, but that it is also capable of becoming a more or less permanent tissue by fibrous transformation; and the last-named change forms, in his opinion, a much more important element in the history of tubercle than is generally recognized. Also, that it consists of a multiplication of nuclei and cells in dense masses, the interstices of which are occupied by a delicate fibre network or by a solid intercellular substance; that this growth may be peri-bronchial and peri-vascular, but that it also appears in the walls of the air-vesicles; that when found in the latter situation, it is often, but not always, accompanied by a proliferation of epithelial cells of an inflammatory character in the interior of the air-vesicles; and that in a large proportion of the so-called "catarrhal," "gelatinous," and "scrofulous" pneumonias the cheesy changes found in the lung are accompanied by this "tubercular" infiltration of the walls of the alveoli; that these "cheesy" changes may occasionally be due to fatty metamorphosis of the epithelium, attended by destruction of the pulmonary tissue, but that in a far larger proportion of cases they are due to a true tubercular change, and that even when they are not the direct cause of such changes in isolated spots, tubercles are almost invariably found in other parts of the same lung, and also in other parts of the system. Patients whose lungs present this peculiarity of "cheesy" or "scrofulous" change, are therefore almost invariably those who are at the same time the subject of tubercle; and the author believes that he is correct in stating that in the vast majority of cases such "cheesy" changes occur under the influence of the tubercular diathesis, and are mostly associated with if not caused by the presence of tubercle. On the other hand, he is fully prepared to admit with Dr. Addison and Cruveilhier that a large proportion of the alterations in the lungs of such patients are due to attendant Pneumonia. This Pneumonia is commonly chronic, and when not destructive, it leads to a thickening of the walls of the air-vesicles by the growth of fibrous tissue. This thickening takes place by means of a fibro-plastic growth with elongated and fusiform cells, independently of the tubercular masses before described. Tubercular masses may, however, be mixed with these, and the two sets of changes may go on *pari passu*, while the tubercular growths may either soften and break down, or may themselves at later periods undergo the same fibrous transformation. Fibroid transformation of the lung tissue is therefore an exceedingly common event in tubercular phthisis, and forms in fact, in one sense, a mode of cure of tubercle, as has been long recognized. The mode of evolution of most forms of tubercular growth in the lungs is indeed closely allied to an inflammatory change, but it presents in addition other phenomena which are not ordinarily met with in inflammatory processes; and until the *purely* inflammatory nature of tubercle is more distinctly proved than has yet been done, it appears desirable, at least in a clinical sense, to maintain the separation of these processes. A discussion of this question is, however, impossible here. As regards the coexistence of "cheesy" changes in other organs being taken as an evidence of the tubercular nature of changes in the lungs, the author is fully aware that this subject is yet *sub judice*, but he believes that the discussions respecting it rather tend to show diversity of opinion respecting the nature of tubercular changes in general than that they affect the questions of the identity of these "deposits" with tubercular changes in other parts. Some recent writers, indeed, appear altogether to ignore the termination of tubercle in a "cheesy" metamorphosis; and forgetting that this is its most common change, and also that tubercle is the most common source of this pathological product, they appear anxious under all circumstances to prove its origin in some other process. The author hopes shortly to be able to lay before the profession in a more complete manner the grounds on which these opinions are based.

[1] Clin. Méd. iii. 489.

[2] Bayle (Rech. Phthisie Pulm. p. 12) described "engorgement" of the lung as a form of Chronic Pneumonia, but the nature of this must be regarded as doubtful.

[3] Archiv für Path. Anat. i. 463. This state is also alluded to by Andral (Prec. Path. Anat. ii. 517); Hasse (loc. cit. p. 227).

ETIOLOGY.—I have already stated, in the section devoted to the clinical history of the acute disease, that I have only known one case of Pneumonia where the patient left the hospital without a perfect resolution of the physical signs in the lung; but I have also given instances where this process was protracted.[1] I do not think that cases of the latter class, in which a somewhat tardy but progressive improvement takes place, can properly be called instances of Chronic Pneumonia. Huss, however, dates the tendency to pass into the chronic state from the fourteenth to the twenty-first day of the acute disease. He says that this protracted course is somewhat more common in Pneumonia of the upper lobes, and that the Pneumonia of drunkards has a similar tendency. Grisolle states that Libermann has asserted it to be common amongst opium-smokers in China, and Dr. Stokes considers that Chronic Pneumonia ending in induration of the lung is more common after the typhoid forms of the disease. Chomel attributed to excessive bleeding an injurious influence in protracting resolution. The pneumonia of the aged has also a similar tendency, particularly after the stage of gray hepatization has been attained. Circumstances interfering with convalescence, and fresh exposure leading to relapses, may also protract the course of the disease and give it a chronic character. Thus Broussais[2] gives three cases of induration of the lung from military hospitals, ending fatally on the twentieth, fifty-first, and ninety-first days after an attack of Pneumonia. In two of these, a condition of induration alone is mentioned, but in the second, the state described approaches closely to Andral's description of the red induration. Grisolle also states that the appearance of the lung, in cases of Pneumonia ending fatally within five or six weeks, presents but little difference from the characters of the acute stage,[3] though exhibiting a more marked degree of induration; the surface on section being somewhat smooth, but in other cases still presenting the granular character of the primary disease. The only case which I have met with of this nature was in a man, aged forty-six, the subject of chronic albuminuria: cough, with hæmoptysis, began two months before admission, but he was only compelled to leave off work a fortnight before admission into hospital. Dropsy in the legs had been present for six months. The sputa were thick, puriform, and uniformly blood-stained. The patient died suddenly three days after admission. The bronchi of both lungs were dilated. Both apices presented a gray infiltration, which was most marked in the left upper lobe, which was also considerably indurated; the kidneys were granular.

The condition of lung, however, most commonly described as Chronic Pneumonia, is that in which the pulmonary tissue has undergone a fibrous induration, more or less deeply pigmented, usually attended with complete obliteration of its vesicular structure, and commonly, but not constantly, traversed by dilated bronchi. It is this state which received from Sir D. Corrigan the name of "Cirrhosis,"[1] and which some modern English pathologists have regarded as the result of an idiopathic change, which has also been termed "Fibroid Degeneration of the Lung,"[2] or "Fibroid Phthisis."[3] The condition, has, indeed been long known. It was described by Morgagni,[4] and later by Avenbrugger,[5] under the title of "Scirrhus" of the Lung, and by Bayle as "Phthisie avec Melanose," and the last-named author recognized its occurrence independently of or complicated by tubercular disease:[6] the same condition was

[1] Also Andral (Clin. Méd. iii. 550). A case where the signs of consolidation only disappeared at the end of four months.

[2] Hist. des Phlegm. i. p. 13 et seq.

[3] Cf. a case by Bayle (Phthisie Pulmonaire, obs. 46, p. 373)—Pneumonia of three months' standing—red, firm hepatization; also a case by Durand-Fardel (Mal. des Vieillards, p. 589), where death took place after two months, and red hepatization was found passing in spots into gray; also (Ib. p. 594) a case of three months' standing, where gray induration existed at the bases, together with recent gray infiltration of one apex. Hourmann and Dechambre (loc. cit.) also speak of this protracted course as common. See also a case by Rayer (Gaz. Méd. 1846, p. 983), duration not stated; also a case by Grisolle (loc. cit. p. 72), of a patient dying on the sixtieth day, when transitions from red and gray hepatization to firmer degrees of induration were found.

[1] Dublin Journ. 1837. Dub. Hosp. Gaz. 1857.

[2] Dr. Sutton, Med.-Chir. Trans. xlvii.

[3] Dr. Andrew Clark, Trans. Clin. Soc. i. p. 174.

[4] Epist. section 23, xviii. section 30.

[5] "Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi," 1761. Trans. by Sir J. Forbes, 1824. He describes this state as having the consistence of cartilage. Scirrhus was the term universally applied by older writers to pulmonary indurations, however originating, as by De la Böe, Sylvius, and Bonetus. (See Waldenburg, "Die Tuberculose," pp. 30, 31, 42.) Avenbrugger does not seem to have described tubercles, though they were recognized before his time. Avenbrugger's commentator (Corvisart) has left almost as complete a description of the symptoms as any subsequently furnished.

[6] Phthisie Pulmonaire, p. 209 et seq. The first two cases are typical illustrations of this

also described by Laennec[1] as occasionally complicating dilatation of the bronchi, and as existing around tubercular excavations. The pigmented form was, however, included by him under the term melanosis, which he regarded as an independent disease, but which Andral first showed to result from a chronic inflammatory action.

The difficulty in arriving at a conclusion respecting the mode of origin of this state is, however, very considerable, owing to the length of time during which pulmonary symptoms may exist before death, and also in many cases from the incompleteness of the reports furnished. I have, however, analyzed thirty-nine cases[2] returned as "Chronic Pneumonia," "Cirrhosis," "Interstitial Pneumonia," or "Induration of the Lung," which are all that I can find in modern medical literature capable of throwing any light on the general bearings of this question. Many of these are more or less imperfect in regard to history or to pathological details, so that the facts thus gained are only of comparative value. As far, however, as they are available, I shall give the results in a numerical form.

Sex and Age.—Of these cases twenty-two were males and sixteen were females. In one case the sex is not mentioned. The *ages* at which death took place in thirty-eight cases are given in the subjoined table; but the smallness of the numbers involved and the uncertain duration of the pulmonary affection in many cases, greatly diminishes the value of these results. They show, however, that the disease materially shortens life, since nearly two-thirds of the patients died before attaining the age of forty. (*See* PROGNOSIS.)

AGES AT DEATH.

1 to 10.	10 to 20.	20 to 30.	30 to 40.	40 to 50.	50 to 60.	60 to 70.	70 to 80.
1	5	10	6	4	9	1	2

state, and were evidently, from Bayle's description, associated with dilatation of the bronchi. Bayle considered melanosis to result from a diathetic disease (loc. cit. 84).

[1] Forbes' Trans. 2d Ed. 1827, p. 112. Laennec's description of melanosis of the lung, under which title he also included melanotic tumors, contains one case of chronic black induration, associated with tubercle (Ib. p. 390).

[2] The cases included in this analysis will be enumerated in the Appendix at the end of this article. Both in the Appendix and in reference to special points I have marked such cases by *; cases not so included I have marked in my references by ‡. I have not included thirty-four cases tabulated by Dr. Sutton as instances of "fibroid degeneration of the lungs" (Med.-Chir. Trans. xlvii.), nor thirty-five cases of bronchial dilatation described by Biermer, many of which presented similar alterations (Zur Theorie und Anatomie der Bronchien-Erweiterung, Virch. Arch. xix.) Both these and Dr. Sutton's cases will be alluded to separately. In addition to these I have only been able to find fifty further cases where any allusion is made to this affection. Many of these are wanting in necessary details of history, or in descriptions of the other lung, or of other organs. Some which relate to cases of recovery, or which illustrate special points, will be again alluded to. I have not, however, included cases *described* as tubercular, but only such published as cases of "cirrhosis," "induration of lung," "interstitial pneumonia," or "chronic pneumonia," and Dr. A. Clark's published case of "fibroid phthisis." I have thought it best to retain in this category some of the cases which appear to me to have been tubercular in their nature, although not described as such, in order to express more clearly the fallacies inherent to this branch of the subject. It must, however, be noticed as remarkable from these numbers how very rare this affection is when uncomplicated with tubercle; and even some of the cases included in this analysis appear to have had a tubercular origin, or to have been thus complicated. Chomel based his description of the disease on eight cases, including two of his own, which were all that were accessible to him.

The great difficulty in the recognition of the true pathenogenesis of pulmonary indurations arise from the occasional impossibility of determining the origin of masses of cicatricial tissue in the lung, when all signs of the affection in which it originated have passed away. A cicatrix is not a disease, but represents the cure of a past disease, and it is only by a knowledge of the diseases which commonly produce such changes in this organ and of their attendant circumstances, that we can form any conclusion as to the probabilities respecting the antecedent conditions in which it may have originated.

There can, however, I think, be little doubt that in the majority of cases of induration of the lung found *post mortem*, whether occurring in isolated patches or extending over very considerable areas, the cause lies in the presence of tubercle and of tubercular pneumonia—using these terms in their wider sense to include all

forms of granulation ordinarily described as tubercular, and also most of the cheesy changes found in the lungs.[1] This condition has been long recognized, and the fibrous or indurating termination of tubercular processes has been fully described in most works on Pathological Anatomy.[2] So commonly is tubercle found as a complication of this state, that out of four cases quoted by Steffen[3] as examples of "Interstitial Pneumonia," three are most probably tubercular, and the fourth is not free from a similar suspicion. Out of the thirty-four cases of "Fibroid Degeneration" given by Dr. Sutton (loc. cit.), I should regard fifteen at least as presenting similar evidences of indurating tuberculosis, and eleven more as probably having been produced by the same condition, inasmuch as they presented this state as a double affection of both apices associated with cavities,[4] making a total of twenty-six. If indeed the indurated gray granulations, whether occurring singly or in masses, are, as Andral thought, the result of Chronic Pneumonia,[1] this hypothesis vastly extends the range of this affection; but this theory of their inflammatory origin is, I believe, just as applicable to the nature of tubercle in general as it is to this special form in which it is sometimes found *post mortem*.

Even out of the thirty-nine cases which I have analyzed, I regard eleven to have been thus associated. In four cases tubercles were found in both lungs; in four others, where the whole of one lung was indurated, they were found in the opposite lung, and in three they were found only in the affected side. Of three cases published by Sir D. Corrigan as instances of "cirrhosis," one was regarded by him as coming under this category, inasmuch as there were cavities in the affected side, and tubercular ulceration of the intestines.[2] Dr. Walshe also alludes to the possibility of "cirrhosis" complicating tubercular disease of the lungs.[3]

The question respecting the other pathological relations of this condition is, however, a complex one, and may be conveniently discussed under the following heads:—

(*a*) The evidence in favor of its origin either in Acute Primary or Broncho-Pneumonia.

(*b*) The evidence of its origin in inflammation of the pleura.

(*c*) The evidence of a simple chronic inflammatory action of the interstitial tissue of the lung, not preceded by either of the above-named acute conditions, and therefore akin to cirrhosis of the liver, or to the granular condition of the kidney.

(*d*) The evidence of an idiopathic "fibroid change" in the walls of the alveoli occurring independently of inflammatory action.

(*a*) The possibility of the origin of fibrous induration of the lung from an attack of Acute Primary Pneumonia is conclusively shown by a case of Andral's,[4] where the acute attack had occurred eighteen months previously, and where after death the lung of the affected side was

[1] The cheesy concretions formed by inspissation of puriform matter in the bronchi are, in my experience, much less frequent than is sometimes supposed.

[2] See especially Rokitansky's work. Sir D. Corrigan speaks of "cirrhosis" representing a species of cure for tubercle. See also Cruveilhier, "Tubercles de Cicatrization" (Anat. Path. liv. xxx. pl. iii. p. 6). He also gives a case where the whole of one lung was indurated by chronic tuberculosis (Anat. Path. Gén. iv. 631). In some cases, however, a microscopic examination will reveal, in cases of fibroid induration, evidences of tubercular growth which are undiscoverable by the naked eye. I have recently observed this in a case which clinically, as well as in the post-mortem appearances, presented a most typical apparent example of "cirrhosis," in the retraction and induration of nearly the whole of one lung with only a small nodule of induration in the other.

[3] Klinik der Kinderkrankheiten.‡

[4] It is undoubtedly true that bronchial dilatations may lead to secondary ulcerations in indurated tissues, but the proportional number of these when independent of tubercle is strangely small when compared with those given by Dr. Sutton. See especially Biermer's paper on "Bronchial Dilatation." Out of thirty-five cases, only twelve were associated with ulcerations of the bronchial mucous membrane, and of these seven were tubercular; while of the five remaining, two were examples of gangrene, and another was a case of abscess of the lung communicating with the bronchi. Barth also considered ulcerations of the bronchi as being very rare, having only met with three instances out of sixty-two cases. The possibility which may be argued that cavities may arise from obstructions of the bronchi only rests upon what must, when actually tested by observation, be regarded as an exceedingly small number of cases.

[1] It is under this title that Dr. Sutton describes most of these granulations, and he attributes the same opinion to Dr. Addison. Dr. Sutton has, however, carefully distinguished these cases, and has thereby avoided the confusion which might otherwise be caused in pathological descriptions when such a reservation is not adopted. It must be remembered that Andral regarded cheesy matter as the type of tubercle, which he believed to result from an inspissated secretion.

[2] Dubl. Hosp. Gaz. 1857.*

[3] Dis. of Lungs, p. 407.

[4] Clin. Méd. iii. obs. 64, p. 474.*

found universally indurated and traversed by dilated bronchi, in the walls of which a gangrenous action was taking place. The opposite lung was in a condition of recent hepatization; the other viscera were healthy. In addition to this instance, five other cases among those analyzed present a similar history, making a total of six, and seven others afford a strong suspicion of a similar origin. Thus, of the only three cases published by Sir D. Corrigan with post-mortem results, one began with an attack of influenza, and in another (the tubercular case before alluded to) the disease appears to have originated with a catarrh, attended with severe pains in the side. Similar evidence is also afforded by three cases reported by Weber[1] of children whom he had himself treated previously for Pneumonia; and he states that he was acquainted with two others still living, who, after attacks of Pneumonia, retained for years the physical signs of induration of the lung, with dilatation of the bronchi; and a similar origin is shown in cases reported by Ziemssen,[2] Reinhardt,[3] Dr. Addison,[4] and Biermer.[5]

The conditions of Catarrhal or of the Secondary Broncho-pneumonias, which are more liable than the acute disease to lapse into a chronic state, appear, however, to be more favorable for the production of this change, and it is not improbable that some cases of induration of the lung with dilated bronchi may owe their origin to this form of the disease. Bronchial dilatation is a common event in the Broncho-pneumonia of children, and this condition may persist in cases where the pulmonary consolidation, instead of resolving, passes into a condition of induration. This is shown conclusively by a very instructive case by Bartels,[6] and by two others reported by Dr. Bennett.[7] Another is afforded by Dr. Addison,[8] where the induration of the lung, associated with dilated bronchi, commenced with hooping-cough. Two others with less details are given by Steiner and Neuretter[1] as secondary to bronchitis, and Barth's[2] fourth case is probably an example of the same kind. If we consider the course of acute bronchitis in children, and recollect how constantly dilatation of the bronchi occurs in this condition, both in the idiopathic form of the disease and also in the course of measles and hooping-cough, it can only be a subject of surprise that permanent lesions of this nature are not more commonly met with as the results of these diseases. It has been already stated that the Pneumonia which attends them has a more prolonged course and undergoes a more protracted resolution than is observed in the typical forms of the primary disease; and it is probably owing in no small degree to the higher reparative powers of childhood that such indurations do not more commonly occur as the sequelæ of these affections. Two cases by Legendre[3] might indeed give rise to the question whether collapse of the lung, together with bronchial dilatation, may not subsequently lead to induration of the pulmonary tissue independently of pneumonic changes, particularly when we recall the statement of Rokitansky,[4] that fibro-nuclear growth in the alveolar walls tends to occur in cases of collapse of long standing.

The mere existence of bronchial dilatation, however acquired, appears to afford a predisposition to pneumonic changes, and to thickening around the bronchi, which may well explain a large proportion of the instances where these conditions co-exist, and when no definite history of their joint origin in a single attack of an acute affection can be obtained. The progress of interstitial thickening does not, however, appear to affect in this manner large tracts of lung when uncomplicated by other changes, though in some instances it extends inwards, through the interlobular septa from the pleura.[5] There can be no question that bronchiectasis and induration of the pulmonary tissue may

[1] Path. Anat. der Neugeb. ii. 58.‡

[2] Pleuritis und Pneumonie in Kindesalter, p. 257.*

[3] Ann. der Charité, i.‡

[4] Collected writings, p. 45.‡ The second of Dr. Addison's cases.

[5] See cases i.‡ and xviii.‡

[6] Virch. Arch. xxi. p. 144.‡ This case, where Pneumonia of the apex succeeded to measles, showed in the course of nine months some improvement in the physical signs, but persistent dulness remained at the apex, with signs of dilated bronchi.

[7] Rep. City of Lond. Hosp. for Dis. of Chest.‡ I have not been able to gain access to the originals of these cases. They are quoted at length in the Journal für Kinderkrank. 1858, p. 305. In both, persistent signs of consolidation of the lung succeeded to measles, and in one case lasted nearly four years.

[8] Loc. cit. p. 44‡. The first of Dr. Addison's three cases of induration of the lung.

[1] Padiätrische Mittheilungen, Prager Vierteljahresch. 1864, lxxxii. p. 22.‡

[2] Loc. cit. p. 501.*

[3] Rech. Mal. de l'Enfance, 223–283.‡ It appears, however, most probable from Legendre's descriptions, that these changes had been the result of a partially diffused Broncho-pneumonia. He applies to them the term "carnization," which, from its undefined meaning, has been a frequent source of confusion. Two cases of a very similar nature are cited in Legendre and Bailly's original papers on "Collapse" (loc. cit.). It is possible that they are identical with these.

[4] Path. Anat. 1861, iii. 50.

[5] Biermer, loc. cit.

reciprocally act as cause and effect to one another, and also that the process leading to induration may simultaneously give rise to dilatation of the bronchi. This explanation, however, fails to explain instances of chronic bronchitis when the clinical evidence of induration of the lung would show that this change is of more recent origin than the cough and expectoration, which in some cases date from an earlier period; and for these I think that the theory of a pneumonia secondary in point of time to the bronchial dilatation affords the best elucidation. The frequency with which such secondary pneumonias occur is variously estimated. Biermer's cases show that they were found in twelve out of fifty-four cases; Rapp (quoted by Biermer) found them in twenty-one out of twenty-four cases; and Barth in twelve out of forty cases.

The pneumonia attending bronchial dilatations is also commonly of the disseminated catarrhal type. It tends especially to occur around the dilatations, when it is frequently set up by the irritation arising from the retained and decomposing products of secretion, or by the direct extension of ulceration or inflammatory action through the bronchial wall. Such forms of Pneumonia are very liable to pass into gangrene, but where this is not the case, the persistence of their cause tends to diminish the possibility of a speedy resolution, and to produce fibrous thickening. Of this tendency several recorded cases afford very good illustrations, which may be regarded as almost conclusive of the nature of this process.[1] Pneumonia having this origin is insidious in its invasion, and does not produce the marked symptoms ordinarily presented by the acute form; and this probably explains some of the reported cases where the commencement of the induration cannot be referred to any single acute attack.

It will readily be understood that when this process has once been established, and when Pneumonia ending in induration has attacked a lung the subject of bronchiectasis, it tends to recur and to repeat itself in other parts of the same organ. The dilated bronchi surrounded by indurated tissue, being a *locus minoris resistentiæ*, are continually liable to become the seat of fresh catarrhal inflammation, from which the process extends to other divisions of the bronchi in the same lung. These in their turn excite disseminated pneumonic changes, which are again prone to the same indurating process. The disease thus tends to progress *saltatim* until the greater part of the lung is invaded. Bronchial dilatation may indeed exist, and apparently long, without giving rise to other changes than those caused by the compression which is produced by the enlarged tubes encroaching on the surrounding tissue, but the proportion of cases in which this state is found to exist alone and without attendant induration is comparatively small, amounting to only eleven out of the thirty-five cases reported by Biermer.

The unilateral character of these pulmonary indurations, which forms a remarkable feature in the history of the disease, and to which allusion will again be made, may also be compared with the frequency with which bronchial dilatations are found limited to one lung.[1] The frequent coincidence of the two affections is also very remarkable, for dilatations of the bronchi are stated to have existed in thirty-one out of the thirty-nine cases of pulmonary induration which I have analyzed;[2] while, conversely, on analyzing Biermer's cases I find that induration was present in twenty-four out of the thirty-five cases of bronchial dilatation reported by him; and Dr. Grainger Stewart[3] also regards it as a very common though not a necessary complication of this condition.

Lastly in this category belongs a very large proportion of those cases where induration of the lung is found in patients exposed by their occupation to the inhalation of irritating particles of solid matter, such as the Sheffield grinders, stonemasons, miners, potters, and cotton-workers. In some cases even of this class I am disposed to believe that tubercular changes may play some part in the production of the indurations discovered; but in others, and as far as is at present known, no evidence of tubercle has been shown to exist. It appears most probable that the passage of these particles into the air-vesicles, and their lodgment in their walls, set up a slow pneumonic process attended by a fibrous growth in the alveolar walls and septa, by which the indurations observed are produced. These diseases form a class which requires a separate consideration, but their relation to the origin of chronic pul-

[1] See Case iv. of Dilatation of Bronchi, by Laennec‡ (loc. cit. 113); also Biermer's‡. Obs. i. ii. xiv. xviii. xxi. xxiii. xxiv. xxix.; also Dr. Stokes, Dis. of Chest, p. 159.*

[1] Barth says that out of forty-three cases of bronchiectasis the affection was unilateral in twenty-seven. Biermer says that it occurs wite about equal frequency as a double or as a one-sided affection (Virchow's Handbuch, v., section i. 245). This is probably in part explicable from the number of cases in which it originates in pneumonia, pleurisy, or collapse. See Laennec's first case (loc. cit. p. 110‡), where unilateral bronchiectasis remained as the result of hooping-cough.

[2] In two others there is no sufficient account. In six only is this condition stated to have been absent.

[3] On Dilatation of the Bronchi, 1867.

monary induration is of no small importance in their features of pathological affinity.[1]

(*b*) Pleurisy again seems to be in some cases the exciting cause of this condition. One such case[2] occurs among those which I have analyzed, and Biermer[3] gives two others. The manner in which this effect is produced is somewhat doubtful. It is possible that in such instances Pneumonia may have complicated the pleurisy. Biermer attributes to pleuritic adhesions an important part in the production of bronchiectasis, but it may still be questioned if they are not rather the effect than the cause, though in some instances, however, there appears to be pretty clear evidence that they have been the first cause leading to the subsequent dilatation of the tubes. Some thickening may at times extend from the visceral pleura through the interlobular septa, but I do not think that any evidence at present exists that, except at the surface of the lung, such a process can extensively invade the alveolar walls of the pulmonary air-vesicles unaccompanied by an attendant Pneumonia.

(*c*, *d*) If now we turn to inquire whether any other conditions may exist tending to produce pulmonary indurations, we find that the number of cases in which such an explanation is required is remarkably limited. The cases in which either a history of phthisis, of acute affections of the lung, or pleurisy, may be inferred to have been the antecedents of this state, amount in those which I have analyzed to twenty-six out of the whole number. The great duration of some of the other cases would afford a probable ground of belief, that to many of these, where no history is obtainable, a similar explanation by the theory which I have raised of progressive attacks of Broncho-pneumonia is also applicable; and the probability of this will become more apparent when the pathology of the disease has been considered.

Morbid Anatomy and Pathology.—(1) The forms of *Red and Gray Induration* of Chronic Pneumonia have been already described as presenting but little difference from the appearances presented in the acute stage. Instead, however, of presenting the usual friability of a recently hepatized lung, they are firm and resistant, and are drier, and sometimes rather paler. The finely granular aspect persists during some time, but tends to disappear with the progress of the case. It may, however, be apparent on tearing the tissue, even when the section appears smooth. In some cases the tissue assumes a yellow tint, but without (from the descriptions given by Hope[1] and Lebert)[2] passing into a cheesy change; and this would appear to result from a gradual fading of the brighter tint of the red hepatization.[3] The induration in this state depends on a gradual thickening of the walls of the air-vesicles—a thickening which is commonly found in large tracts of the forms of Pneumonia associated with tubercle, as well as in the simpler forms. I have met with this chronic red induration of the base in one case only, and in this there were also masses of tubercular induration in the apex of the same lung, the other lung being free. The patient was an old woman with syphilitic cicatrices in various parts of the body, and a history of earlier syphilis. She had had hæmoptysis seven years before, and no distinct history could be obtained of the date of the invasion of the Pneumonia, but she was under observation for three and a half months with the physical signs of consolidation of the base. Pericarditis with effusion formed the immediate cause of death. The bronchi were dilated in spots of cicatricial contraction of the apex, where indurated tubercles were present, and also in the tract of red induration at the base. This tract (Fig. 36) showed on microscopic examination a dense fibre tissue consisting of a network interlacing in all directions, thickening the walls of the pulmonary alveoli, and spreading in all directions through them (*a*, *a*). The contents of the alveoli (*b*, *b*) were round nucleated cells mostly resembling the pyoid forms seen in the third stage of Pneumonia, but mingled with occasional epithelial cells, and with granular corpuscles and free fat granules. In places (*c*, *d*) the air-vesicles are seen to be almost obliterated by this growth, and in some tracts scarcely any traces of them were discoverable. There was compara-

[1] For an almost complete series of references to the literature of this subject, see Zenker, Die Staubinhalations Krankheiten der Lungen, Deutsche Arch. Clin. Med. vol. ii. Also ib. Seltmann, Anthracosis der Lungen. See also Peacock, Brit. For. Med.-Chir. Rev. xxv. 1860; Dr. Greenhow, Path. Soc. Trans. xviii. xx.; Dr. Hall, Brit. Med. Journ. March and April, 1857; Calvert Holland, Edinb. Journ. 1843.

[2] Dr. Peacock, Edinb. Journ. 1855, p. 281.*

[3] Cases v.‡ and xxvi.‡ Biermer's cases are for the most part merely pathological studies without any clinical history.

[1] Morbid Anatomy; "Yellow Induration."

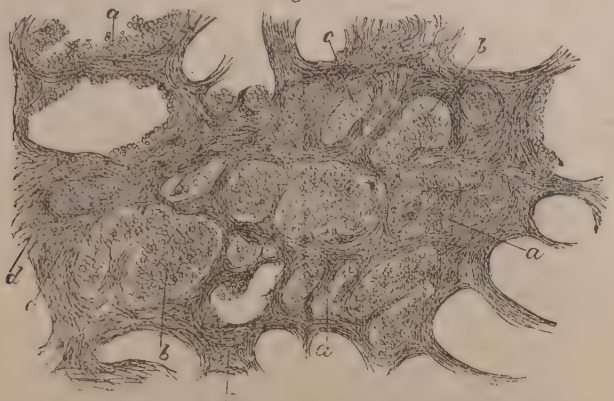
[2] Physiol. Pathologique, i. 137: "Yellow Hepatization."

[3] I have never seen this state. Hope and Lebert each only speak of one instance. Lebert's case was in a child, and the disease was of two months' duration. Another is quoted by Charcot from Monneret (case ii. loc cit. p. 30). The disease was here only of three months' duration.

tively little nucleated growth discoverable in the walls in this case. The process in this condition appears to be only slowly evolved; the growth and thickening of the fibres is gradual, and a rapid development of nucleated cells is not discoverable. In the earlier stages, however, this is sometimes seen as figured in Figs. 37 and 38.

The fibro-nucleated growth is commonly, as is seen in these figures, in the form of elongated fusiform cells, they are

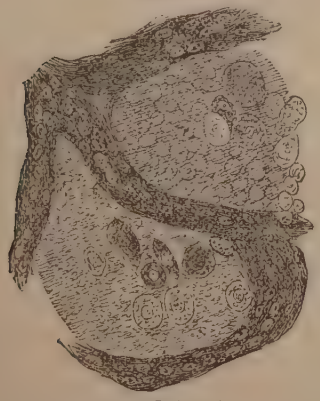
Fig. 36.



Chronic Red Induration in a tuberculous case.—*a*, *a*. Fibrous network in walls of alveoli. *b*, *b*. Round nucleated cells within alveoli. *c*, *d*. Air-vesicles, almost obliterated.

not densely massed, as in the tubercular growths. Heschl has convinced himself that the nuclei of the capillaries participate in the change, and my own observation would confirm his, inasmuch as all

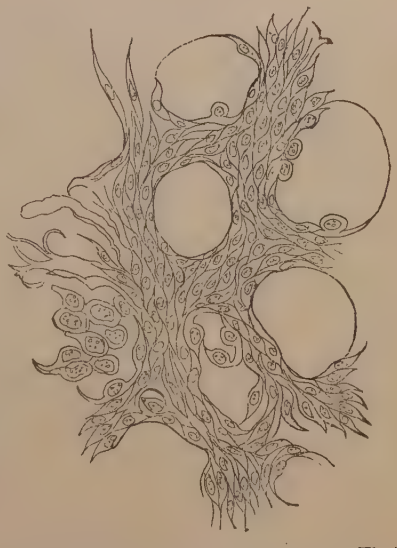
Fig. 37.



Case of Chronic Gray Induration associated with Tuberculosis, but without tubercle in this part. The contents of the vesicles are an amorphous exudation with few cell-forms.

the nuclei of the alveolar wall appear to multiply, and to yield fibrous elements. In tubercular indurations, the process may take place in a manner similar to those above described, and without any growths differing from the ordinary fusiform cells of the fibro-plastic type, or on the other hand they may be associated with a dense growth of nuclei characteristic of tubercle. In some cases, indeed (see Fig. 39), the two may be found proceeding side by side, and occasionally it may even be doubtful what the destination of the nucleated tissue thus originat-

Fig. 38.



From Heschl (Lungen Induration, Prager Vierteljahresch. 1856, vol. xii.). This is given by Heschl as the mode of growth of the dense fibrous induration, but his case also presented some reddish-gray and rus y granulations, though the tissue was indurated to the consistence of fibro cartilage.

ing may be, and whether it shall ultimately form a fibre tissue, or a tubercular mass. The latter, indeed, may finally shrivel by a species of fibrous transformation, or it may be the seat of cheesy trans-

formation or softening leading to the naked-eye appearance of scattered yellow cheesy masses in the midst of indurated tissue. I have only alluded to this mixed growth as an illustration of the frequency with which this combination occurs. With the exception of cases where tubercle is mixed with the indurating growth,

Fig. 39.

Mixed tuberculous and fibro-plastic growth. *a, a, a,* Alveoli filled with enlarged epithelial products. *b.* Recent tuberculous growth of round nuclei, imbedded in a fine alveolar network, mingled with masses of pigment. *c.* The same growing into the interior of an alveolus. *d.* Fibro-plastic growth of fusiform and nucleated fibre cells. *e.* The same mixed with round nuclei like the tubercular mass. (700.)

I believe that little and probably no histological distinction exists between the forms of indurating Pneumonia unassociated with tubercle, and those where the pneumonic process occurs in a lung in which tubercle is also present, but without the necessary formation of this growth in the inflamed portions.

Other authors have described the induration of the lung as depending on an infiltration of an amorphous substance between the interstices of the alveoli. This is, I believe, the condition described by Dr. Addison as the "iron-gray induration," or the "uniform albuminous induration," and also by MM. Bouchut and Robin.[1] My own observations have failed to show this condition. Dr. Addison's descriptions were anterior to the use of the microscope, and I believe that when this appearance is found in large tracts of indurated tissue, it arises from the thickening and fusion of large tracts of fibrous growth into a uniform semi-cartilaginous material, closely analogous to the tissue produced during earlier stages of ossification, and by a process which in the two cases presents very striking forms of resemblance.[1] The material occupying the interior of the alveoli is often mainly amorphous, particularly in the forms of

[1] Mal. des Nouveaux-nés, Ed. 1852, 371. Their description is quoted by Grisolle and Charcot as the type of the process. Bouchut and Robin describe this state as being very frequently associated with gray granulations. In the sense in which I have used these terms I regard such cases as instances of tubercular Pneumonia. It must be remembered that Robin, whose descriptions Bouchut gives, does not regard the gray granulation as a form of tubercle, but as a product *sui generis* —a view further developed by Empis, "De la Granulie." This confusion meets us at every turn in relation to this subject. Bouchut, however (loc. cit. p. 386), says that he has twice seen acute Pneumonia pass into the chronic stage. See also Lorain and Robin, Comptes Rend. Soc. Biol. 1854, 2d ser. i. 62.

[1] This formation of tissue with dense fibrous bands is an exceedingly common complication of the fibrous forms of tubercle. It is beyond the limits of this article to enter into a minute histological description, or to give further illustrations of the processes by which this result is obtained. I hope shortly to be able to give in another place a fuller description of these changes.

the "gelatinous infiltration" of Laennec, as seen in Fig. 37, which is equally liable, with the other forms of Pneumonia, to undergo the same thickening of the walls of the alveoli; but cell-products mingled with a variable amount of exudation may also be seen in them.

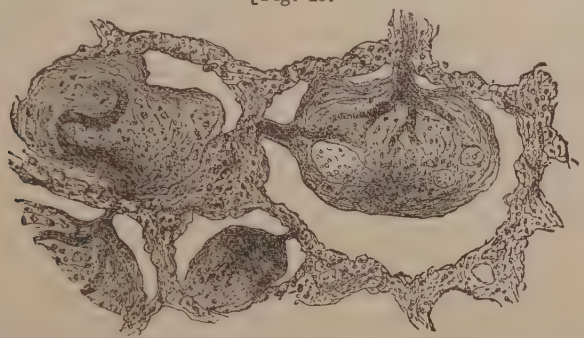
(2) *The Gray, Black, or Fibroid Induration* of the lung presents a further stage than those last described.

In the former cases the lung may retain apparently its natural volume, but when the change now in question has been undergone, it is almost always shrunk and diminished in size, to a degree proportioned to the extent of the process.

The period in which this change and the loss of the ordinary characteristics of pneumonic consolidation may follow an acute attack, varies in different instances. Grisolle reports a case where the transition between the two forms was apparent within sixty days, and the first case of Sir D. Corrigan's,[1] of three months' duration, still showed by its color traces of its origin. A case of Charcot's,[2] however, showed marked gray induration, with black mottling, in less than three months from the acute attack.

In characteristic cases of this nature the cut surface of the lung is smooth and glistening; it is hard, and creaks like cartilage, or resembles the tissue of the uterus. It tears with the greatest difficulty, and no longer presents the granular appearance of ordinary Pneumonia.[3] No fluid can usually be expressed from this tissue. The surface is homogeneous, except where traversed by dilated bronchi or by dense white lines, which may rep-

[Fig. 40.



Chronic Pneumonia.—Vascularization and fibroid development of intra-alveolar exudation products. Bloodvessels are seen in the exudation products, which bloodvessels communicate with those in the alveolar walls. The alveolar walls are also thickened by a fibro-nucleated growth. X 100, and reduced ½. (Green.)]

resent either these tubes when obliterated, or thickened and obliterated bloodvessels, or which may arise from thickening of the interlobular septa. In some instances, when the disease is less advanced, and particularly when the induration appears to have been secondary to bronchial dilatation, these bands tend to pass as thickenings around the larger bronchi, and thence to extend into the surrounding tissue. The tissue is variously pigmented, and the irregular dissemination of black coloring matter among the white fibrous growth gives it a marbled gray appearance, which is very characteristic. The alveolar texture of the lung is entirely destroyed, though portions may still be found which show traces of pulmonary tissue, and representing earlier stages of the process. In general, however, the indurated parts, except when occurring around dilated bronchi, are pretty sharply circumscribed; and the change is usually lobar, or it affects the greater part of a lobe or the whole of one lung.

The state of the bronchi in the affected lung is somewhat variable. In the majority of cases they are dilated, this condition being mentioned in thirty-one out of thirty-nine cases. In eight only is a negative stated. Charcot says that neither in his, nor in Monneret's, nor in Hardy and Behier's cases was this dilatation present, but these must to some degree be regarded as exceptional, and Charcot's own cases refer to earlier stages of the disease. In some instances, when the bronchi have been found dilated in both lungs, induration has been discovered in one only,[4] but usually in such cases the dilatation is greatest on the indurated side. In other instances the dilatation has been general throughout a single lung, a portion of which only has been found occupied by the indurated tissue; while in a third and most common form, the dilatation of the bronchi has been limited to the indurated part.

[1] Dub. Journ. 1838.*

[2] Loc. cit. p. 19 (Charcot's third case).*

[3] Laennec (loc. cit. 233) described indurated portions around gangrenous excavations as presenting an appearance of granulations resembling the eggs of insects. I should regard these as indurating tubercles.

[4] See Ziemssen's case, before quoted.

The origin of this dilatation of the bronchi has been a subject of much discussion. In some it is, as before explained, extremely probable that it has existed prior to the induration; and in others, as in the form of Broncho-pneumonia of childhood, the two may not unfrequently originate simultaneously. In acute primary Pneumonia, however, dilatation of the bronchi is a rare event, and its absence is probably due to the consolidation of the pulmonary tissue preventing their enlargement within the period at which death usually occurs in this disease. When the Pneumonia passes into the chronic form, which is attended with retraction of tissue, various explanations have been offered of the mechanism of the process.[1] This subject, however, belongs rather to the history of the dilatation of the bronchi than to that of Chronic Pneumonia. Sir D. Corrigan attributed it to a compensatory dilatation of the tubes, in order to fill the space within the thorax left by the contracting lung. It appears, however, to me to be most probable that the mechanism of this condition is similar to that in which bronchial dilatation takes place under other circumstances, and that it is mainly due to the expiratory force of cough acting on tissues which in the earlier stages of the disorder are softened, and have lost their elasticity through the inflammatory processes going on in them. It must be remembered that though in the later stages the fibrous tissues formed in this process have a tendency to shrink and contract, they are still deficient in natural elasticity. This defect persists even after they have consolidated into a denser material, and the subsequent contraction would rather have a tendency to diminish the calibre of the bronchi than the reverse. It is less easy to explain the occasional absence of such dilatations, but much would depend on the degree in which the bronchial walls participate in the inflammatory softening, and possibly also on diversities in the rapidity of induration with which we are not yet familiar.[2] In Chronic Tubercular Pneumonia, dilatation of the bronchi is a very common phenomenon, but it is not always easy to decide whether it has been prior or subsequent to the pneumonic changes.

The extent to which this bronchial dilatation may proceed is sometimes very remarkable, and the enlarged tubes may constitute a considerable part of the bulk of the retracted and shrunken lung.[1] The form of the dilatation is not uncommonly globular, and the dilated ends may then form large cavities. It may, however, be simply fusiform. The mucous membrane of the tubes is sometimes smooth; more commonly it is intensely congested, thickened, and villous: in some cases it is ulcerated, but this is rare, unless the dilatations are of large size, or except in the presence of tubercle or of sloughing action in the surrounding tissue. Their contents are either the usual muco-purulent secretion, or they may be highly offensive even without the presence of discoverable gangrene in the rest of the lung.

Secondary inflammation in the indurated parts is not uncommon; probably in some instances it extends from the bronchi. It leads to the formation of excavations, and is prone, in some instances, to take on a gangrenous action. Traube, indeed, regards this process as one of the most common causes of gangrene of the lung.[2] In the cases which I have analyzed, I find gangrene mentioned twice on the same side as the induration, once on the side opposite.[3] Biermer found gangrene in five out of fifty-four cases,[4] and Barth in three out of forty-three cases of bronchiectasis.[5]

[1] Charcot considers that the dilatation of the bronchi in "Cirrhosis" distinguishes it from Chronic Pneumonia, where he believes it to be absent; but indubitable evidence is afforded that it attends induration of the lung secondary to Pneumonia. See cases by Andral, Weber, and Biermer, before quoted.

[2] The same difference exists with respect to the inflammatory softenings of the aorta, which in some cases are the origin of aneurismal dilatations, while in others they indurate without having yielded to the pressure of the blood current.

[1] See a case by Dr. Wilks, Path. Soc. Trans. viii. 39*; also Sir D. Corrigan's cases. This condition is common in the extreme degrees of the affection. The resemblance noticed by Sir D. Corrigan to the bronchi of the tortoise aptly expresses this appearance in many cases.

[2] Deutsche Klinik, 1853–1859. From Prof. Traube's manner of speaking of chronic Pneumonia, he would appear to regard the disease as more common than many other observers do.

[3] Case by Dr. Walshe, Med. Times and Gaz. 1856, i. 156.*

[4] In the thirty-five cases reported by Biermer, gangrene is mentioned in three. In two of these there was induration of the lung. I do not regard bronchiectasis as synonymous with chronic induration, but introduce these numbers for the sake of comparison. Gangrenous Pneumonia may take place in this condition as an acute affection.

[5] In Cruveilhier's Path. Anat. liv. xxxii. is an illustration of this process in a case of chronic tubercular Pneumonia, when a large portion of tissue was separated and lying in a cavity. Cruveilhier does not, however, regard this as a case of gangrene. Dittrich (Lungen-Brand im Folge der Bronchien-Erweiterung) regarded these inflammatory effects as septic, and as arising from retained secretions, and when occurring in the opposite lung, as resulting either from the gravitation of the fluids into the bronchi of the

The Pneumonia in other cases, of which four are reported, has led to ulceration of the tissue, and the formation of cavities. The total number of cases in which Pneumonia is reported on the same side as the induration is four. In seven others it occurred on the opposite side. In one of these it was gangrenous, and in two others it had led to the formation of abscess.

The pleura is almost invariably thickened, and adhesions to the costal wall are also nearly constant when the disease has made any extensive progress, or has reached the surface. The thickening is sometimes extreme, and occasionally it extends through the interlobular septa into the tissue of the lung.

There is one remarkable feature about this condition to which allusion has been already made, and that is the preponderant number of instances in which one side only has been indurated, amounting to thirty-one out of thirty-nine cases. In five there was Chronic Pneumonia of the opposite side. The whole of the right lung was effected in ten cases, the whole of the left in fourteen, the base alone in eight, and one apex alone in three cases. A double affection of the apex existed in three, but in two of these there is evidence that the affection was tubercular.

Chomel's data are, that out of eight cases, in five the base was affected, in one the whole of one lung, in one the apex, and in one the middle two-thirds of the posterior part of the lung. He states also that in these the bronchi were generally dilated. Durand-Fardel[1] says that in his observations the upper lobe was affected five times, the lower lobe three times, and the middle lobe twice.

The non-affected parts of the lung sometimes present emphysematous changes. This change, usually of the hypertrophous type, is often exceedingly well marked in the sound lung, when only one is extensively affected by retraction and induration.

The bronchial lymphatic glands have sometimes been found to be much enlarged. In other instances they have been simply indurated. When tubercle has existed in the lungs, cheesy spots have in some cases been found in the glands.

PATHOLOGY.—There appears to be but little to add in explanation of cases where the ordinary appearances of pneumonic consolidation in the forms of red, gray, or yellow induration can be traced in direct continuity from a recent but acute attack of primary Pneumonia. Some points, however, require to be noticed with respect to the state of fibrous induration and its relation to other diseases.

Addison denied that this state ought to be called a *chronic Pneumonia*, and so far as Pneumonia is a process this criticism is probably correct as applied to the final condition of complete induration, for, as I have before stated, a cicatrized tissue can hardly be termed an inflammatory disease. The question is, however, a different one when we consider the process by which such indurations are produced; and I believe that the evidence which I have analyzed will suffice to show that they are very frequently the result of a pneumonia which has passed into a chronic stage.

It remains to be asked whether these indurations result from a process which, as Sir D. Corrigan supposed, has any analogy to cirrhosis of the liver, and from such a condition I believe that sufficient points of difference may be found, to cause serious hesitation in placing the two diseases in the same nosological category.

In the first place, there is this marked diversity between these indurations of the lung and cirrhosis of the liver, that in the lung the fibrous induration of the walls of the pulmonary alveoli is almost invariably, if not constantly, associated with the accumulation of the products of inflammation in the interior of the air-sacs. In a very large number of cases this is demonstrably the result of acute inflammation, and in many more it proceeds, though in a more chronic form, as an accompaniment of the inflammatory process attendant on the presence of tubercle, or determined by the tubercular diathesis. Further, the change in the liver takes place in a great measure through an increase of the fibrous tissue between the acini; while in the lung, though some thickening is found in the interlobular septa, the most important pathological alterations are those which occur in the walls of the pulmonary alveoli, which certainly have not yet been shown to be the anatomical analogues of the interstitial tissue of a glandular organ, but rather to correspond to the walls of the terminal extremities of the ducts of a gland. Or, to state the difference more briefly, in cirrhosis of the liver the change is external to the lobules and perilobular, while in induration of the lung the fibrous thickening is intralobular. In the liver it is still a question whether the condition known as cirrhosis can be called an inflammation, but in it, at least, there is scarcely any evidence that the cells of the acini of this gland have undergone any changes analogous to those seen in the interior of the pulmonary alveoli.

previously sound side, or from constitutional septicæmia. See also Briquet, Mém. sur un Mode de Gangrene du Poummon dependant de la Mortification des Extrémités dilatées des Bronches (Arch. Gén. de Méd. 1841).

[1] Mal. des Vieillards, 601.‡ Durand-Fardel's cases do not all refer to instances of induration.

The granular contracted condition of the kidney, which may be regarded as the most marked analogue of the cirrhotic liver, offers in another respect a striking contrast to these indurations of the lung. In the kidney—a double organ—the affection is almost invariably bilateral, and it is a very rare event to find a single kidney alone affected.[1] In the lung, the double affection is the exception, and generally explicable by a tubercular origin, and the single affection is the almost invariable rule when tubercle is not present.

On these grounds, therefore, I am strongly disposed to doubt whether, in the vast majority of cases, these thickenings originate in the alveolar walls as a primary affection, but rather to believe that they are an almost constant sequence of an alveolar Pneumonia which has passed into the chronic stage.

That thickenings of the interlobular septa may at times extend inwards into the lung as a consequence of chronic pleurisy is an undoubted fact; but more proof is at present required than has, I think, been afforded, that these can implicate the walls of the pulmonary alveoli to such an extent as to produce a general induration of the lung with obliteration of the air-vesicles, independently of a superadded pneumonic process, or of the co-existence of tuberculosis. I am only acquainted with two recorded cases which would appear to bear out such an opinion. One is in a note of a post-mortem by Dr. Wilks, reported by Dr. Sutton, where it is stated that "sections of the lungs showed that they were uniformly invaded by a tough fibre tissue, which had destroyed the natural structure and rendered them partially airless and very hard. There were no circumscribed masses of hard tissue, as is sometimes seen, but the pulmonary texture appeared invaded in all parts; thus the natural aspect was lost, being striated or interwoven with fibrous filaments."[2] Parts of the lung were emphysematous; the other organs were healthy. The other case is reported by Drs. Barlow and Sutton,[3] where one lung only was affected. Islets of normal pulmonary tissue appeared among the indurated portions, and thickenings could be seen around the bronchi.

[1] Curiously, a unilateral affection of one kidney has been noticed by Dr. Hilton Fagge, in a case of induration of the lung (Path. Soc. Trans. xx.*). The kidney was partially atrophied in its cortical substance. A calculus, however, existed in one of the calyces.

[2] Med.-Chir. Trans. xlvii. 309.‡

[3] Path. Soc. Trans. xvi. p. 39.* The liver and spleen were enlarged. The heart was enlarged, and tricuspid regurgitation had existed during life. The other organs presented nothing special.

It would require, however, a larger body of proof than these two cases appear to me to afford, in order to establish the existence of an independent pulmonary disease, whose essential characters consist in the thickening of the aveolar wall, as a *primary* affection occurring independently of inflammatory processes or of tubercular or syphilitic changes, and it is necessary that this proof should be fully established before such a class can be admitted into our nosological categories. I must confess that, though during many years I have paid much attention to this subject, I have never seen any pathological specimens supporting such a view, and nearly all the cases of pulmonary induration which have fallen under my own observation have been connected with previous Chronic Pneumonia associated with the presence of tubercles.

For this reason I think that the term "fibroid degeneration," when applied to this state, fails to express its true nature. The new tissue is a *growth* produced under conditions of irritation, and though pre-existing tissues may disappear in its progress, and so far it may be appropriately termed, as by my friend and colleague Dr. Bastian,[1] an instance of "fibroid substitution," it appears to me most important that the inflammatory conditions of its origin should be borne in mind.[2]

The associated pathology of chronic induration of the lung presents some fea-

[1] Cirrhosis of the Lung (Trans. Path. Soc. xx.).

[2] The term "Fibroid Phthisis," proposed by my friend Dr. A. Clark, has been very largely debated of late. If it is used to include all diseases tending to produce induration of the lung, it must necessarily comprehend many and widely different pathological processes which conduce to the same result. It is undoubtedly true that the symptoms of "phthisis" may arise from some non-tubercular diseases, and so far the exclusive limitation of the word to tubercular affections may be in a certain sense illogical; but as in the lung, at least, these form an enormous proportion of the whole, we shall have no option but to retain the term in the present sense, or to fall back upon the heterogeneous classification of Sauvages and Morton; and the former plan appears likely to be productive of the least amount of confusion in our nomenclature. It is important, doubtless, to recognize the origin of the induration of the lung, and to distinguish the purely pneumonic forms and those which are the result of bronchial dilatation or pleuritic thickenings, from those complicated by tubercle. In the same manner, while recognizing the "phthisical" tendency of ulcerative Pneumonia, or of some cases of chronic bronchitis, it would appear more desirable to classify these diseases in their pathological relations rather than in their occasional clinical aspects.

tures of interest. The heart is very commonly displaced when retraction of the lung is considerable. It also tends to hypertrophy, but not constantly, as I only find this condition described in eight cases. In four the heart is described as having been healthy. In thirteen its state is not mentioned. In two cases there was contraction of the mitral orifice, and in one tricuspid regurgitation, attended by a characteristic murmur.[1] In one it is described as fatty. In some cases thrombi were found in the pulmonary artery, which under these circumstances has been contracted:[2] Dr. Walshe, however, found it dilated. It may be a subject for further inquiry whether the coagulation of the blood in the branches of this vessel may not in some cases be a cause of protracted resolution of acute Pneumonia, or even of the secondary changes which have now been described. The fact that their mere obstruction may, as shown by Virchow,[3] give rise to inflammatory changes in the pulmonary parenchyma, which are usually persistent, would at least be an argument in favor of this hypothesis. Thickenings have been found in the coats of the pulmonary artery, both by Dr. Schmidt and by Dr. A. Clark. The liver is reported as healthy in eleven cases; granular and cirrhotic in six; enlaged and congested in two; fatty in one; in nineteen cases there is no mention of its condition. The kidneys were healthy in eight cases; granular in twelve; congested in one; their state is not mentioned in eighteen. The spleen is not mentioned with sufficient frequency to make any analysis useful.

The intestines are commonly reported as healthy: tubercle existed in them in some of the tubercular cases: diarrhœa without tubercle is reported in a few others. Chronic catarrh and congestion of the stomach are reported in a few cases; but the data of a large proportion are imperfect as regards the condition of the gastro-intestinal canal.

When ulceration or gangrenous action has ensued in the indurated parts, metastatic abscesses may be found in other organs. Three instances of this nature are reported where the brain was affected,[1] and another where abscesses of the same kind were found in the liver, spleen, and kidneys.[2]

It does not appear to me, on looking at the general results of this analysis, that the state of the other viscera affords any special ground for the assumption of a "fibroid diathesis" which has been recently maintained to exist as a primary cause of the pulmonary induration. The alterations of the liver and kidneys do not appear to be more common in chronic pulmonary induration than they are in many other chronic diseases, and particularly in those affecting the main conduits of the circulation, whether directly through the heart, or indirectly through the lungs. Both cardiac and pulmonary diseases, which give rise to systemic venous congèstion, are liable to cause induration both of the liver and of the kidneys, associated with an increased growth of their interstitial tissue; and these changes appear to me to be equally common in cases of simple chronic bronchitis and of chronic tubercular phthisis, as in the special affection now under consideration.

There are some other conditions which appear most properly to take their place under the category of Chronic Pneumonia, but which are also of rare occurrence. The chief of these are Chronic Ulcerative Pneumonia and Syphilitic Disease of the Lungs.

Chronic Ulcerative Pneumonia.—The recorded cases of this state occurring independently of tubercular disease are comparatively few. Broussais,[3] indeed, speaks of having met with several, but none are recorded by him except a case of ulceration secondary to the lodgment of a bullet in the lung. Dr. Stokes also speaks of being acquainted with cases of chronic pulmonary abscess arising from Pneumonia, and gives one case where cicatrization had ensued.[4] Bayle,[5] under

[1] Drs. Barlow and Sutton's case, before quoted.*

[2] Dr. Dickinson, Path. Soc. Trans. xvi.‡ Schmidt, Zwei Fälle von Chronischen Pneumonie. Schmidt's Jahresb. 1866.*

[3] Gesammelte Abhandlungen, 368. One case of Virchow's (loc. cit. p. 274), where old thrombi were found in the pulmonary artery, associated with indurated Chronic Pneumonia, would appear to give a further support to this view. Lebert's and Wyss's experiments (Virchow's Archiv, xl.) on the introduction of solid particles into the circulation have shown that this may give rise to thickening around the obstructed branches of the pulmonary artery, and that such thickenings may extend into the tissue of the lung.

[1] Biermer (loc. cit. p. 244); Lancereaux (Gaz. Méd., Par. 1863); Herard and Cornil (loc. cit.) A very similar case is also reported by Virchow (Archiv für Path. Anat. v. 276).

[2] Lancereaux, loc. cit.

[3] Examen, iv. 156, 336; Hist. des Phlegmasies, ii. 6, note. Broussais here says that during a long period he never met with an instance of this disease uncomplicated by tubercles except when caused by a foreign body in the lungs.

[4] Loc. cit. 316.

[5] Phthisie Pulmonaire, obs. 25 and 26. All the other cases reported by Bayle are more or less complicated by tubercles, but in obs. 28 the only evidence of this consisted in laryngeal ulcerations.

the title of "Phthisie Ulcéreuse," gives three cases of this nature. The first, of about two months' duration, showed one lung only affected with several ulcerated cavities, the contents of which appear to have been gangrenous. In the second, which was of three years' standing, and where a portion of bone entering the larynx was supposed to be the exciting cause, both lungs were indurated and contained numerous cavities. Bayle says that he has seen several other cases in which, commonly, there was only one ulcerated cavity. The size of these cavities was sometimes very considerable. Two are reported by him where a large cavity existed in one lung without disease of the other. One of these (Obs. 27) appears to have been a case of secondary ulceration, such as I have before described as occurring in a lung which has already undergone fibrous induration, and the same condition is present in some cases reported by other authors, so that it is difficult to come to a conclusion whether the induration or the cavity formed the primary lesion.[1] A case is recorded by Dr. Risdon Bennett,[2] where the history of the symptoms, which dated from an attack of scarlatina eighteen months previously, would appear to support the latter view, since a large cavity existed at the root of one lung surrounded by a gray infiltration.

The twenty-ninth case recorded by Biermer[3] bears a very close analogy with that last quoted, but here the disease in the lung appeared as secondary to typhus (typhoid ?), and was only of a month's standing. Numerous spots of Broncho-pneumonia passing into abscesses or forming cavities were found in both lungs. Dilatation of the bronchi was also present, and it may be questioned whether this was not of recent origin, since Buhl has shown that this condition tends to occur under identical circumstances, after continued fever associated with acute destructive Broncho-pneumonia.[4]

There are two fallacies to be guarded against in estimating the pathological significance of ulcerative processes in the lungs, which are (1) their origin in tubercle, and (2) their origin in pyæmic processes.

The latter need only to be mentioned as a frequent cause of pulmonary abscess, the origin of which may at times be difficult to discover. It is not unimportant also to remember that hemorrhagic infarcta may be the cause of indurated spots of cicatricial character, which, after long periods, may show but few traces of their origin.

Ulcerations may also take place from nodules of tubercle situated in the midst of gray or gelatinous hepatization, either recent or of a more chronic and indurated type, and the tubercle, having perished by softening, may leave only a cavity surrounded by gray infiltration, or by more or less induration.[1]

Symptoms and Physical Signs.—A considerable variety has been noticed in these, depending on the stage of the inflammatory action, but still more on the co-existence of bronchial dilatation, or of secondary ulceration or gangrene of the pulmonary tissue, and also on the presence or absence of secondary Pneumonia in the opposite lung.

(*a*) *In the cases where the state of consolidation has been traced in continuous sequence from an attack of acute primary Pneumonia*, the symptoms present have been chiefly those indicating a prolongation of the pyrexial state, together with a persistence of the physical signs of consolidation of the lung.

The fever does not, however, maintain the acuteness or the typical course observed in the primary disease. In some instances it is scarcely apparent, though the patient remains weak and continues to lose flesh.

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[1] See a case by Dr. Green (Path. Soc. Trans. xx.*); also the eleventh case by Barth (loc. cit.‡).

[2] Path. Soc. Trans. xii.‡ The sudden expectoration of a large amount of puriform matter in this case led to the suspicion during life of the evacuation of a loculated empyema through the lung, but no distinct evidence of this was afforded by the post-mortem examination. The symptoms and the subsequent expectoration would be quite explicable by an abscess communicating with the bronchi. There was some evidence of a tubercular diathesis.

[3] Loc. cit. p. 274.

[4] Virchow's Archiv, xi. 275, "Ueber Acute Lungen Atrophie." The name does not appear well chosen, since the cases alluded to were those of disseminated gangrenous Pneumonia, following typhoid fever and associated with collapse. Buhl considers that such conditions lead to subsequent shrinking and induration of the pulmonary tissue. The distinction which Buhl establishes for this form of Pneumonia appears to be that it is associated with collapse, and that it passes into acute desquamation and fatty degeneration of the epithelium of the air-vesicles; a process which he regards as being allied to acute atrophy of the liver.

[1] A case reported by Charcot as Chronic Ulcerative Pneumonia (loc. cit. Appendix, p. 66) appears to me to be of this character. Tubercle existed in the opposite lung. A case recorded by Louis (Case iii. Phthisis, Syd. Soc. Ed., trans. by Dr. Walshe, p. 19) was considered by him to belong to this class, inasmuch as tubercle was found in a lymphatic gland in the neck. There were, however, no tubercles in other parts of the body.

In other instances, however, it assumes more of the character of hectic, with irregular exacerbations and remissions, and usually a marked febrile movement takes place towards night. Exact thermometric observations on this subject are wanting, owing to the rarity of the disease in this form. I have already described the characters of the pyrexia in the only case of the kind which has come under my own cognizance. Night sweats sometimes, but not constantly, follow the evening exacerbations; and emaciation may be very rapid.

There is usually dyspnœa, but this is not always present in a subjective form. The rapidity of respiration also remains greater than natural; but as the pulse is usually accelerated, the degree of perversion of their ratio to one another, witnessed in the acute stage, is not commonly maintained.

Cough may in some cases be slight, in others it is persistent and troublesome, and may cause a return of the pain in the side. The sputa may in some cases retain a rusty tinge—more commonly they are mucoid or puriform, and with the latter character they may sometimes be expectorated in considerable quantities. Hæmoptysis has not been observed at this period of the disease, though it is common when dilatation of the bronchi and ulcerations have occurred.

The physical examination of the chest reveals at this period phenomena differing in little from those observed in the acute stage.

Retraction of the side to any notable degree does not take place until further induration and contraction of the pulmonary tissues have occurred; but the tendency is shown even at earlier stages by the case already quoted, of recovery after a protracted convalescence.

Respiratory movements are diminished on the affected side.

Percussion gives a toneless want of resonance which increases in intensity with the progress of the case. Bronchial or tubular breathing, bronchophony or pectoriloquy, with increased vocal fremitus, are the typical phenomena accompanying this state; but in some instances these have been noticed either to be entirely absent[1] or to have only been intermittingly present, alternating at times with an entire absence of breath-sound.[2]

Râles are generally heard during this period. They are commonly subcrepitant, and the fine crepitation of the acute stage does not appear to persist in the chronic form; large bubbling râles are more common, and they may be sufficiently metallic as to stimulate the characters of an abscess or an excavation even when none exists. The respiration in the opposite lung is commonly exaggerated.

If the progress of the case is unfavorable, the digestive system suffers, congestion and catarrh of the stomach supervene, and vomiting is occasionally observed. Thirst is a common symptom. Diarrhœa may also be present, without tubercle or ulceration of the intestines. Anasarca and ascites occasionally occur in the latter stages,[1] without any appreciable cause, other than that afforded by the disturbed circulation through the lung.

(*b*) *When the condition has passed into the more advanced stage of induration*, the symptoms present depend, in great measure, on the coexistent conditions. In some cases the cicatricial tissue formed is perfectly quiescent, and life may be long protracted, without much manifest impairment of the general health, and with only a minor degree of dyspnœa on exertion, although the physical signs of pulmonary induration persist. The presence, however, of dilatation of the bronchi, or the existence of ulcerations of these extending into the pulmonary tissue, or the occurrence of secondary Pneumonia, imparts to the disorder a gradually progressive character, which may strongly simulate the features of tubercular phthisis. These correspond closely to the description of the disease furnished by Avenbrugger and Corvisart. Avenbrugger pointed out that want of respiratory movements and of resonance on percussion were the leading physical signs, while the symptoms present chiefly consisted in dyspnœa on exertion and distension of the jugular and external veins; cough being unfrequent, expectoration scanty, and the decubitus of the patient remaining unaffected. Corvisart, in his Commentary, adds to these symptoms a progressive emaciation, and also a febrile diathesis, occasional partial perspirations, loss of appetite and of sleep, paroxysms of dyspnœa, and, in rare instances, œdema, which occasionally is limited to the limbs of the affected side.

When the complication of *bronchiectasis* is absent, the contraction of the indurated pulmonary tissue produces a gradual retraction of the chest-wall on the affected

[1] Requin, quoted by Grisolle, p. 340. Chomel, loc. cit. 277.

[2] Charcot, loc. cit. p. 39. Charcot only mentions the disappearance of the breath-sound, and not of the other phenomena. Neither he nor Requin have described the state of vocal fremitus. Charcot regrets that the relation of these phenomena to the expectoration was not noticed. It may be remembered that the same condition is sometimes observed in the acute stage. It has also been noticed by Bamberger over bronchial dilatations: Oest. Zeitsch. 1859 (Charcot).

[1] Durand-Fardel, loc. cit. 608.

side, which is general when the whole of the lung has been affected, or partial in the upper or lower parts of the chest, according to the site of the induration. If the affection is extensive, displacement of the heart occurs either upwards when the consolidation is seated at the apex of the lung, or if the affection be general, or implicates a large part of the base, the heart is drawn towards the affected side. The contraction of the side has been stated by Dr. Stokes to be as great as that following pleurisy, with the same approximation of the ribs, and procidentia of the shoulder. Dr. Walshe, however, denies that this form of retraction is produced by simple "cirrhosis."

The respiration in some cases, when there is no dilatation[1] of the tubes, has been observed to be bronchial; but there are very few, if any, authentic records of the physical signs in this state. Weak or suppressed breathing must be admitted as being *à priori* possible. We have, I believe, no data respecting the condition of the vocal fremitus and resonance in this condition.

(c) *When dilatation of the bronchi coexists with chronic pulmonary induration*, many variations occur both in the symptoms and in the physical signs. As a whole, as before stated, they closely simulate those of tubercular phthisis; but the progress of the disease is usually slow, and the deterioration of health and strength proceeds rather through a series of exacerbations than by any marked continuously progressive disease. Dyspnœa is almost constant, though not an absolutely invariable symptom. The decumbency (when mentioned) is commonly on the affected side. The pulse perspiration ratio does not appear to be necessarily or notably perverted.[2] Cough is usually persistent, and is liable at times to marked exacerbations. It may be dry, as originally noticed by Avenbrugger and Chomel, but more commonly it is attended by expectoration.

The sputa, when present, are variable in their characters: sometimes they are simply mucoid; more commonly they are puriform. Under the influence of intercurrent Pneumonia they may become at times rusty in tint. When ulceration is proceeding they acquire a brick-dust appearance, and under these circumstances they are often profuse. They often present a dirty greenish-gray appearance, which may approach a bottle-green or inky tint,[1] and they are either confluent, or consist of floating masses of irregular outline, marked by black specks and spots of the size of a millet or hemp seed, and they may contain a large amount of pigment and fragments of the elastic tissue of the lung.[2] The color of the sputa described by Traube has also been noticed by other observers. That these characters are common to dilatation of the bronchi is apparent from Barth's description[3] of their appearance under these circumstances. In one of his cases, where there was a "black softening" of the pulmonary tissue, they were of a chocolate tinge.

Fetidity of the sputa and also of the breath is a very frequent accompaniment of this condition. I find it mentioned in eleven out of the thirty-nine cases which I have analyzed. In four of these it accompanied gangrene of the lungs, but in five others there was no evidence of this state. In two cases, however, the data are imperfect. This offensive character of the sputa may indeed coexist with simple bronchitis or with bronchial dilatation without pulmonary induration; but the latter is the most common condition in which it occurs, particularly when ulceration and the formation of cavities[4] have taken place.

Hæmoptysis is comparatively a com-

[1] *E. g.*, in Dr. Andrew Clark's case. Respiration was bronchial under the clavicle of the affected side, where there was only a "thickened sub-pleural nodule," and where the lung tissue otherwise appeared healthy. There were, however, cavities in the central parts of the lung.

[2] Dr. Walshe's case.

[1] Traube (Deutsche Klinik, 1859, 477) considers these characters, and particularly the dark specks, as sufficient to distinguish the sputa of Chronic Pneumonia from those of tubercular phthisis, which he says are masses of yellow or whitish color, float in water, and keep their round shape, and do not present these black specks. The sputa of phthisis are, however, more varied in their appearance than those here stated, and masses of pigment are not, I believe, uncommon in them.

[2] Dr. Walshe.

[3] Loc. cit. 524.

[4] See Dr. Laycock on "Fetid Bronchitis," Case iv. p. 27. Fetidity of the sputa and of the breath, in connection with chronic pulmonary induration, was recognized as early as by Willis. Dr. Laycock, (p. 9) quotes a case by this author, of a dignitary of the Church who was long troubled with this symptom, and in whom some years later the whole of one lung had undergone the change in question. Dr. Laycock distinguishes this odor as fecal, in contrast to the special odor of pulmonary gangrene. In a chemical investigation undertaken of one case by Dr. Gamgee, the reaction of the sputa was alkaline. I have found the reaction alike in several cases of fetid sputa; in one also, now under my care, presenting the physical signs of the state at present under consideration. Dr. Walshe (loc. cit.) notes that the sputa are particularly fetid without being gangrenous, though in his case there was gangrene of the opposite lung, but probably of more recent date.

mon symptom. I find it mentioned in sixteen out of thirty-nine cases; in seventeen the data are imperfect; in six others its absence may be reasonably inferred. In six cases it was the first symptom that attracted attention, though cough had existed in some antecedently; in two, however, of these there is evidence of tuberculosis, and tubercles were also present in three other cases in which it appeared in the course of the disease. In nine others there were either ulcerations or cavities present, and in two only (in both of which it was slight) are neither of the above conditions recorded. In some cases where it appeared early, it is possible that the ulceration of previously dilated bronchi may have been the cause of its appearance, and that the Broncho-pneumonia thus excited may have led to the subsequent induration of the lung, since in most of these many years intervened between its appearance and the final fatal issue.

The amount of blood expectorated varies considerably. In the majority of cases it has been moderate, but it is occasionally repeated and it may prove the cause of death.[1] In a case by W. Schmidt,[2] where there was no evidence of tubercle, the patient had had seventy attacks of pulmonary hemorrhage in the course of twenty-three years.

The physical signs depend on the existence of dilated bronchi and of cavities in an indurated and retracted lung. The retraction of the side and the displacement of the heart reaches its extreme degree in this condition; and when the right lung is affected, the right ventricle of the heart may be found beyond the nipple[3] on that side. There is an absolute deficiency of expansion, though some elevation movements may persist over the affected lung. Percussion gives a high-pitched wooden resonance, which may even be amphoric or tubular, when large dilatations occur near the surface, and particularly in the infra-clavicular regions. The respiration presents in varying degrees the characters of bronchial or blowing. Bronchophony and pectoriloquy, the latter also occurring in the whispering variety, are most commonly met with over the affected parts. The vocal fremitus is usually exaggerated, but its absence has been noticed when both bronchial breathing and bronchophony have been present.[1] Râles of variable size are usually heard over the dilated tubes. They are commonly large and bubbling, and are not unfrequently metallic or cavernous in character. When one lung only is implicated, or unless recent secondary Pneumonia has supervened on the opposite side, the unaffected side is generally hyper-resonant, and the increased clearness on percussion may extend across the middle line, so that under the clavicle on the affected side, as was pointed out by Sir H. Marsh,[2] the dulness is greater towards the acromial angle than towards the sternal articulation of the clavicle. The respiration in the sound side is exaggerated—puerile; râles are only heard here when bronchitis or Pneumonia is present as a complication. Lividity of the face, amounting to a minor degree of cyanosis, is observed, and distension of the jugular veins appears to be comparatively not unfrequent. Peculiar white spots on the face have been noticed by Drs. Barlow and Sutton, and by Dr. Andrew Clark.[3] Gangrene of the extremities has been noticed as a complication in elderly people.[4] Clubbing of the fingers was noticed in a case by Ziemssen.

Pyrexia, though commonly existing only to a slight degree, is more or less present during the progress of these cases. There are periods when fever is not present, and its occurrence appears to be due either to intercurrent Pneumonia or to ulcerations and inflammatory action in the indurated tissue surrounding the dilated bronchi. In some cases of long continuance it is noticed as having occurred at variable intervals, alternating with apyrexial periods of considerable duration during the course of many years.[5] A large proportion of the recorded cases show a certain intermitting degree of febrile action, which in some instances has been severe and of a hectic type towards the close of life. In other instances, even under these circumstances, the febrile action has been slight, or almost imperceptible. Exact thermometric observations appear, however, to be entirely wanting in all the recorded cases of this disease.

Dropsy is a very common symptom. It is mentioned in thirteen out of thirty-nine cases. Its absence is only distinctly recorded or presumably inferable in eight. It is seldom extensive, and most commonly affects only the lower extremities.

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[1] As in Dr. Sutton's second case,‡ and in one by Dr. Foot, Dub. Journ. 1866, xli.* The latter case was probably tubercular, and the former is not free from the same suspicion.

[2] Zwei Fälle von Chronischen Pneumonie; Erlangen, 1863. Schmidt's Jahrb. 1866, p. 132.*

[3] Dr. Walshe's case.

[1] As in a case by Dr. Green, Dub. Quart. Journ. 1846, p. 510.*

[2] Note by Sir D. Corrigan, Dub. Med. Gaz. 1857, p. 284.

[3] Dr. A. Clark considers these to be indications of fibroid degeneration of the skin.

[4] Durand-Fardel, loc. cit. 604.

[5] Schmidt's second case, loc. cit.

It is sometimes associated with albuminuria and casts of tubes, but in other cases the absence of these has been noted.[1] Ascites, as noticed by Durand-Fardel, is extremely rare.[2] Diarrhœa is not uncommon even without ulceration of the intestines. The latter cause, and probably of a tubercular character, existed however in some cases.[3] Vomiting is also occasionally observed. In some cases where it has been severe, albuminuria has been simultaneously present. In other instances it is excited by spasmodic cough, by difficulty of expectoration, or occasionally by the offensive character of the sputa. In a few instances no explanation of its occurrence has been recorded. Emaciation and loss of flesh proceed in some cases to an extreme degree; in others, though cough had lasted long, there does not appear to have been much wasting of the tissues, but there are very few cases which do not present this to some extent.

Diagnosis.—The diagnosis of Chronic Pneumonia rests on the recognition of the consolidation of the lung, with or without the presence of cavities.

As a general rule, the affection being unilateral, we have to deal only with causes which may affect one lung singly; and the diseases with which this state may be confounded are—pleurisy with effusion, or pleurisy with retraction, collapse of the lung, tubercle, and cancer.

The possibility of *Pleuritic Effusion* can only be admitted in the more recent cases when retraction of the side has not already occurred. Under ordinary circumstances in chronic induration, the absence of enlargement of the side, the distinctness of the intercostal spaces, and the presence of bronchial breathing, bronchophony, and increased vocal fremitus, are usually sufficient to exclude the idea of fluid in the pleura. In the rare cases where the respiratory sounds together with the vocal resonance have been inaudible, the mistake has actually occurred,[4] but attention to the state of the intercostal spaces would probably prevent this fallacy. A further guide in such cases is the position of the heart, particularly when the affection is on the left side. In effusion it is pushed from the dull side. In Chronic Pneumonia without retraction it is not displaced. Depression of the diaphragm would also aid in the recognition of pleurisy. In the majority of cases, however, these difficulties are unlikely to occur.

Pleurisy with retraction of the side may offer greater difficulties, particularly if the indurated lung does not contain dilated bronchi.

In Chronic Pneumonia, however, the retraction is more general than in pleurisy, and is not, according to the most recent evidence, attended with the same degree of twisting of the ribs on their axes, or with the procidentia of the shoulder and tilting outwards of the angle of the scapula, which attends the retraction following pleurisy.[1] Bronchial breathing and bronchophony are common to both affections; and in pleurisy, even signs of excavation may occasionally be simulated by marked pectoriloquy. But such cases, which are the exception in chronic pleurisy, are the rule in pulmonary indurations associated with dilatation of the bronchi; and therefore the existence of signs of cavities, particularly when general, and when associated with large and metallic râles heard over the affected lung, will be strong evidence in favor of the latter condition. The diagnosis will also be aided by the other features of the case. The characters of the expectoration are often peculiar in Chronic Pneumonia. In pleurisy, if bronchitis be present, they are simply bronchitic, and their source may be discovered by the râles in the opposite lung. Hæmoptysis can only take place in *simple* pleurisy under some conditions affecting the opposite lung. If these be undiscoverable, the presumption would be in favor of Chronic Pneumonia. Pyrexia and emaciation are rarely found in simple chronic pleuritic retraction; and diarrhœa is also uncommon except in the later stages of cases complicated with albuminuria. Regarded as a whole, although no single diagnostic sign exists for the discrimination of the two affections, the group of symptoms characterizing Chronic Pneumonia are sufficiently distinct to prevent, in the majority of cases, the possibility of this error in diagnosis.

Simple Collapse of the Lung, sufficiently extensive to simulate retraction with induration, can only occur in the adult as a consequence of obstruction of one of the main bronchial tubes. Except from the introduction of a foreign body, this can only occur from external pressure—as from an aneurism or a tumor originating in the bronchial glands—and would then be attended with other pressure symptoms,

[1] In Ziemssen's case (loc. cit.) albumen was absent from the urine when anasarca was first noticed, and only appeared later together with casts of tubes. The data respecting the urine are, as a general rule, imperfect in most of the recorded cases.

[2] Loc. cit. 612.

[3] As in one by Sir D. Corrigan, Dub. Hosp. Gaz. 1857.

[4] Grisolle, p. 342.

[1] Dr. Walshe, Med. Times and Gaz. 1856, i. 1858. The author is indebted to Dr. Walshe's masterly analyses for most of the data on the subject of diagnosis.

or by the physical signs indicative of the nature of the disease. Collapse of the lung is further attended with weakened or suppressed respiratory murmurs. Bronchophony, also, is rarely heard over such parts, and would be practically impossible when the collapse originated from obstruction of the bronchi. Signs of dilated bronchi are also entirely wanting except in the acute form of Broncho-pneumonia. Pyrexia, and other symptoms enumerated, are also absent in uncomplicated cases of collapse.

Cancer of the Lung produces retraction of the side, and may lead to signs of excavation. In it, however, the displacement of the heart is much less considerable. The dulness commonly extends across the middle line, while in retraction from Pneumonia the sound lung usually encroaches on the affected side. In cancer, also, pressure signs may be present: the excavations are more extensive, and hæmoptysis is commonly more profuse and repeated. The peculiar currant-jelly-like expectoration of cancer has not been met with in any of the recorded cases of pulmonary induration. Pain in the chest is also much more common in cancer than in Chronic Pneumonia; in the latter disease it appears scarcely ever to be a prominent feature. Local limitation to one side, and the absence of secondary cancerous affections, are less certain guides, but they are *pro tanto* in favor of simple induration. The existence of the cancerous cachexia has not been distinct in the cases which I have seen of primary cancer of the lung. Emaciation and pyrexia are common to both classes of disease. The duration of the case, where it has exceeded more than two years, is, according to Dr. Walshe, almost sufficient to exclude cancer; and this, together with the features first enumerated, are adequate for the diagnosis of the two diseases.

The diagnosis from *tubercle* presents considerable difficulties in some aspects of the question. It is, I think, by no means improbable that a tubercular Pneumonia may, under favorable circumstances, produce a local induration of a part or even of the whole of one lung without necessarily entailing a secondary affection of the opposite lung or of other organs. The indurations attending chronic tubercular phthisis are almost, if not absolutely, identical in their nature with those resulting from a non-tubercular inflammation; and the final result in both cases, of cicatricial tissue traversed by dilated bronchi, produces a condition in which the physical signs and symptoms of the two affections are precisely similar. Such cases are, however, exceptional. In tubercle the disease is commonly progressive, and it is only in very rare cases that the opposite lung is not implicated. The difficulty of diagnosis, however, refers rather to affections of the apex than to those of the base of the lung. A double apex affection, attended with the signs of cavities, is immensely in favor of the tubercular origin of the disease. When, as in some instances, the consolidation affects the base of one lung and the apex of the other, this presumption has also the greatest amount of probability in its favor. A unilateral induration of the whole or the greater part of one lung, when the opposite lung is hypertrophous and healthy, affords on the other hand strong evidence against the tuberculous nature of the affection. Neither the presence nor absence of hæmoptysis, of pyrexia, or diarrhœa, afford any material additional aid, since they may all occur in chronic induration, and may be absent in cases of chronic phthisis, at least during long periods. Fetidity of the sputa is rare in phthisis, very common in Chronic Pneumonia with dilated tubes; but it may exist in the former and may be absent in the latter. Under all the circumstances to which I have now alluded, the history of the patient may afford some aid. The history of a previous acute attack occurring on the affected side is largely in favor of the diagnosis of simple induration. The existence of an antecedent but long-continued bronchitis, coupled with occasional attacks of pyrexia, and gradually increasing dyspnœa, would lead to the same conclusion; and practically—though some cases have been recorded as errors in diagnosis—the data for the recognition of the disease are usually sufficiently clear to avoid most of the fallacies which may lead to an erroneous conclusion.

Prognosis.—This may be best studied by considering the duration of the recorded cases. Of these the duration in eight was unknown. In four, death appears to have taken place within twelve months from the first serious symptoms, though whether this represents the whole duration of the disease may be considered as doubtful. The shortest recorded period is three months, mentioned in two of Charcot's and in one of Sir D. Corrigan's cases, and both the former appear to have been simply cases of the acute disease running a protracted and fatal course and ending in ulcerative excavation of the lung. In the case before quoted from Andral, where also the pulmonary induration succeeded to an acute attack of Pneumonia, death took place within eighteen months; but in a similar case recorded by Ziemssen in a child, nine years of progressive illness elapsed between the first attack and the fatal termination. In the remainder analyzed,

the disease was of unknown duration in six cases. Dating from the first symptoms, death took place in four cases within 2 years, in two within 3 years, in four within 4 years, in four within 5 years, in two within 6 years, in two within 8 years, in three within 11 years, and in six cases life was protracted to 14, 20, 23, 27, 34, and 44 years. Excluding the cases of unknown duration—in many of which, however, pulmonary symptoms had existed during some years—and excluding also those where death took place in less than 12 months, we find that fourteen died within 5 years, and twelve, or nearly an equal number, lived for variable, and sometimes for considerable, periods beyond this date.

The general conclusion which may safely be drawn from these figures is, therefore, that under favorable circumstances life may be considerably protracted after distinct signs of consolidation have become apparent, and even when there has been an occasional recurrence of threatening symptoms.[1] The possibility, indeed, of a nearly perfect restoration to health, though a considerable extent, sometimes amounting to nearly the whole of one lung, is rendered impervious to air, may be seen in some of the cases before quoted, especially in children, where the affection has succeeded to an acute attack of Pneumonia or of Bronchopneumonia. In none of these cases, however, is there any evidence that the affected part ever regained its respiratory function; and it may be regarded as more than doubtful whether organized cicatricial tissue of the lung is ever removed by any process, so as to restore the natural condition. In some cases, where only a part of the lung has been thus indurated, its gradual contraction is compensated for by an hypertrophous emphysema of the remainder; and thus the area over which the physical signs of induration exist may gradually diminish, and may become replaced by those of healthy pulmonary tissue.[2] If the whole of one lung has been indurated, the process of respiration is necesarily confined to the other, and this increases the danger arising from any subsequent disease, by which the sound organ may be incapacitated from performing its functions.[1]

The other elements in the prognosis depend on the progress of the disease in the affected parts, and on the general constitutional state of the patient.

Evidences of chronic inflammatory action in the bronchi, as shown by catarrh, with pyrexia and profuse expectoration, have, in addition to the exhausting effects which these directly produce, the further unfavorable significance, that they threaten an extension of the inflammatory action to the surrounding pulmonary tissue, and are thus a source of danger from ulceration and the formation of cavities. The more quiescent these symptoms appear, the more favorable therefore is the ultimate prognosis.

The existence of ulceration as shown by hæmoptysis, or by the expectoration of grumous fragments of lung tissue, are unfavorable signs;[2] and though hæmoptysis may, as before stated, be in some cases frequently repeated during many years, yet, in the majority of instances, cases presenting this symptom to a marked degree have a more unfavorable course than those in which it is slighter in amount or altogether absent; and it must further be remembered, that such hæmoptysis may occasionally prove immediately fatal. Cases commencing with hæmoptysis are always open to the suspicion of being of tubercular origin, but the very existence of an indurating tendency in tuberculous disease imparts to such cases a comparatively favorable prognosis; and some of these, as recorded, appear to have quite as favorable a chance of prolongation of life as those cases where the primary induration appeared to be of simple inflammatory origin.

An offensive character of the sputa almost invariably gives a more serious character to the case; but life may in some instances be long protracted even after this has appeared. Fetidity of the expectoration may coexist with simple dilatation of the bronchi, but the character of the secretion in such cases imparts to them a septic tendency, and increases the liability to septic or gangrenous Pneumonia, either in the affected or in the opposite lung. In many cases, indeed, such fetidity is immediately associated with ulcerative processes, and the recognition of

[1] This is particularly seen in two cases recorded by Schmidt before quoted, where the patients with signs of pulmonary consolidation with cavities were under observation during periods of fifteen and twenty-three years, and where it is stated that the second of these outlived three physicians who attended him. A boy under the care of Dr. Mayne, whose case is not free from the suspicion of tubercle, lived six years of a laborious life involving great exposure, after distinct disease had been observed in one lung, and died only of acute bronchitis affecting the other. (Dubl. Hosp. Gaz. 1860, vii.*)

[2] As in Bartel's case, before quoted.

[1] As in Dr. Mayne's case (Dubl. Hosp. Gaz. 1860, vol. vii.), where death was caused by acute bronchitis affecting the previously sound lung.

[2] The discovery of elastic lung fibres in these fragments is the only positive evidence of pulmonary destruction; but when ulceration affects an old induration, the elastic fibres may be undiscoverable.

these may probably be aided in great measure by the coexistence of pyrexia with this state.

The presence of fever under such circumstances usually depends on the existence of secondary Pneumonia; and when it is undiscoverable on the sound side, it is a matter of great probability that it is extending in that already affected. Pyrexia, when of long continuance, has been indeed one of the final phenomena in nearly all the recorded cases, and its existence is always to be regarded with the gravest suspicion, both in its diagnostic significance and also through its exhausting effects on the nutrition and strength of the patient.

Emaciation is naturally an unfavorable sign, but in some cases death may ensue without any extreme degree of marasmus being attained.

Dropsy is always an unfavorable symptom. Very few cases attained to any considerable prolongation of life after it had appeared. Even when albuminuria is absent, it is indicative both of serious disturbance of the circulation and probably also of an hydæmic condition of the blood; and the coexistence of albuminuria and the presence of casts of tubes in the urine adds an additional gravity to the prognosis, by their significance as expressions of a general impairment of nutrition.

Diarrhœa and vomiting are also signs of the gravest import. They rapidly exhaust the patient, and in some cases lead directly to the fatal issue. Finally, when ulcerative action is proceeding, the possibility of secondary metastatic affections must also be recollected. In one case, by Andral, sudden death is reported to have occurred without any adequate explanation being afforded of the cause of such a termination.[1]

TREATMENT. — The treatment of Chronic Pneumonia may be considered under two categories. The first includes the cases where the disease has *recently* lapsed from the acute stage. The second comprehends those where thickening and fibrous growth have taken place in the walls of the air-vesicles.

(*a*) Cases of the first type are often serious in their character, and the continuous pyrexia and progressive emaciation tend to a fatal issue.

The great principle to be followed in such cases is steadily to maintain the strength of the patient, and to meet individual symptoms as they arise.

Sufficient evidence exists to show that under these conditions progressive improvement may be observed, and that the lung may be restored to its healthy state. I have the strongest doubts whether medicinal agents have any direct effect in accelerating the absorption of the exudation matter from the interior of the air-cells of the lung, and I believe that the administration of mercurials, or even of the preparations of iodine with this purpose, is likely to defeat the main object which should be pursued of maintaining and improving nutrition.

As long as pyrexia persists the patient should be kept in bed, in order to economize, as far as possible, the expenditure of strength and the waste of tissue. The diet should be liberal, but proportioned in quantity and quality to the digestive powers; and milk may, when it agrees, be freely taken with advantage. Alcoholic stimulants, in moderation, are required in most cases; the form selected is best determined by their effects on the individual case. It is important to watch their effects on the pyrexia, which should be made the object of careful and repeated thermometric observations. It has appeared to me that they are best given as far as possible during the periods of remission, and that they should be withheld or given in diminished quantities before and during the febrile exacerbations. When night perspirations are present, a moderate dose of stimulant will often have the effect of checking these, and also of obviating some of the exhaustion which is felt on the succeeding morning.

Cough may be allayed by small doses of opiates combined with three or four drops of the vin. ipecacuanhæ, and with from five to ten grains of the muriate of ammonia. Iron, quinine, bark, and the mineral acids may all be employed according to the special indications of anæmia, failure of appetite, sweating, or profuse expectoration. If the latter be present, ipecacuanha should be withheld, and ammonia with infusion of senega or serpentaria may be given. The existence of gastric catarrh often forms an unfavorable complication of these cases. Its presence appears to me to contraindicate most of the remedies last enumerated, and it is usually aggravated by the whole class of "tonics." If it co-exists with much cough and expectoration, these may be met with opiates and the muriate of ammonia, while the gastric disorder is best treated by bismuth, alkalies, and occasionally by small doses of hydrocyanic acid. Under this management a gradual improvement will often take place, and it has appeared to me that this may be aided by small blisters repeatedly applied over the affected side—a method which I think better than the use of extensive vesication, which may disturb sleep and exhaust the patient.

(*b*) In the treatment of chronic induration, it is, I believe, best to recognize the

[1] The suspicion here may arise of thrombosis of the pulmonary artery.

fact that fibrous tissue once formed is incapable of being removed by medicinal treatment. The management of such cases is therefore mainly hygienic, and should be directed to maintain the health and to prevent extension of the disease in the affected side or secondary affections of the opposite lung.

The dilatation of the bronchi which commonly attends this condition, renders the patient liable to catarrh; and since it is from repeated attacks of bronchial inflammation that the most dangerous effects of the disease are likely to arise, the avoidance of all causes of this nature is therefore of primary importance. Flannel worn next the skin, the avoidance of exposure, and the resources of a climate suited to the individual case, and which may be in part determined by experience and in part by the conditions of irritability or of more or less tendency to bronchial secretion, are among the most important elements to be considered among the prophylactic agencies.

Every fresh catarrh should be at once and promptly met, at least in its acute stage, by confinement to a regulated atmosphere, by counter-irritation to the chest, and by opiates, ipecacuanha, or muriate of ammonia.

The maintenance of the general health and of the nutrition are also of essential importance. A liberal diet, of which milk may form a large share, and a judicious use of alcoholic stimulants whenever pyrexia is absent, are almost always required. Cod-liver oil may also be taken with advantage during long periods when the digestion will tolerate its use. Iron and bark should be given, if the indications for their administration arise.

If any of the severer complications are present, they should be appropriately treated—hæmoptysis by gallic acid or the acetate of lead or ergot: diarrhœa by astringents; vomiting by small doses of opiates or by hydrocyanic acid; and gastric catarrh by bismuth and alkalies.

Profuse expectoration may be met by inhalations of oleum picis, creosote, or carbolic acid, either in the form of vapor or by means of a weak solution in an atomizer; and the muriate of ammonia may sometimes be used with advantage in the same manner. The inhalation of the vapor of the oil of turpentine has been tried in some cases where the sputa have been fetid, but unfortunately without much success. The vapor of iodine has appeared to me to give more relief in some cases when gangrene has imparted this character to the sputa. Port wine in full doses often has a most beneficial effect in cases characterized by profuse expectoration, and preparations of bark and the mineral acids have also a favorable influence.

Opiates not only check secretion, but diminish the violence of the cough, and relieve the bronchi from the continual tendency thus excited to produce further dilatation.

The treatment of albuminuria, dropsy, or ulceration of the pulmonary tissue, when this has occurred, is unfortunately almost beyond the reach of remedial agents: and though much may be effected before these complications have occurred, the later stages of this condition can only be met by such indications as may promote the comfort of the patient, rather than with any hopes of restoration.

APPENDIX TO ARTICLE ON CHRONIC PNEUMONIA.

The rarity of this disease and the difficulty attending some points of its pathology induce me to believe that references to the most important published cases illustrating it may possibly be useful to others. I shall continue to mark the cases which I have tabulated by an asterisk (*).

Bayle (Phthisie Pulmonaire), two cases of "phthisie avec mélanose."* Laennec (Forbes's translation, 2d Edition, p. 112), a case of dilatation of the bronchi with pulmonary induration.* Andral (Clinique Médicale, iii. obs. lxiv.), a case where induration of the lung succeeded an attack of acute pneumonia.* Dr. Stokes (Diseases of Chest, p. 150), a case of chronic pulmonary induration with dilatation of the bronchi.* Jaccoud Clinique Médicale, p. 82), sclerosis of the lung with dilatation of the bronchi—*tubercular?** Herard and Cornil (Phthisie Pulmonaire, 167), induration of lung with bronchial dilatation.* Ziemssen (Pleuritis und Pneumonie im Kindesalter), a case of nine years' standing in a child, probably resulting from an attack of acute pneumonia; dilatation of bronchi in both lungs; induration of one.* Barth (Rech. sur la Dilatation des Bronches; Mém. Soc. Méd. Obs. 1856), six cases;* *some probably tubercular.* Heschl (Prager Vierteljahresch. 1856, ii.), two cases: only pathological details.)* W. Schmidt (Zwei Fälle von chronischen Pneumonie, Diss. Erlangen, 1863).* Charcot (De la Pneumonie chronique), three cases,* two acute. Dr. Green (Path. Soc. Trans., vol. xx.), ulcerative pneumonia.* Dr. Peacock (Edinb. Journ. 1855).* Raimbert (Journ. Méd. et Pharm. de Bruxelles: analysis in Gazette Hebdom. 1856), one case incomplete;* others referred to are described as "carnification." Dr. Andrew Clark, a case of fibroid phthisis* (Trans. Clin. Soc. i.), *probably tubercular.*

The following are described as cases of "cirrhosis:"—

Sir D. Corrigan (Dublin Journal, 1838, and Dublin Hospital Gazette, 1857), three cases with post-mortem results.* Dr. Walshe (Med. Times and Gazette, 1856), the most fully-recorded case extant with commentary.* Dr. Mayne (Dub. Hosp. Gaz., 1857 and 1860),

two cases,* the last *doubtfully tubercular*. Dr. Green (Dublin Quarterly Journal, 1846).* Dr. Law (ib. 1848),* *probably tubercular*. Dr. Jennings (ib. 1866).* Dr. Foot (ib. 1866), *doubtfully tubercular*.* Dr. Wilks (Path. Soc. Trans., viii.), *probably tubercular, mentions "spots of strumous deposit" in the opposite lung*. Drs. Barlow and Sutton (Path. Soc. Trans. xvi.).* Dr. Fagge (ib. vol. xx.).* Dr. Barlow (Guy's Hosp. Rev. 1847, 2d Ser. v.).*

The following cases not included in the analysis as being either of doubtful nature or imperfect in general details, may also be referred to:—

Dr. Dickinson (Path. Soc. Trans. xiii.). Dr. Risdon Bennett (Path. Soc. Trans. xii.), a case of ulcerative pneumonia of doubtful origin Biermer (Zur Theorie und Anatomie der Bronchien-Erweiterung, Virch. Arch. xix.), many instances of induration of lung. Macdowell (Dublin Quarterly Journal, 1856), entitled as "Cirrhosis," but lung described as "carnified;" no dilatation of bronchi; thickened pleura and fluid in pleural cavity. Weber (Path. Anat. der Neugeborenen und Säuglinge, ii.), three cases referred to where induration of lung commenced with acute pneumonia, with post-mortem results, and two more of recovery. Bartels (Virch. Arch. xxi.), a similar case commencing with broncho-pneumonia. Steffen (Klinik der Kinderkrankheiten), four cases of interstitial pneumonia; three at least *tubercular*. Legendre (Rech. Anat. Path. Maladies de l'Enfance), two cases of induration of lung in children; secondary to catarrhal pneumonia. Dr. Bennett (Rep. City of London Hosp. for Dis. of Chest), two cases of induration with signs of dilated bronchi in children; recovery. Traube (Deutsche Klinik, 1859, § iv.), two cases, chronic ulcerative pneumonia; general remarks. Dr. Addison's Works (Syd. Soc. Ed.), three cases. Steiner and Neuretter, Padiätrische Mittheilungen (Prager Vierteljahresch. 1866, lxxxii.), two cases of induration after broncho-pneumonia. Macquet (Bull. Soc. Anat. xxii.). Gabalda (ib.). Charnal (ib. vol. xxx.), induration of lung in consequence of acute pneumonia; other data imperfect. Barth (ib. vol. xxix.), induration of base with dilated bronchi. Barset (Bull. Soc. Anat. xxx.), microscopic examination by Robin after maceration. Lancereaux, two cases of gangrenous pneumonia, surrounded by induration, and associated with secondary abscesses in other organs (Gaz. Méd. Paris, 1863). Powell (Trans. Clin. Soc. ii.), cases of phthisis with contracted lung; excellent description of appearances, all of *tubercular nature*. Sutton, fibroid degeneration of lungs (Med.-Chir. Trans. 1865, xlviii.). Bastian, Cirrhosis of Lungs (Path. Soc. Trans. xx.), tabulated series of thirty-four cases of induration. Durand-Fardel (Mal. des Vieillards), various forms of chronic pneumonia. Cotton on a prevailing form of Chronic Pneumonia (Med. Times and Gaz. 1855. i.); general description, pathological appearances of two cases. Hardy and Béhier (Path. Interne); general description, ref. to four cases. Grisolle (Traité de la Pneumonie), ref. to various cases. Lebert (Physiologie Pathologique), ref. to pathological appearances in two cases. Chomel (Dict de Méd. xvii.), general description. Avenbruegger (Inventum Novum, &c., 1761), "Scirrhus of Lung," with commentary by Corvisart (Eng. Trans. by Sir J. Forbes). Sir J. Forbes (Appendix to Trans. Laennec, Ed. 1824), two cases, one probably tubercular, the other doubtful, tissue of lung floated. Broussais (Hist. der Phlegm. vol. ii.), three cases. Hasse (Path Anat., Syd. Soc. Ed.). Hope (Morbid Anatomy). Rokitansky (Path. Anat.). Cruveilhier (Anat. Path., liv. xxxii.). In addition to these, the following works, to which I have not been able to obtain access, are referred to by Grisolle, Durand-Fardel, and Charcot, but the data of many appear from the statements of these authors to be unreliable or imperfect: Letenneur (Diss. Pneumonie chronique, Thèse de Paris, 1811). Bazière (Diss. sur l'Emploi du Séton dans la Pneumonie chronique, 1819). Rat, Thèses de Paris, 1845. Raymond, Sur la Pneumonie chronique simple, Diss. 1842. The author is indebted to Dr. Bastian's tables and to M. Charcot's thesis for several references to cases, some of which are included in the foregoing analysis.

(*Since this article was printed I have met with two other illustrations: Dr. E. Long Fox* (*Med. Times and Gaz.* 1870), *a case of chronic ulcerative pneumonia—thermometric observations; Immermann* (*Deutsch. Arch. Klin. Med.* V. p. 235 *et seq.*), *a case of chronic pulmonary induration* (*tubercular?*), *attended with stenosis of the pulmonary artery.*)

SYPHILITIC AFFECTIONS OF THE LUNG.[1]

By Wilson Fox, M.D., F.R.C.P.

The manner in which the tissue of the lung may be affected by the syphilitic poison, although it has been made the subject of much recent research, still requires a more accurate definition than has yet been attained.

The opinion that certain forms of phthisis may arise from changes in the pulmonary tissue due to the syphilitic poison, is no new one. Morgagni noticed the frequent connection of tubercle with this dyscrasia, and Portal and Morton described a syphilitic phthisis, but failed to show that any special pathological changes were connected with this condition. Dr. Graves and Dr. Stokes[2] have both entertained a similar opinion, based upon the success of the mercurial treatment of bronchitis in patients who had formerly been the subjects of venereal sores. Bayle, Laennec, and Louis failed to find any evidence of a special form of phthisis which could be distinguished as syphilitic, and it is only within recent periods that any changes have been identified in the lungs, which can probably be attributed to this cause.

The difficulty of the inquiry lies in establishing any certain criteria by which such alterations can be distinguished from the changes produced either by simple inflammatory, or by the tubercular processes. Each of these may affect syphilitic patients, and may run a course apparently unmodified either clinically or pathologically by the specific dyscrasia; and looking at the general history of syphilitic affections, it is at least probable that the lungs are less prone to suffer from secondary or tertiary affections of a syphilitic character than the mucous membranes of the upper air-passages, or than the skin, the eye, or the bones. What their comparative liability may be in respect to the liver, the spleen, the testicle, or the brain, is a point which must yet be determined by further research. In the lungs of syphilitic patients which I have examined, I have seen no appearances differing from those of ordinary pneumonia, of ordinary tubercle, or of tubercular or cheesy infiltrations; and one marked case of this kind has come under my observation, where there was the most distinct syphilitic ulceration of the larynx, but where the lungs only presented a gray infiltration, together with tubercles and indurations referable to a previous attack affecting the apices, the cure of which I had myself witnessed at an earlier date. Other instances of an analogous kind have come under my observation, where the most careful microscopic examination failed to reveal any peculiarities which I could ascribe to a syphilitic process.

The inquiry into the nature of changes attributable to syphilis is therefore for the present almost a purely pathological one, though the importance of the question in its clinical aspect can scarcely be overrated. A large amount of the evidence on this subject is derived from premature or stillborn children, the offspring of syphilitic parents; but some cases are recorded where syphilitic gummata have been found in the lungs of adults.

There are two sets of changes in the lungs, regarding the syphilitic nature of which there is a considerable unanimity of opinion. In another large class there is more doubt as to their true connection with this poison. The former are at least rare, and only isolated instances are recorded by observers having large opportunities for pathological research. The

[1] I have not met with any indubitable instances of these affections in my pathological studies on the disease of the lungs, and the information contained under this head has been drawn from the following authors, in addition to those alluded to subsequently: Virchow, Archiv, xv., Krankhaften Geschwülste, vol. ii.; historical data and complete references. E. Wagner, Arch. der Heilkunde, 1863, vol. iv. Foerster, Würzb. Med. Zeitsch. 1863, vol. iv.; Berkeley Hill, Syphilis and Local Contagious Disorders, many references. Von Baerensprung, Die Hereditäre Syphilis; many cases; microscopic figures of gummata in the lungs. Lancereaux, Traité Hist. et Pract. de la Syphilis; extensive bibliography, numerous cases. Lebert, Traité d'Anat. Path. Pl. xciii., figures of gummata in the lungs. Wilks, Guy's Hosp. Rep., 1863, and Path. Soc. Trans. ix., figures of gummata in the lungs; also A Lecture on Syphilis. Pihan Dufeillay, Des Dégénérescences Syphilitiques des Viscères, Union Méd., 1861, and in Bull. Soc. Anat. 1861; comments in a case of Cornil's; numerous references and critical observations.

[2] Graves, Clin. Med. ii. 27. Stokes, Dis. of Chest, 94–432.

latter class requires a most careful and critical examination before their specific nature can be admitted.

The most authentic changes in the lungs which can be ascribed to syphilis are gummata, or masses of low fibrous growth, evincing a great tendency to necrobiotic changes of the dry cheesy type, and which are very closely analogous to similar masses found in the liver and in other internal organs. They are found in the lungs of adults, and in newly-born syphilitic children. In the former, however, they are so extremely rare, that Lancereaux has only been able to collect ten cases by different authors. They are irregularly distributed through the lungs, having no special seat of predilection, but according to Wagner they are more common in the deeper than in the peripheric parts. They may be single or multiple, and their dimensions may vary from the size of a pea to that of a walnut, or even of a goose's egg.[1] They are generally rounded, rarely irregular in outline, and are sharply defined, but are not always encapsuled.[2] In their earlier stages they are gray or brownish red, completely homogeneous to the naked eye, and are firm and dryish:—later they become of a comparatively uniform yellowish tint, but still maintaining their dry firm character. In some instances, however, they soften and form actual, or, more commonly, potential cavities.[3] On microscopic examination they are found to consist of imperfectly formed fibres, which are often granular and are intermixed with abortive nuclei and a few fibre cells. Both the nuclei and the cells are commonly found in various stages of fatty degeneration. The lung tissue is entirely destroyed by this growth, by which the walls of the alveoli become progressively thickened, until the cavity of the vesicles is obliterated, while the epithelium lining them appears to participate but little in the change. In some cases the bronchi show an infiltration of the submucous cellular tissue with a fibro-nucleated growth, which may form small prominences on the surface. Similar masses are sometimes found in their deeper structures, but these as a general rule are unaltered. In these changes the preponderant and distinctive character consists in the growth of an indurating fibrous tissue, mingled with abortive nuclei, into distinct masses, and presenting a strong tendency to an early necrotic change.

Another form, termed by Wagner the "diffused," is the appearance described by Virchow[1] and Weber[2] as the "white hepatization of the lungs" of newly-born children: it has also been named "Epithelioma of the lungs," by Lorain and Robin:[3] and its syphilitic character has been shown by the last-named authors, who traced a relation between it and syphilitic pemphigus, and also by Hecker,[4] Howitz,[5] and Wagner,[6] and this has also been admitted by Virchow. Lungs in this state are distended so as to completely fill the cavity of the thorax, and to bear the impress of the ribs. The pleura covering them is usually found unaffected. They are white, dense, firm, and hard. They occasionally admit of partial insufflation, but this is not constant. Their weight when the affection is general is four or five times greater than natural. Their color is whitish with a shade of yellow, and it is uniform without any shading. Their section is smooth and opaque. They are resistant in some cases;—in others, as described by Weber, the finger can be pressed into them as into a fatty liver. They are quite exsanguine, and not a trace of blood or of the smaller bloodvessels can be discovered in them. The lobular texture is apparent—the interlobular tissue sometimes presents a slightly reddish tinge. The bronchi contain a tough mucus. The bronchial glands are enlarged, grayish, homogeneous, or in parts presenting a dry cheesy aspect. The extent of infiltration varies —sometimes the whole of both lungs are affected[7], sometimes only parts. When

[1] E. Wagner.

[2] Ib. (loc. cit.) Von Barensprung describes smaller masses in the lungs of newly-born children as sharply defined by a layer of well-developed fibrous tissue. The nodules in Dr. Wilks's case do not appear to have been thus encapsuled.

[3] Dr. Wilks (loc. cit.) Ricord (Clin. Iconograph. Pl. 28) gives a case where numerous softened masses were found in the lungs, but he questions whether they were not the result of pyæmic infection. Depaul, one of the earliest authors who has published authentic observations on this subject (Bull. Soc. Anat. 1837; Gaz. Méd. 1851; Mém. Acad. Imp. de Méd. 1853), has also found the centre of these masses softening into a puriform fluid, and sometimes presenting real abscesses, whose walls were formed by a yellowish gray and indurated tissue.

[1] Archiv, i. 146.

[2] Path. Anat. der Neugeborenen, ii. 47.

[3] Gaz. Méd., Par. 1855.

[4] Verhand. der Berlin. Geburtshülf Gesell. 1854, viii. 130.

[5] Behrend's Syphiologie, 1862, iii. 611.

[6] Loc. cit.

[7] Wagner in six cases found the whole of both lungs affected four times; once the half, and once the sixth part of the lung. Kostlin (Arch. Phys. Heilk. xvii.) met with it in four cases, generally limited to the lower lobe, or in isolated masses, varying in size from a pea to a pigeon's egg. In one child, who lived a fortnight after birth, the signs of the disease in the lungs appeared coincidently with ecthymatous pustules, with a measly rash, and with excoriations of the skin.

the affection is partial, there may be found in addition to the general infiltration isolated spots of the same kind, but resembling more or less the gummata before described, which sometimes merge at their margins into the neighboring infiltration. There is some discrepancy between the statement of different observers regarding the histological characters of this consolidation. Virchow described the air-vesicles as filled with epithelial cells, and Robin and Lorain make the same statement, and add that this process extends into the ultimate bronchial ramifications —but that at the same time the walls of the alveoli are thickened and rigid. Weber described the contents of the alveoli as cellular; while Wagner, from his recent researches, says that the characteristic by which this change may be distinguished from gray hepatization is, that nothing can be brushed or washed out from the interior of the vesicles, and that the disease essentially consists in a thickening of the alveolar walls, by which the cavity of the vesicles is gradually obliterated, and that in this process the epithelial lining is but little affected. This thickening takes place by the growth of an imperfect and scantily fibrillated tissue mingled with nuclei, and of a few fibre cells which are found in various stages of fatty and molecular disintegration; granular and fatty débris are also found in large proportions throughout the tissue. The interlobular texture is normal or contains a small amount of nuclear and cell growth. The vessels and capillaries are almost completely destroyed in the affected parts.

The submucous tissue of the bronchi is affected in the same manner as has been described as occurring in connection with the gummata, by a growth of nuclei limited to the superficial structures.

The bronchial glands are enlarged, and show concentric masses of cells bounded by a tough fibre tissue.

It will be observed that in both of these forms of disease the essential characteristic of the change described consists in a thickening of the walls of the air-vesicles by a growth of imperfect fibre tissue mingled with nuclei which tends to pass into an early molecular detritus, and that this change thus produces a structure apparently identical with the syphilitic gummata found in the liver.

Even in this form it would be very difficult to state any precise definition which might absolutely distinguish the process from the similar changes which occur in tubercular growths, and in the thickenings which affect the walls of the air-vesicles in tubercular pneumonia.[1] This difficulty is further increased in relation to some of the other changes which are frequently found in the lungs of syphilitic children, and also in some cases of adults. These, if separately distinguished, may be enumerated as follows:—

(*a*) Foerster has shown that lobular, vesicular, and broncho-pneumonia, either in a disseminated or in a confluent form, is very common in the lungs of children affected with hereditary syphilis and dying shortly after birth. In the majority of cases such pneumonias are identical in character with the ordinary forms of the disease, and consist only of an excessive development of epithelial cells and of their derivatives filling the vesicles.

(*b*) Suppurative changes occur at times in these spots and give rise to abscesses, the specific nature of which, however, may still be considered doubtful, since similar processes also occur in the non-syphilitic forms of catarrhal pneumonia.

(*c*) Foerster, however, has in some of these cases met with a gradual thickening of the walls of the alveoli, by the growth of a fibre tissue mingled with ovoid nuclei surrounding the spots of lobular pneumonia. These then become hard, smooth, pale and glistening, and in a later stage they show a yellow change which gradually extends throughout the nodule. This process has the greatest analogy with the growth of tubercular granulations, and if due to the syphilitic poison it would establish a close anatomical affinity between its effects and the changes which are most distinctive of tubercles. Similar appearances have been described, though on rather a larger scale, by Von Baerensprung and others, when the nodules so formed may attain the size of a walnut. Virchow has also remarked that these may coexist with peribronchitic thickenings, and that they may pass in spots into ulceration; and he further observes, that when met with in stillborn children of syphilitic parentage, their specific nature is rendered the more probable from the fact that tubercle proper is never met with as a disease of the fœtus.

(*d*) Virchow is also disposed to regard as being in some cases of syphilitic origin, indurated masses of fibrous structure more or less pigmented, and presenting a raspberry-like appearance, which are found scattered through the lungs. They are either seated immediately under the pleura, where they cause puckering and contraction, and also around the bronchi, where they form a cicatricial tissue, and they are often attended by pleural adhesions; cheesy spots are not uncom-

[1] Lancereaux (426) says that large granule cells are not found in tubercular growths; but this distinction is not, I believe, to be relied upon. My own observations on tubercular formations have convinced me that such granular cells are by no means uncommon in these.

monly found scattered through them. The nature of these is however still more doubtful, since such masses are very common in the indurating form of tuberculosis when there is no suspicion of syphilis.[1] Virchow states that the more fibrous structures present no distinctive features of difference from the indurating forms of chronic pneumonia which occur in the "grinder's asthma," and probably also in the whole class of diseases produced by the inhalation of irritating solid particles into the lungs.

(*e*) Virchow is further disposed to consider that fibrous induration of the pleura, and also certain forms of peribronchitic thickening which extend into the pulmonary tissue, may be due to the syphilitic dyscrasia, and that they may hold a place analogous to the cirrhotic indurations of the liver, and to indurations which are met with in the testicle under the same influence. Dr. Wilks has also raised the question whether some forms of "cirrhosis" of the lung may not have a similar origin, but this point still remains to be settled by further observation[2].

(*f*) Virchow has also met with a change in the lungs closely analogous to the brown induration to be hereafter described, but occurring independently of heart diseases, and which from its associations he thinks may also be placed in this category.

(*g*) Dr. Hermann Weber,[3] in a case where there was evidence of constitutional syphilis, and where nodules which he was disposed to regard as early forms of gummata existed in the liver, found in the lungs a general enlargement of the superficial lymphatics, which were filled with a thickened cheesy lymph which could be expressed from their interior. These enlarged lymphatics presented on section the appearance of white spots scattered over the lungs: their contents presented granular corpuscles with multiple nuclei. The bronchial glands were also enlarged, softened, and crowded with cells exhibiting considerable activity of growth. Dr. Weber regarded it as doubtful whether the pathological condition of the pulmonary lymphatics or of the bronchial glands constituted the primary affection. The appearances described, as Dr. Weber himself considered, differed in many respects from those which have hitherto been regarded as syphilitic.

Syphilitic growths in the lungs certainly bear a closer resemblance to tuberculous formations than is presented by almost any other morbid change in this organ. It is useless at present to revive the former speculations which have been held with respect to the influence of syphilis on the production of tubercle. The question, however, may be looked at in another aspect, and it would appear to be a subject for inquiry, how far a pre-existing "tuberculous" or "scrofulous" constitution may aid in the development of these special local manifestations. Syphilis has long been known to exhibit its most virulent characters in patients of this diathesis, and it appears to be not impossible that such a predisposition may render the lungs specially liable to suffer from the syphilitic affection, the characters of which may be partially modified by the tuberculous tendency. Tubercular changes are in many points of view so closely allied to the processes of inflammation that it has become increasingly difficult with further research to assign to them any specific character; but in the lungs at least, whether occurring in the form of granulations or of an infiltration, they are almost constantly attended by a fibro-nucleated growth of the alveolar wall, in which sometimes the fibrous and sometimes the nuclear element predominates. It would appear also by no means improbable, in the light of recent researches on the production of tuberculosis in the lower animals,[1] that various poisons, as well as simple irritants, may serve as the starting-points for tubercular changes in predisposed individuals. I would not, without much further personal experience than I possess on this subject, venture to affirm that syphilitic changes in the lung are identical with tubercle; but it is impossible to study the observations of those who

[1] Addison regarded these as pneumonia, and Virchow also speaks of them as the results of chronic pneumonia. For the reasons before given, I venture still to express an opinion respecting their tubercular nature. An appearance of this kind is described by Cornil as syphilitic (Bull. Soc. Anat. 1861).

[2] Wagner relates a case of the same kind. Vidal (Traité des Mal. Vén.) describes in a syphilitic patient a condition of fibrous induration surrounding the bronchi, and extending into the pulmonary tissue. It was chiefly limited to the lower lobes. The condition of the bronchi is not mentioned. Vidal notices the resemblance of the tissue to that produced by a chronic periostitis. Proof of the syphilitic nature of these is, however, wanting. Lancereaux (loc. cit. p. 424) considers that cicatricial contractions of the lung may also be due to this cause, but this must be regarded at present as being simply hypothetical.

[3] Path. Soc. Trans. xvii. (Two plates of the appearances in the lungs and liver.

[1] It is certainly a remarkable fact that in my experiments guinea-pigs inoculated with syphilitic virus were the only class that completely escaped secondary tuberculization; but this, when the difference of species is considered, would be no argument against the possible effects of this virus in the human subject. See lecture by the author "On the Artificial Production of Tubercle."

have investigated both processes, and particularly the researches of Virchow, without being convinced of the close analogy between them; and it would appear to me that the conclusion that some of the changes thus described as syphilitic have a quasi-tubercular nature, is at least quite as likely to be correct as the converse, viz., that a large number of processes hitherto considered tubercular should be ascribed, when found in syphilitic patients to the exclusively specific effect of this dyscrasia.

The clinical history of these changes is as yet an almost untrodden ground. The majority of the reputed syphilitic affections of the lungs have been observed in stillborn children, or when found in adults have only been accidentally discovered on post-mortem examination. Lancereaux cites a few instances where pulmonary symptoms had been present before death. In one of these, quoted from Vidal, and where the chief change was peribronchitic induration, there were the physical signs of consolidation at the bases, associated with slight hæmoptysis, little cough, and no fever, but with a dyspnœa gradually increasing in intensity, and apparently proving, at last, one of the causes of death. The duration of the disease in this case, after pulmonary symptoms were first observed, extended over two years.

In another case under Lancereaux's own observation, and where the presumed gummata had formed cavities surrounded by much induration, the affection was limited to one lung, and the physical signs were those of induration with excavation; hæmoptysis, however, occurred also in this instance, and the sputa, at first scanty, became subsequently copious and fetid; œdema of the legs, and slight pyrexia were present, and the patient died cachectic.

Lancereaux remarks that a unilateral affection of the lung, with signs of chronic induration or excavation, and in the presence of a syphilitic history, may lead to the diagnosis of its specific origin, but it must be remembered that the syphilitic affection is not invariably confined to a single lung.

In respect to treatment, Lancereaux cites several cases where a mercurial course has been followed by the cessation of phthisical symptoms, and by the improvement in some instances of the physical signs of the disease. I have more than once subjected phthisical patients with a history of syphilis, to treatment both by mercury and by iodide of potassium, but the results which I have hitherto obtained have been by no means favorable. The treatment by iodide of potassium would appear to be the least dangerous, and the most deserving of a more extensive trial.

BROWN INDURATION OF THE LUNG.

By Wilson Fox, M.D., F.R.C.P.

Synonyms.—Pigment Induration (Virchow); Brown Condensation (Zenker); Carnification Congestive (Isambert and Robin).

History.—This state of the pulmonary tissue occupies a doubtful ground between the indurations which succeed to long-continued congestion and the process defined as chronic inflammation. Some of the difficulty of determining its exact nosological position depends on discrepancies in the statements made by different observers with respect to the exact changes which lungs to which this term has been applied have presented.

It was described originally by Andral[1] under the title of Hypertrophy of the Lung, and as existing in cases of chronic catarrh. He states that the change consists in an enlargement of the vesicles, together with thickening of their walls, and Rokitansky[1] appears to have observed a very similar condition. It has also been noticed by Hope[2] and Hasse[3].

Morbid Anatomy and Pathology.—The state to which the term Brown Induration is applied is the result of long-continued congestion, most commonly

[1] Prec. Path. Anat. ii. 516.

[1] Path. Anat. 1861, iii. 46. A drawing accompanies this description.

[2] Morbid Anatomy.

[3] Loc. cit. Hasse appears to have proposed the title of "Brown Induration," which seems, from its simplicity, to be the most eligible for this affection.

arising either from marked incompetency or constriction of the mitral valve. Virchow,[1] who first after Andral gave a minutely detailed account of its appearances, also describes the lungs as enlarged, prominent, and not collapsing when the thorax is opened. They feel more compact than the normal lung, and they are also heavier and inelastic; they crepitate but little, and have a peculiar tint of yellow, shading into a brown or a reddish-brown. On section the tissue is dense and is speckled with red spots of variable size, shading into blacker tints, and between these also the tissue has a more or less rusty appearance. A brownish fluid (brown œdema, Virchow) exudes on pressure. Virchow described the essential characteristic of this condition as depending on the accumulation of hæmatoidine in the epithelial cells of the air-vesicles, which are either natural or more or less enlarged, and also in granule cells, which probably result from the transformation of the former. The pigment is for the most part in the form of granules, insoluble in acetic acid, but which are destroyed by caustic alkalies and by sulphuric acid. Various transformations of the yellow pigment into black granules can be seen within the cells themselves, and later it is found free in the walls of the alveoli and in the interstitial tissue. Further accounts of this state have been given by Friedreich[2] and by Buhl.[3] The latter describes and figures a series of varicose dilatations of the capillaries coexisting with the pigment in the walls of the alveoli. Friedreich's description of the filling of the aveoli with enlarged epithelial cells, and with the products of their proliferation, agrees very closely with Virchow's.

The point on which most difference of opinion exists is that which refers to the thickening of the alveolar walls. Virchow does not describe this change, and Zenker[4] says that he has not met with it. Rokitansky, however, figures it, and Isambert and Robin,[5] who, under the title of "Carnification Congestive," have described a very similar condition, state that the walls of the alveoli and the interstitial tissue, in addition to containing a large quantity of pigment, are infiltrated with an amorphous exudation matter. In the specimens which I have examined I have found such thickenings in considerable tracts, together with a distinct increase of fibrous tissue in the walls of the alveoli; but this change is not uniformly present, and in other places the alveoli are found filled with catarrhal cells, while their walls present no other change than that arising from the distension of the capillaries. I have also observed a considerable thickening of the coats of the branches, both of the pulmonary artery and of the pulmonary vein; an appearance, however, which has not been described by some writers.

The change in the lung has appeared to me to be referable to two stages. In the first there is intense congestion, sometimes general, but more commonly found in limited parts, and in these congested parts a considerable amount of pigment may be seen in the pulmonary epithelium. Such parts float in water, and are more or less œdematous, yet crepitant, but comparatively inelastic. Their tint is of a uniform reddish brown. In the later stages the pulmonary alveoli gradually become filled more or less completely with epithelial products resembling those of catarrhal pneumonia, and the tissue, to a great extent, loses its crepitant character. In this stage also it is not so prominent, and closely resembles a congested and collapsed lung, except that the surface is finely granular, and is mottled with spots of yellowish pigment on the brown and indurated tissue. It has none of the friability of ordinary pneumonia, but is comparatively tough and inelastic. This latter change corresponds with the "carnification congestive" of Isambert and Robin, and the parts so affected sink in water, but not in all portions. The alveoli are loaded with epithelial cells and with granule cells containing an excessive amount of hæmatoidine. Isambert and Robin describe the pigment as sometimes existing in the crystalline form, and this, in the condition of melanine, can be seen in the walls of the alveoli and in the fibrous tissue surrounding the arteries and veins.

The extent of lung thus implicated varies considerably. The change may exist only in patches, or it may extend to considerable tracts of tissue. I have seen it throughout the greater part of the lower lobe. Isambert and Robin have seen it affecting the whole of one and the greater part of the opposite lung.

The appearances thus presented are quite different from those of hemorrhagic infarcta, though these are not unfrequently present in other parts of the lung. The parts affected want both the density and also the prominence of portions of lung into which hemorrhage has occurred, and the escape of the coloring matter of the

[1] Archiv, i. 1847, p. 461 et seq.

[2] Virchow, Arch. x. 201. Friedreich describes corpora amylacea as existing in such lungs. I have also seen these under similar circumstances.

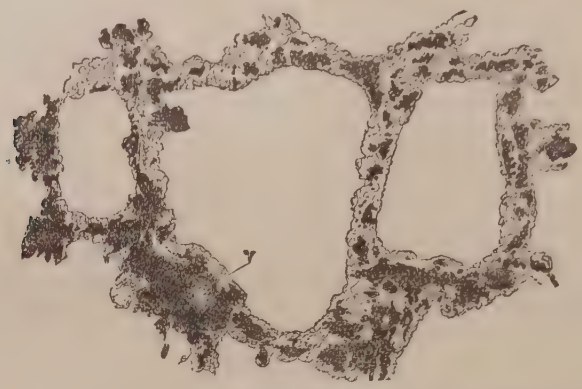
[3] Ib. xvi. 559. Drawings accompany this description.

[4] Beiträge zur Normalen und Path. Anat. der Lungen.

[5] Mém. Soc. Biol. 1855, 2e Ser. ii. p. 3 et seq.

blood appears to be due to mere capillary rupture, and not to any extensive extravasation. The nature of the change seems to depend on long-continued congestion gradually giving rise to a catarrhal pneumonia of a chronic type, and the thickening of the alveolar walls may probably occur in the later stages of the process—

[Fig. 41.



Pigmentation of the Lungs.—From a woman, æt. sixty-five, with slight emphysema. Showing the situation of the pigment in the alveolar walls, and around the bloodvessel *v*. × 75. (Green.)]

thus creating an analogy with some of the other forms previously described.[1] Zenker states that this pneumonia may pass into true hepatization, though fibrinous exudations are commonly wanting. The pneumonia, however, may at times be mingled with so much extravasation as to give it a hemorrhagic character.

The enlargements of the lung described by Virchow has not appeared to me to be essential to the process—at least such enlargement has not existed in two of the best marked instances of the disease which have come under my own observation. It would appear not improbable that, when such enlargement has existed, the lungs had been affected by emphysema during or prior to the other changes. Zenker states that an extreme degree of atrophous emphysema existed in some specimens which he examined.

This change, according to Zenker, appears to be more common before than after the age of forty.

Symptoms.—Bamberger[2] describes the earlier conditions of this state as associated with diminished resonance on percussion, together with weakened respiratory murmur; but these physical signs are common to many cases of pulmonary congestion from cardiac disease when the induration now described does not exist.

Dyspnœa is commonly present, and cyanosis is observed in extreme cases; but neither these nor the rusty sputa often seen are necessary signs of the condition in question.

Isambert and Robin describe dulness on percussion, together with bronchial breathing over the affected parts, and this also has existed in one case which I have observed.

The temperature in both the best marked cases which have come under my observation has been elevated, but not exceeding 102° Fahr. In one case a fluctuating pyrexia, sometimes reaching 102°, and on other days not exceeding 99½° or 100°, continued during nearly a month—death finally taking place from gradually increasing asthenia and cyanosis: the heart was much hypertrophied, adherent to the pericardium, and presented extensive disease of the mitral valve. Another case was complicated by erysipelas of the leg passing into gangrene. It must therefore be regarded as doubtful whether the pyrexia depended on the pulmonary condition. Rusty and blood-stained sputa are common, but neither these nor any of the physical signs as yet observed afford any positive grounds for the diagnosis of this affection, though there might be strong reason to suspect its existence from the persistence of signs of consolidation during a long period, and associated with cyanosis and dyspnœa depending on marked disease of the mitral valve.

The Treatment must be mainly directed to the cardiac conditions present.

[1] Grisolle, p. 71, describes two cases of chronic pneumonia associated with heart affection, but the appearances observed do not show positively that they belonged to this class. In one the condition was simply that of gray induration; in the other the tissue was of a reddish tint and finely granular, but in other parts it presented more the appearance of being of recent origin.

[2] Lehrb. Krank. des Herzens, 204.

The indications for the relief of pulmonary congestion, such as the application of revulsives and counter-irritants, and the internal administration of stimulants, are those which would appear to be the most suited to this state. (See Secondary Pneumonias, p. 235.)

CIRRHOSIS OF THE LUNG.[1]

By H. Charlton Bastian, M.A., M.D., F.R.S.

Nature and History.—This is a rare disease, mostly of a chronic type, in which the individual has suffered, perhaps for many years, from cough and muco-purulent expectoration, with or without haemoptysis; in which the wasting is not very marked, whilst the constitutional symptoms of the ordinary form of phthisis are almost absent. There is usually marked dulness, accompanied by immobility and retraction, of one side of the chest, with or without cavernous sounds on auscultation; whilst there is generally increased resonance, accompanied by puerile respiration on the opposite side. The heart is more or less displaced towards the affected side; whilst there may be signs of dilatation and hypertrophy of its right cavities, associated with anasarca and ascites. After death the lung on the retracted side is found to have become shrivelled to one-half or even one-fourth of its natural size—owing to its conversion into a tough fibrous material, with obliteration of its air-cells and usually more or less dilatation of its bronchi; whilst that on the opposite side is much enlarged, and presents no evidence of the existence of tubercle or chronic disease.

This pathological condition was incidentally alluded to by Laennec[2] as a variety of dilatation of the bronchial tubes, and was afterwards referred to by Dr. C. J. B. Williams, only a few weeks before the appearance in 1838 of a most interesting memoir on the subject by Sir Dominic Corrigan.[3] In this memoir the disease was first really described, so far as the state of knowledge at the time allowed, and an entirely new interpretation was given of its pathology. The above name was proposed, on account of the close resemblance between the pathology of this affection and that of cirrhosis of the liver. And so far as it serves to indicate the pathological relationship between the two diseases it is a good one; though, if its derivation be considered, the word "Cirrhosis" (from κιρρός, yellowish or tawny) is as inapplicable as it can well be to the lung affection about to be described.[1]

Whilst Laennec, in his admirable account of dilatation of the bronchi—a morbid state which had never been previously described—looked upon the condensation of tissue around the dilated tubes as being invariably secondary to and the effect of the dilatation, Corrigan, on the other hand, maintained that in a certain number of cases, which he proposed to range under the name "Cirrhosis of the Lung," the fibroid metamorphosis and induration was the primary and essential anatomical lesion, and that the dilatation of the bronchi was only a secondary effect. Omitting for the present the consideration of the question as to whether Corrigan was correct in the explanation he offered of the mode of origin of the bronchiectasis, I may state that his main position appears to have been a correct one. It seems to be undoubtedly true that, in a certain number of cases in which dilated bronchi have been met with after death, an original fibroid conversion and shrinking of the lung-tissue has entailed this as a consequence: the bronchiectasis has been secondary, and not primary.

Notwithstanding the enunciation of Corrigan's views, however, the French

[1] This article also includes some account of the pathology of Bronchiectasis (p. 826 et seq.).

[2] Diseases of the Chest, translated by Forbes, 4th Ed. 1834, p. 107.

[3] Dublin Medical Journal, 1838.

[1] The name "Cirrhosis" was, in fact, originally given by Laennec to the now well-known liver disease, under the influence of a misconception as to its nature. He thought that it was due to the deposition within the organ of a peculiar morbid substance of a tawny or rust-brown color. These patches and islets, however, are now known to be only the natural acini of the liver, bile-stained and isolated by what is the real anatomical element of the disease—the new growth of fibre-tissue.

pathologists, with the exception of M. Jaccoud, adhere to Laennec's interpretation of the sequence of these phenomena; and Cirrhosis of the Lung is, moreover, scarcely considered to be entitled to rank as a distinct disease by many English and German pathologists.

From facts subsequently to be mentioned, it will be seen that Corrigan placed too much stress upon the dilatation of the bronchial tubes. This is not an essential element in the disease, but is, rather, a very frequent accompaniment. It will be observed that in several recorded cases bronchiectasis was either absent altogether or only very slightly marked. In these cases the fibroid infiltration and shrinking of the lung, which are the essential characters of Cirrhosis, existed alone. Those who still doubt the propriety of regarding this as a disease with clinical characters of its own, distinguishable from bronchiectasis, may perhaps be influenced by an attentive consideration of the following facts.

From the analysis of 43 cases by M. Barth,[1] and by Lebert[2] of 24 cases of bronchiectasis, it appears that this affection most notably increases in frequency with advancing age, and that by far the larger proportion of cases are met with in persons who are more than 60 years old. Thus in Lebert's 24 cases, it was met with four times before the 10th year, ten times from the 10th to the 55th year, and ten times from the 55th to the 85th year; whilst, according to Barth it was met with as follows:—

No. of Cases.	Age.
2	1 to 20th year.
3	20—30 "
3	30—40 "
4	40—50 "
5	50—60 "
7	60—70 "
19	beyond the 70th year.

But an analysis of 30 cases of Cirrhosis of the Lung, which I have collected, appears to show a most striking difference as regards the prevailing age at which this lung affection is met with in the post-mortem room,[3] and that at which dilatation of the bronchi is encountered. Thus, in the 30 instances of Cirrhosis the following ages were attained:—

No. of Cases.	Age.
2	1 to 15th year.
3	15—20 "
7	20—30 "
9	30—40 "
2	40—50 "
3	50—60 "
4	60—70 "

From these figures it appears that 19, or almost two-thirds of the total number of cases of Cirrhosis, occurred between the ages of 15 and 40; whilst of Barth's 43 cases of bronchiectasis, only 7, or less than one-sixth of the total number, were met with at the same ages. On the other hand, more than one-half the cases of bronchiectasis (26 : 43) were in individuals over 60 years of age; whilst rather less than one-seventh (4 : 30) of the cases of Cirrhosis were encountered after the same year. Even these facts alone tend strongly against the view that well-marked fibroid infiltration with shrinking of the lung is to be considered as a sort of sequence of dilated bronchi. Whilst, on the other hand, seeing that bronchiectasis is met with in such a large proportion of the cases of Cirrhosis of the Lung occurring at the ages above mentioned—when dilatation of the bronchial tubes is otherwise very rare—there are strong grounds for the opinion that such a condition of the lung is especially favorable to the production of more or less dilatation of the bronchi. Other striking differences, however, exist between the two affections. Thus, well-marked Cirrhosis is almost invariably confined to one lung: not so with bronchiectasis. More or less hæmoptysis was present in more than one-half (17 : 30) of the cases of Cirrhosis, in only four of which was anything which could be called "tubercle" said to be present in one or other of the lungs;[1] whilst the same symptom was met with in less than one-sixth of Barth's cases of dilated bronchi (7 : 43)—and of these no less than four were also suffering from phthisis. There are differences, moreover, as regards sex. According to Lebert,[2] dilatation of the bronchi is as common in females as in males; whilst only one-fifth of the total number of cases of Cirrhosis have been observed in females.

Though believing that in the majority of cases condensation of the lung-tissue from fibroid metamorphosis precedes the dilatation of the bronchi with which it is so often associated, still it would appear quite obvious that in some other cases the order is just the reverse. What

[1] Rech. sur la Dilatat. des Bronches, Mém. de la Soc. Méd. d'Observat. de Paris, tome iii. (1856), p. 469.

[2] Anat. Patholog. tome i. p. 620.

[3] Although it is perfectly true that Barth's cases (with one exception) were collected at the general hospitals for adults, and also at the Salpêtrière, and therefore may not at all fairly represent the frequency of the disease in childhood; still, that his figures do show the determining influence of age may be also seen from the fact that in the course of six years 25 examples were met with at the Salpêtrière, whilst only 18 were met with during 25 years at the general hospitals of Paris.

[1] In only one of these four cases did the "tubercle" exist in the cirrhosed lung.

[2] Barth does not give the proportion of males to females in his observations.

Laennec maintained to be the rule, does really obtain in some cases: the cirrhotic change is then secondary to the bronchiectasis. The two morbid conditions have undoubtedly most intimate relations with one another, and occasionally it may be difficult to pronounce which was the primary lesion. There is no reason, of course, why the cirrhotic change should not invade a lung whose bronchi are dilated, just as it invades one in which the bronchi are healthy. But in the cases where this has been the order of events, the amount of condensation and induration of lung-tissue is far greater than what is often entailed by the mere dilatation of the bronchi. So that, although there may have been some amount of pre-existing bronchiectasis, Cirrhosis afterwards becomes the predominant affection.

But there is another condition of the lung, known for the most part by the name "chronic pneumonia," under which are recorded cases that may better be regarded as instances of Cirrhosis of the Lung. Much uncertainty and confusion have resulted from the use of the former term, on account of the shifting signification which has been given to it by different writers. But the perusal of Charcot's memoir, "De la Pneumonie Chronique,"[1] and of the account given by Grisolle in the last edition of his work "De la Pneumonie," cannot fail to convince the reader that, instead of referring to any condition of lung especially characterized by the impaction of the air-vesicles with a more or less solid accumulation of cells or cellular débris, these writers understand this name to imply a fibroid infiltration of more or less of the organ, and the gradual substitution of a tissue of this kind in the place of the proper substance of the lung.

Pathological states of the lung very similar to, or even identical with, this, were originally described by Laennec[2] as forms of infiltrated "tubercle," under the names of *gray tubercular infiltration* and *jelly-like infiltration*. The tubercular nature of these morbid states was afterwards denied by Chomel,[3] who looked upon them as evidences of a non-specific chronic pneumonia—a view which has been more or less adopted since his time by succeeding pathologists. Andral[4] described a *red*, a *yellow*, a *gray*, and a *melanic induration* of the lung, which seems to represent only different stages and varieties of a fibroid infiltration of the organ, and correspond with what Hasse,[1] Rokitansky,[2] Förster,[3] and other German pathologists mean by *interstitial pneumonia* and *lungen-induration*. Lebert[4] also described a *hepatization indurée*, and a *hepatization jaune;* whilst Cruveilhier,[5] referring to the later stages of the same pathological transformation, spoke of a *phlegmasie indurée* and an *induration mélanique ardoisée*, understanding that the essence of these conditions was a "métamorphose fibreuse" of the proper lung-tissue. Addison[6] also described two of the sequences of acute pneumonia under the names *albuminoid induration* and *iron-gray induration*. With regard to the second of these pathological states, this undoubtedly corresponds with the fibroid induration of other writers, and a careful examination of Addison's plates, together with a comparison of the descriptions given of the two conditions, almost suffices to show that the first is but a rarer modification of the second pathological state—into which it often seems to pass by insensible gradations. Dr. Wilks[7] also describes chronic pneumonia as a fibroid induration of the lung substance, due to an actual new growth of fibre-tissue which slowly increases in amount.

Thus there seems to be a pretty general agreement between the writers I have named (some of the principal of those who have written upon the subject), concerning the essential nature of the condition which often goes by the name "chronic pneumonia." When affecting any considerable extent of the lung it has been generally recognized as a condition of great rarity. Charcot imagined that at the time he wrote there were only about ten or twelve cases on record, which could be indubitably regarded as examples of this disease. Grisolle, also, had only met with six cases during his long experience. Both these writers believe that this state of the lung may be the almost immediate sequence of an ordinary acute pneumonia, although they think that at other times it is chronic from the first, and commences in the most obscure and insidious manner. Both these writers also, as well as most of the others I have mentioned, are fully satisfied that the

[1] Paris: Thèse, 1860. Containing copious references.

[2] Diseases of Chest; translated by Forbes, 4th Edit. 1834, p. 256.

[3] Art. "Pneumonie Chronique," Dict. in 25 vols., 1842.

[4] Précis d'Anat. Patholog. tome iii. p. 517, and Clinique Médicale.

[1] Patholog. Anatomy (Syd. Soc. Translation), 1846.

[2] Man. of Path. Anat. vol. iv. (Syd. Soc. Translation), p. 60.

[3] Lehrb. der Patholog. Anatom. p. 296. Jena, 1862.

[4] Anat. Patholog. tome i. p. 648.

[5] Ibid., livraison xxxii. p. 8; and Anat. Patholog. Génér. tome iii. p. 608.

[6] Guy's Hosp. Reports, 1843, p. 365.

[7] Lect. on Path. Anat. 1859, p. 236.

minute anatomical characters of this so-called chronic pneumonia are essentially similar to that of the ordinary indurated tissue surrounding vomicæ or foreign bodies in the lungs. I need only add that the tissue-changes in these cases are essentially similar to those which Dr. Sutton has described as "fibroid degeneration of the lung," and that such a change, prevailing to a wide extent, is the anatomical characteristic of Cirrhosis of the Lung.

It seems to me expedient to do away altogether with the name "chronic pneumonia," as an appellation for the pathological changes in question. This seems desirable for the following reasons:—1. Any pathological state to which the term "chronic pneumonia" is applied ought to be characterized by anatomical characters similar in kind to those which are met with in the acute condition—conditions which are fulfilled by the "chronic lobular pneumonias" of phthisical patients. 2. Admitting that the fibroid overgrowth and substitution, which has been hitherto styled "chronic pneumonia," is sometimes the direct sequence of an acute pneumonia, still this secondary condition is not a modified persistence of the old state, but is due to the supervention of an entirely new and different process: in these cases, in fact, we have to do with a sequence to, rather than with a chronic persistence of, the original malady. 3. Although such a pathological state is occasionally the direct sequence of an acute inflammatory condition, still in the large majority of cases it seems to be due to an essentially chronic process—to one which is deficient in some of the most important characters of an inflammatory change, and which more closely resembles a mere infiltrating new growth.

The term "interstitial pneumonia" seems to be almost as unsuitable as that of "chronic pneumonia," as an appellation for the fibroid indurations in question. Whether such changes are met with in the lung, in the liver, or in any other organ, their mode of initiation, progress, and minute anatomical characters, seem to be essentially similar. They advance insidiously, in the great majority of cases, without affording the least clinical evidence that the patient is suffering from an inflammatory disease;[1] and when the organs in which such changes had been advancing are submitted to microscopical examination, there is a similar absence of the signs of an inflammatory process. A new growth is met with, supplanting the proper anatomical elements of the part, and it seems to me to be no more suitable to speak of such a process as an inflammation than it would be to apply the same term to a slowly increasing but infiltrating cancerous growth.

The more partial and local changes might simply be styled "fibroid indurations," reserving the term "Cirrhosis" for the more extensive and advanced change, when it affects either an entire lung or at least one lobe of the organ.

From what has been said it will be seen how intimately related Cirrhosis of the Lung is, not only to bronchiectasis, but also to what has been hitherto called "chronic pneumonia." It will not be so much a matter of surprise, therefore, that some of the cases of which I have given an abstract in this paper, have been originally recorded under one or other of these names. No sharp lines of demarcation can exist between fibroid indurations of the lung ("chronic pneumonia" of other writers) and Cirrhosis, because they are merely different degrees of one and the same pathological condition. Therefore, one or two of the cases that I have included amongst the thirty instances of the disease on which this paper is based, may seem doubtfully entitled to the latter name; but I have placed them in this series precisely because they serve to indicate this relationship, and to show what are the early stages of the disease which we are now describing.

Since diseases have no distinct and independent existence, but are merely groups of symptoms, or of pathological changes, which tend to repeat themselves with varying degrees of frequency, it is only to be expected that intermediate conditions should at times present themselves. Our nomenclature and classification of these sets of symptoms, or pathological changes, must inevitably be more definite and sharply defined than actual facts or occurrences would warrant. We can but seize upon certain combinations of symptoms or changes which are apt to recur, and ticket them in their typical condition as so many "diseases;" though in doing this we should ever recollect that the symptoms or changes are not distinct and independent, but are variously related to, and miscible with other possible combinations. With the distinct understanding that the diseases enumerated in our nosologies vary immensely, not only in respect to the frequency, but also in respect to the definiteness of character, with which they tend to recur; still, one must regard all such described diseases as little better

[1] Dr. Wilks says (loc. cit. p. 237): "For my own part I believe such a process is essentially chronic, and at no time, if an opportunity had been given for examining such a lung, would it have presented any different appearances, except in amount; growing, indeed, like a tumor, and, like it, having, no doubt, some elementary forms preceding the fibrous structure, but the mode of production and development so slow and continuous that no distinct stages or changes in the structure can even be distinguished."

than rallying points, round which special groups of symptoms or changes may be conveniently ranged.

The disease which we are now considering is comparatively rare, and it can only be arbitrarily marked off from the fibroid indurations of smaller extent, out of which it is developed. Still a certain set of symptoms do tend to recur in association with a certain set of advanced anatomical changes, and these have been ticketed as a disease which is distinguished by characters of its own, as much clinical as anatomical. On these grounds Cirrhosis of the Lung has the same right to be considered as a distinct disease that many others possess whose claim to a place in our nosologies is unquestioned.

This paper is essentially based upon an analysis of thirty recorded cases of the disease.

One of these cases was originally reported by Sir Dominic Corrigan; one has been taken from Dr. Sutton's paper; and two others are from M. Charcot's memoir, "De la Pneumonie Chronique." On the other hand, seven cases have been included which had been described under the head of Bronchiectasis.[1] One of these was recorded by Laennec (though quoted by Corrigan as an instance of Cirrhosis); one was observed by Dr. Stokes; one by Dr. Bright and Dr. Hughes;[2] two are from M. Barth's memoir; whilst the last of the cases, previously recorded under the head of Bronchiectasis, has been taken from MM. Hérard and Cornil's recent treatise.[3] The remaining nineteen cases of which I have given abstracts were recognized as cases of Cirrhosis of the Lung, and sixteen have been published as such—fifteen in one or other of the periodical publications of Great Britain and Ireland, and one in Paris during the present year (1867)[4] by M. Jaccoud. The other three cases have not been hitherto published: one occurred in the practice of Dr. Gull at Guy's Hospital, and one in that of Dr. Pollock at the Brompton Hospital, and to each of these gentlemen I have to express my best thanks for their kindness in placing reports at my disposal. To Dr. Wilks I am also much indebted for granting me access to, with permission to publish, the records of a case which formerly occurred in the practice of Dr. Addison at Guy's Hospital.

In order to show the kind and range of variation met with in different cases, I have deemed it most advisable to give a tabular abstract of these cases, which it is hoped will be of use for future reference.[1]

Pathological Anatomy.—Adhesions of the pleural surfaces, serving to unite the affected lung to the parietes of the chest and to the diaphragm, have been met with in almost every case. They were reported as present in twenty-six out of the thirty cases; only in one case were they stated not to exist, whilst in the remaining three the presence or absence of adhesions was not noted. Of the twenty-six cases in which the adhesions are described as existing, they were somewhat loose in five, but firm, tough, and often even cartilaginous in consistence in the twenty-two other cases. In nine of these the adhesions were more or less partial, whilst in thirteen, or nearly one-half of the total number, they were general, and the lung was at the same time usually much reduced in size. Adhesions between the diseased lung and the pericardium were not uncommon, and in one case the posterior surface of the greatly enlarged opposite lung was also united to the diseased organ. Where the adhesions were well developed and general, it was frequently necessary to cut the tongue out of the corresponding side of the thorax; and more or less extensive plates of firm fibro-cartilaginous looking material were found covering a certain portion of the surface of the organ, and gradually shading away peripherally into an ordinary tough fibrous coating. This layer over certain parts of the lung, having a fibro-cartilaginous appearance, may be more than an inch in thickness—and then its inner strata evidently correspond in situation to what had previously been proper lung-tissue. In only one case was any fluid found, and in this the pleura is stated to have been nearly one inch in thickness; whilst in a cavity between the adhesions, which were only partial, there was contained nearly a quart of clear serum. The lung was, moreover, only as large as a man's fist; its tissue was remarkably hard, and its bronchi were not dilated.

The size of the lung, in the more recent cases, has undergone no appreciable alteration; in all the more chronic cases, how-

[1] These are here recorded, because they not only serve to show the intimate and natural relationship existing between the two "diseases," but also because, owing to the extent and character of the morbid changes met with, they have almost an equal title to be ranked under either head.

[2] See Guy's Hospital Museum, with description in catalogue.

[3] De la Phthisie Pulmonaire, Paris, 1867.

[4] It seems only right to state that this paper has been written nearly three years and a half—ever since October, 1867. A few other cases have been recorded since this date.

[1] These tables, except those of the first five cases, have been omitted from this edition; their substance being fully conveyed in Dr. Bastian's article.—H.]

ever, it has exhibited a variable amount of shrinking. This is often very considerable: in one remarkable case it was scarcely the size of a man's hand, and there was no pleural effusion of any kind (to help to bring about the contraction), similar to what existed in the other case, in which the size of the lung was reduced to such an extreme degree. All intermediate grades are to be met with between this amount of contraction and the normal dimensions of the organ. On section, it is often seen that the lobes of the lung are firmly connected together by a dense fibro-cartilaginous material, similar to what more frequently occurs in connection with the pleura on the surface of the lung. The tissue of the organ varies much in appearance in different cases, owing to the different degrees of progress which the disease may have made; and also to the varying amount of black pigment present, and to the number and mode of distribution of dilated bronchi or ulcerated caverns throughout its substance. Occasionally, islets of healthy lung-tissue are left here and there, in the midst of the fibroid induration. The disease may affect only one lobe, the two lobes unequally, or the whole organ pretty uniformly. When existing in its early stages either generally or partially, the nature of the pathological change is even then most obvious to the naked eye. The texture of the lung being firm, tough, dense, and incapable of being broken down by the finger, one sees a smooth or only very slightly granular surface of a blackish or iron-gray color, intersected in all directions by white bands of ligamentous-looking tissue, often forming a sort of trabecular network, and dotted with white circles of varying sizes, produced by the cut walls of the thickened smaller bronchial tubes. Very often, in its early stages, this invasion of fibre tissue is most distinctly seen to extend inwards from a greatly thickened pleura; for continuous with it may be seen portions of lung tissue which have been completely converted into a fibro-cartilaginous looking material; while this may pass, internally, into a simple ligamentous-looking texture. Still further internally there is a trabecular structure, such as I have just described, the white bands of which become narrower and narrower, and may gradually fade away into almost unaltered lung-tissue. In other cases where the consolidation spreads from different centres within, rather than from the surface of the organ, the fibroid thickening and white bands seem to radiate principally from the thickened walls of the bronchi. In its more advanced form, almost the whole organ, or large parts of it, grate under the knife when a section is made, cutting more like a tendon or mass of fibro-cartilage than anything approaching to normal lung-tissue, and whole tracts of it may in this later stage present the smooth yellowish-white appearance of cut tension, and be almost free from pigment. As the fibroid induration advances, air-cells and vessels become more and more obliterated; the lung-tissue gradually yields less and less fluid when squeezed, and becomes at the same time more incompressible.

In the great majority of instances, changes such as I have mentioned are those which are apparent from the very commencement of cases of Cirrhosis of the Lung. But on those rarer occasions when the cirrhotic process is the direct sequence of an acute pneumonia, the first process is one which has been termed *induration rouge* by Andral and other writers. We have an instance of this change in the case recorded by Dr. Sutton,[1] when, on section, the upper lobe of the right lung was of a *dark red* color and the interlobular tissue appeared to have undergone an increase. Only a very small quantity of fluid appeared on the divided surface, and the tissue did not easily break down under the finger. The whole of the lower half of this lung was solid, firm, and somewhat tough. It had a *reddish-gray* color and offered some amount of resistance to the knife; whilst it sank in water, and exuded scarcely any fluid on pressure. Here the lower lobe was evidently in a more advanced stage of the disease than the upper, and it seems to have been in much the same condition as the upper lobe of the left lung in the case of M. Legendre. A more advanced stage is recorded in the case of the child reported by Sir D. Corrigan, where the right lung was solid, non-crepitant, *grayish-red*, tough, and traversed in all directions by thickened white bands of fibro-cellular tissue—though there still seemed to have been no contraction of the affected organ. But in the case of the man observed by M. Charcot, who had suffered from an attack of acute pneumonia about four months before his death, the disease seems to have made rapid strides, and presents us with a still more advanced phase. The whole right lung was pretty equally affected,

[1] It seems to me most probable that this state of the lung was the sequence of an acute pneumonia. This must either have been the case, or else it must have been due to the supervention of an acute fibroid change without the existence of a previous pneumonia. Even if the latter alternative were true, the results would seem to be much the same in either case, since the condition of this lung appears to agree in all respects with Andral's description.

and had undergone an evident diminution in volume.[1] Its tissue was so dense that the finger could not penetrate it, and, on section, it resisted the scalpel like fibro-cartilage. The three lobes were seen to be firmly united, and the surface of the section was smooth, non-granular, *grayish-blue marbled with black*, whilst pale ligamentous partitions of fibrous tissue subdivided the lung in all directions, and formed a minute network.

These are the stages by which the Cirrhosis that supervenes as the sequence of an acute pneumonia appears gradually to approximate to the condition of the lung which is characteristic of the earlier stages of the more chronic process.

In only one out of the thirty cases which I have tabulated is there any certain evidence of the existence of even a small quantity of "tubercle" in the cirrhosed lung. This was in the case of M. Jaccoud, when a very small quantity of crude and slightly softened[2] "tubercle" was found in the posterior part of the apex of the lung affected. In three other cases, however, a small amount of "tubercle" was said to have beer found in the non-cirrhosed lung.

But, although the existence of "tubercle" in cases of Cirrhosis seems to be a perfectly accidental occurrence, the same cannot be said with regard to the presence of *ulcerated caverns* in the indurated lung-substance, since these have been met with in about one-fourth of the total number of cases. Sometimes these caverns appeared to have been formed slowly, owing to the molecular disintegration of portions of the new tissue which had undergone a fatty metamorphosis, whilst at other times they have originated by a gangrenous process, as occurred in one of M. Charcot's cases. Here, one of the excavations had irregular walls, and seemed to have arisen by a gangrenous process about two months previous to the patient's death; whilst another appeared to have been on the eve of forming, and was represented by a softened patch of yellowish tissue, with a disagreeable, though not gangrenous odor. In a case reported by Dr. Mayne, the patient died from the supervention of gangrene in the diseased lung, though there were no caverns. Towards the lower part of the consolidated organ the tissue had the olive or purple tint of gangrene with a corresponding odor. In all the recorded cases, however, in which caverns existed in the lung, save the one previously mentioned, they seem to have been formed by the slower process of ulceration or molecular disintegration, since there was no preceding history of gangrene. In two cases a recent coagulum of blood was found in the ulcerated cavity; in one the cavity was old and very large, being 4″ in length by 2¾″ in breadth; in two the cavities were single and small; in one there was ulceration of portions of the walls of two bronchial dilatations; whilst in another case there was a small excavation of the size of a hazelnut, whose nature was doubtful. In only one of these cases were there several excavations existing in the same lung.

In addition to these ulcerated cavities—having more or less ragged walls, and bounded by lung-tissue rather than by an altered mucous membrane—there are usually found other cavities and enlarged canals resulting from *dilatations of the bronchi*. These are not commonly met with in the early stages of the disease, such as have been hitherto spoken of under the name of "chronic pneumonia," and they are by no means always present, even when the disease is fully established and when great contraction of the lung has taken place.

There was no dilatation of the bronchi at all in one-fifth of the thirty cases which I have collected, and in four other cases it was present only to a very slight extent. In one-third of the cases, therefore, it has been either altogether absent, or else an insignificant feature of the disease. In the remaining two-thirds of the cases it existed to a variable extent. In one of the cases—that of a child—where the amount of dilatation was extreme, and in which, moreover, the fibroid change seems to have advanced upon a lung whose bronchi were already dilated, it was a most typical instance of what has been called *uniform* dilatation. The bronchi were found to be healthy as far as their first division, but beyond this point, instead of diminishing at the successive bifurcations, they preserved the same calibre as far as their termination—and in some places the diameter of a distal branch was even greater than that from which it proceeded. At their extremities there was a simple *cul-de-sac*, and no tendency towards the formation of an ampulla. The mucous membrane was grayish-black,[1] slightly villous, and evidently thickened. In two other cases recorded by Barth and occurring in adults, in which the amount of dilatation was extreme, it was of the *mixed* kind—consisting partly of cylindrical and partly of spheroidal dilatations. But in these two cases also it seems most probable, from a consideration

[1] It was one-third smaller than the right lung, which was noted as being very large. The actual amount of contraction of the cirrhosed lung, therefore, had not been very great.

[2] Even this was, therefore, in all probability, merely a cheesy patch of chronic lobular pneumonia.

[1] Most probably a post-mortem coloration.

of the histories of the patients, that dilatation of the bronchi had existed for many years before the fibroid change made any notable advance in the diseased organs.[1] The mucous membrane lining the variously dilated bronchi was in both cases smooth, dark red, and thickened. In only one other case was the amount of bronchial dilatation extreme. Here the lower lobe of the affected lung contained an extensive series of bronchial cavities between the size of a fowl's egg and that of a sparrow, some of which were partly filled by a semi-solid mucous secretion. In one case one of the cavities was as large as an apple, and in one two large cavities—each as large as an egg—were the only ones existing. In other instances the dilatations were much smaller; thus in one case a vast number of little cavities existed, varying in size between that of a pea and a marble, and all full of a muco-purulent secretion. In other cases caverns, varying in number, and of all sizes between these extremes, were encountered. The more or less spheroidal cavities were almost invariably associated, also, with cylindrical dilatations of the tubes; and in some cases the rounded enlargements were decidedly more common towards the periphery of the organ. The condition of the membrane lining the dilated bronchi has only been specified in twelve cases: in seven of these it was dark red, congested and thickened (and in two of them even velvety or slightly villous), whilst in the five others it was rather a smooth, dull, or glistening membrane. In none of the cases is there any mention made of the slightly prominent transverse striæ which are so often met with in dilated bronchi according to Barth, and which I have myself seen extremely well developed in one instance, where the mucous membrane covering an enormous extent of dilated bronchi had quite a reticulated aspect, owing to the thickening of transverse and longitudinal fibres external to the mucous coat.[2] The bronchial dilatations are occasionally empty, though they are generally found to contain a considerable quantity of pus or muco-pus,—this being often thin, but at other times thick, tenacious, or even semi-solid in consistence, owing to partial inspissation. This fluid may be blood-stained, and it has often a peculiarly stale, disagreeable odor, amounting in some instances even to fetidity.

In two cases there were emphysematous bullæ observed on some parts of the surface of the diseased lung; in one they were situated on the upper lobe, and in the other, dilatations the size of a nut skirted the anterior border of the lower lobe.

Modifications in the state of the pulmonary artery of the diseased lung have been noted in five cases. In one its branches were said to be simply dilated, whilst in another case, observed by Dr. Wilks, he thus describes its condition: "The pulmonary artery was very much diseased. It was, in the first place, considerably dilated, the branches throughout the tissue being much larger than natural. The coats of the vessel were also very much thickened, and the whole under surface was covered with atheromatous deposit. The vessel, in fact, very much resembled a diseased aorta. Some of the smaller branches were entirely obstructed by ante-mortem coagula, as were also some of the pulmonary veins. In the main pulmonary vessel there was a layer of fibrine closely adherent to the wall, and with difficulty separable." Of the remaining three cases, in one the pulmonary artery was *contracted* to about the size of the coronary artery, whilst within it was a mass of fibrine which occupied the entire course of the artery even to its smaller branches, and at the same time was continuous with an adherent fibrinous mass in the right ventricle; in another the pulmonary artery "seemed to be quite contracted;" and in the last the vessel is not stated to have undergone contraction, but to have been completely filled with firm laminated colorless fibrine which adhered to its walls.

In four cases the bronchial glands were enlarged and had become more or less indurated from a fibroid infiltration of their texture.

In only four out of the thirty cases I have tabulated was there any fibroid induration of the opposite lung, and, except in one of these cases, it was very small in amount, forming only two or three patches. In almost every case the lung of the opposite side was enlarged, and sometimes to a very considerable extent, it being mostly soft and crepitant throughout, and occasionally emphysematous. In many cases it extended as far as and beyond the opposite border of the sternum,

[1] These cases, in fact, seem to me to belong just as much to the subject of "Bronchiectasis" under which they were described by Barth) as to that of "Cirrhosis." I have included them here simply because they serve to show the intimate and natural relationship that occasionally exists between the two diseases.

[2] After describing the lining membrane as smooth or granular, generally of a dark-red color, and as almost invariably thickened, Barth adds: "Mais ce qui les distingue particulièrement, ce sont des espèces de stries irrégulièrement circulaires qui apparaissent plus ou moins distinctement sous la membrane interne, laquelle se continue manifestement avec la membrane muqueuse des conduits aëriens."—Loc. cit. p. 511.

and in one case where its development was most extreme, it was just double its natural size, and seemed to fill almost the whole thoracic cavity.[1] In three cases only, as previously stated, was any "tubercle" found in the non-cirrhosed lung, and in these it was small in quantity. In one there was a "tubercular" cavity in the apex, about the size of a walnut, filled with a coagulum of blood, death having been produced by a severe hæmoptysis; in another "a few tubercles existed," and in the third a few "gray granulations" were said to have been scattered throughout the lung. As a rule, the only morbid characters belonging to the enlarged lung were those characteristic of the acute bronchitis, complicated with more or less of recent pneumonia—conditions which had been the immediate cause of the patient's death.

In nearly all the cases where the contraction of the lung had been great there was a proportionate traction of the heart out of its normal position. Where the right lung was involved, the heart was frequently found behind the right mammary region, and its displacement seemed generally to be greater where this lung was affected than when the left organ was the seat of the disease. Cirrhosis of the left lung tends to raise the heart, and in one remarkable case reported by Dr. Law, it was found immediately under the left clavicle. In no less than ten out of the thirty cases there was more or less hypertrophy with dilatation of the right cavities of the heart, and in seven of these more or less dropsy also existed; whilst in two cases only was the heart reported to be rather small. In one case it was fatty, and in two of those in which the right ventricle was hypertrophied the left was said to be small and weak.

Pathology.—Various views have been entertained as to the pathology of this affection, to which we must allude before entering more fully into the relative importance of those having the most decided claim to recognition.

(*a*) Laennec first attracted attention to the disease, and considered it to be one of the modes in which dilatation of the bronchi occurred. He believed that chronic catarrh, giving rise to an increased secretion from the bronchial tubes, caused an accumulation of mucus within them, which led to their dilatation. The dilated bronchi, by pressure upon the surrounding lung-tissue, then led to its collapse and condensation.

(*b*) Dr. C. J. B. Williams[1] held that it was the sequence of a pleuro-pneumonia. His words were: "In pleuro-pneumonia the lung is inflamed, and at the same time compressed by an effusion in the sac of the pleura. If it remains long in this state, the smaller air-tubes and cells become obliterated by the adhesion of their sides, so that when the liquid is removed from the pleura they will not expand again with the enlargement of the chest; but the large and middle-sized bronchi are not obliterated; they bear the whole force of the inspired air, and become consequently dilated by it. This kind of dilatation is usually conjoined with contraction of the affected side. These cases, although not very uncommon, were first noted by the writer." And in a note to a subsequent work he said: "Dr. Corrigan has since described cases which appear to be similar, although he has given the disease the name *Cirrhosis* of the Lung."

(*c*) Sir D. Corrigan maintained that the obliteration of the air-cells and condensation of tissue were primary, and were owing to the growth throughout the organ of a fibre-tissue similar to that existing in cirrhosis of the liver. The dilatation of the bronchi was a secondary effect, due partly to the greater stress of the inspiratory force, and partly to the traction, in different directions, exercised upon the tubes by the contracting fibre-tissue.

(*d*) By M. Grisolle, M. Charcot, and others, what may be considered the early stages of this disease have been supposed to be the results of a "chronic pneumonia," or inflammation of the lung-tissue.

(*e*) Dr. Hughes Bennett seems to ignore Cirrhosis of the Lung as a substantive disease, and to maintain that all cases of so-called Cirrhosis are, in reality, instances of tubercular disease advancing towards a cure.

Laennec's theory seems to be quite inadequate to account for the production of such a disease as Cirrhosis. And with regard to the second theory—that of Dr. C. J. B. Williams—it cannot be considered to apply to the class of cases to which Sir D. Corrigan gave the name Cirrhosis of the Lung. The mode of origin of these latter, as subsequent examination has fully shown, is entirely different; the contraction of the lung being produced quite independently of the compressing effects of an effusion into the pleura.

Although Dr. Hughes Bennett is quite right in the view that there are certain cases in which a cirrhotic process is associated with "tubercle" (chronic lobular pneumonia) in the same lung, and in the opinion that this combination may, very rarely, terminate in a result differing but slightly from what may be produced by

[1] The Pathology and Diagnosis of Diseases of the Chest, 1840, p. 99: his first allusion to the affection being in "Lectures" published in the Med. Gaz. for 1838.

the pure cirrhosing process, still, what has been already said fully shows that in many cases Cirrhosis of the Lung is an independent affection, having no relation whatever to the presence of "tubercle" in the organ.

The relations of Cirrhosis to the common forms of Phthisis will be immediately considered; and also the anatomical affinities between the tissue-changes in this disease and those which characterize Tubercle. The real relationship existing between dilated bronchi and Cirrhosis, will also be carefully considered. These questions will be discussed under the following heads: 1. The Relations existing between the Cirrhosing Process and Chronic Lobular Pneumonia. 2. The Anatomical Affinities between the Early Stages of Fibroid Indurations and Tubercle. 3. The Mode of Production of Dilatation of the Bronchi, and their Relations to surrounding Induration of Tissue.

1. *The Relations existing between the Cirrhosing Process and Chronic Lobular Pneumonia.*—The evidence I have brought forward in the last section seems to show very conclusively that the cirrhosing process as it invades the lungs has no *necessary* connection with the development of "tubercle" in the same organ, whilst other considerations seem to show just as conclusively that its occurrence is not dependent upon the presence of a "tubercular diathesis." In four only, out of the thirty cases of Cirrhosis, did any morbid product, which the observer was able to call "tubercle" exist in one or other of the lungs, in company with this fibroid conversion; and even in these cases the amount of the product (which most pathologists now consider as the anatomical mark of "Chronic Lobular Pneumonias"[1]) was so slight as not to interfere with our belief that its presence was an accidental rather than a necessary element of the disease. There is nothing antagonistic between these two pathological changes,—far from it. There cannot be a doubt, however, that each may, and does, exist by itself[2] as an independent affection, although they are so frequently combined in ordinary cases of phthisis—which differ from one another principally in respect to the relative proportion, and different modes of distribution, of these two tissue-changes. In proportion to the number of times in which the two processes are met with in combination, however, it may fairly enough be considered somewhat exceptional for either of them to exist, to a fatal extent, alone.

The facts at present known seem fully to establish the independent nature of the fibroid change met with in Cirrhosis. The word Phthisis is now generally admitted to be merely a generic term, under which are included different morbid conditions of the lung, which may either exist alone or in various degrees of combination. Thus amongst other forms, there may be an almost pure pneumonic phthisis, due to the infarction of the air-cells and minute bronchi with epithelial products, the whole mass of which rapidly degenerates, and may break down into ulcerous cavities;' or a pure tubercular phthisis,[2] understanding by this a lung filled with products after the type of the gray granulation; or a pure fibroid phthisis, such as exists in Cirrhosis of the Lung.[3] On the other hand, any two of these changes, or even all three of them, may co-exist in various proportions in one or both lungs of the same individual, and thus give rise to the more common forms of phthisis.[4]

[1] In only one of the cases is there any mention made of the existence of "gray granulations," and in this case their nature is more than doubtful, since no similar granulations were found in any other organ.

[2] Occasionally, in some cases of "galloping phthisis," both lungs may at the autopsy be found thickly studded from base to apex with soft patches of "lobular pneumonia." These patches, of the size of a mustard-seed and upwards, are whitish or yellowish, breaking down here and there into minute cavities, whilst there may be a singular absence of all indurating tissue.

[1] In these cases the amount of new fibre-tissue is reduced to a minimum. Some slight amount, however, always exists, even in situations where there is no perceptible induration. The very early stages of the fibre overgrowth, when it is principally in a nuclear condition, do not produce indurations of the organs in which it occurs.

[2] If indeed such an affection is entitled to be considered as a form of phthisis, since those suffering from it usually die before destruction of lung ensues.

[3] Here, again, I do not mean that absolutely no trace of chronic lobular pneumonia exists, but rather that, in the typical cases, this is reduced to a minimum. A microscopical examination may often show a minute amount of such tissue-changes even where none is visible to the naked eye. It is almost impossible that any one portion of lung-tissue should overgrow to a considerable extent without entailing some amount of increase in contiguous tissue elements. In some cases, however, one kind of change almost wholly predominates.

[4] Since this paper was written, Dr. Andrew Clark has proposed to range a certain number of cases of lung disease under the term "Fibroid Phthisis" (see Trans. of Clinical Soc., vol. i. p. 174), with the understanding that they differ from what he terms "common cirrhosis." After a careful study of his very able report, I entirely fail to see any good reason for separating the case which he records from those which are here ranged under

When an extensive process of fibroid overgrowth is set up in a lung, around or intermixed with patches of lobular pneumonia, this tissue-change may invade not only previously healthy portions of lung, but also those which are filled with the old pneumonic accumulations, so that, at a later stage, portions of tissue previously widely dissimilar, may become almost indistinguishable from one another.[1] And in this sense, so far as the two processes are associated in the same lung, we may agree with Sir D. Corrigan,[2] and with Dr. Hughes Bennett, when they maintain that the process of Cirrhosis has a curative agency in many cases of phthisis.

2. *The Anatomical Affinities between the Early Stages of Fibroid Indurations and Tubercle.*—The process of fibroid substitution characterizing Cirrhosis of the Lung advances by two or more successive histological stages. This seems to hold good of fibroid substitution, in whatever organ it may occur—whether arising in the brain or spinal cord, in the kidney, in the liver, or in the lung. In all these situations it appears to commence by an excessive growth and the multiplication of nuclei in the part affected. These nuclei[3] are not necessarily fusiform, but are far more frequently round or oval, about $\frac{1}{4000}''$ in diameter, containing no distinct nucleolus, but only a few granules. They are interspersed with a few fine fibres so as to form a fibro-nuclear stroma. These are the anatomical characters of the first stage of fibroid substitution, and though, even at this early stage, the nuclear tissue may have supplanted the proper elements of the organ in some parts, this as a whole is not found to have undergone any contraction or diminution in bulk.[4] But gradually the nuclei disappear, and where the change is older, actual fibre tissue becomes more and more apparent. As this is developed contraction in bulk commences, and induration of the organ becomes more and more distinct. In the lungs this nuclear overgrowth seems to commence either in the connective tissue which enters into the formation of the walls of the bronchi and of the bloodvessels, in that lying between the larger and smaller lobules of the lung, or in that on the inner surface of the pleura. Or, on the other hand, it seems quite possible that the new growth may originate in a hyperplasia of certain masses of adenoid or lymphatic tissue in these situations, which, from the researches of Dr. Sanderson[1] would seem to be widely distributed throughout the healthy organ. From any, or all of these situations, the nuclear and fibrous growth spreads in various directions—gradually obliterating the air-cells, the bloodvessels, and the proper tissue of the organ, and substituting itself in their place. This is what occurs when fibroid indurations alone advance in a chronic manner, and, as I have already stated, the tissue changes are identically the same when induration is gradually set up round a cavern existing in a phthisical lung. Now, as I have also previously stated, this induration was originally described by Laennec as due to what he called "*gray tubercular infiltration*," though Chomel and succeeding patholo-

the head of Cirrhosis of the Lung. It seems to have been an instance of Cirrhosis in which cheesy patches of lobular pneumonia existed in rather larger quantity than in any of those which I have brought together. What more likely, however, to occur in some cases than such a combination? Its association with a distinct constitutional tendency I cannot help considering to be more than doubtful. (See p. 294.)

[1] This subject will be again alluded to in the section on Etiology.

[2] Dub. Hosp. Gaz., Dec. 15, 1857.

[3] In later stages, when some of them undergo a fatty change, the nuclei become enlarged, and assume the form of cells resembling "granulation corpuscles."

[4] I have examined a kidney which presented an excellent example of this first stage of Cirrhosis. The organ was of its natural size, only pale, with an extremely adherent capsule, and a very tough, leathery consistence. When examined microscopically, it was found to be more or less pervaded throughout with a nuclear overgrowth, such as I have described, though in some parts this was replaced by a more decided fibre-issue.

[1] See "Eleventh Report of the Medical Officer of the Privy Council," 1868. The too extensive use of the terms "adenoid" or "lymphatic" tissue, seems to me undesirable. Even if it be true that in all or some cases the morbid tissues of which we are speaking take their origin as hyperplasias of real though microscopic nodules of lymphatic tissue, still, in a very large number of cases, the new tissue soon loses these characters altogether, and becomes an unmistakable fibroid growth. What was "adenoid" or "lymphatic" tissue, thus gives place in a short time to a simple fibroid tissue, to which the former names are no longer applicable. There is, however, another inconvenience of even graver import. New views are being advanced concerning tubercle, of such a kind that, after a time, those who consistently adopt them will be compelled to look upon all chronic indurations as "tubercular." Cirrhosis of the liver will, in fact, become a tubercular affection. This result can only be avoided by the recognition of the non-specific nature of the new growths which may be artificially induced in the rodent animals. If we cease to call this new growth Tubercle, science will have lost nothing; if we persist, another almost hopeless confusion will be introduced into pathology. (See a paper by Dr. Sanderson, entitled, "Recent Researches on Tuberculosis," in Edin. Med. Journ. 1869, p. 387.)

gists denied its tubercular nature, and considered it to be a non-specific result of chronic inflammation.

From a histological point of view, however, there is now much more to be said in support of this nomenclature than was admitted by many of Laennec's successors; though their inability to perceive the relationship is not to be wondered at, seeing that though this has only come out strongly since the date of the renunciation of many of Laennec's views as to the constitution and nature of "tubercle," and since pathologists have begun to recognize the fact that, if the word "tubercle" is to be preserved[1] at all, the gray granulations of Acute Tuberculosis must be considered as its type. This alone of all the morbid products which have been so named has a definite constitution in whatever organ of the body it may be encountered; whilst the so-called "crude tubercle," and cheesy products generally, may have had the most diverse origin in different cases, and are always nothing but the dead and impacted remains of various secretions and tissues. An examination of very thin transverse sections of gray granulations in the lungs, brain, liver, kidneys, and other organs, suffices to convince one that its structure—closely allied to that of lymphatic tissue—is always that of a small fibro-nuclear tumor infiltrating and supplanting the normal tissues of the part in which it is found. But, further, it seems to me that the structure of tubercle is almost indistinguishable from that of the tissue existing in the first stage of that state which I have just been describing—hitherto known by the various names of "gray tubercular infiltration," "chronic pneumonia," or "fibroid degeneration." There are in each case the same round and oval nuclei or embryo-cells, imbedded in a fine and somewhat scanty fibrous stroma. This resemblance only exists, however, between one temporary stage of the process of fibroid substitution, and the gray granulation.[2] Tubercle seems to be the mark or index of a general constitutional disease, and how long the gray granulation may remain as such, or what may be the degree of frequency with which it undergoes changes, are questions to which we are unable to give very satisfactory answers. Although fibroid induration may, on the other hand, owe its origin partly to a constitutional cause, it seems much more dependent upon special local conditions operating in the organ or part in which it is set up; then again, it exists not only in minute patches, but spreads over considerable areas, and advances through stages of development which are well known and pretty constant.[1]

Where the process of fibroid substitution is advancing in a lung, there appears to be not only an increased growth of the connective tissue and lymphatic elements, but also a rapid formation of epithelial products, as evinced by the number of cast-off and fattily degenerated cells of this kind which are seen within the air-vesicles. These are always to be seen in places where the fibro-nuclear growth has not completely invaded the tissue, though after a time they appear to be stifled, and stamped out as it were, by the superior energy in growth of the advancing fibre-tissue. This, in fact, appears to be the rule in pathological conditions of the lung, that a morbid change is rarely or

[1] For my own part, I think that pathological science would gain much if this word, and all the erroneous associations, as to specificity, which its use seems inevitably to entail, could be entirely forgotten, save as errors of the past. Old things might receive new names, and thus, at last, old theories might possibly be shelved.

[2] It is interesting to find that, nearly forty years ago, Andral seems to have anticipated, in a measure, the results of recent microscopical research, since he fully recognized that the gray granulation was quite distinct from other kinds of what was then called "tubercle," and was closely allied rather to the forms of pulmonary induration which we now know to be of fibroid origin. His words were (Précis d'Anat. Patholog. 1829, t. ii. p. 518): "Les granulations pulmonaires de Bayle ne sont ni un tissu accidentel *sui generis*, comme il le pensait, ni le premier dégré du tubercle comme l'ont admis MM. Laennec et Louis, mais qu'elles consistent dans l'induration de quelques vésicules. . . . . Or, ce qui arrive à un lobe dans sa totalité peut aussi arriver à quelques vésicules; la lésion est seulement moins étendue; mais du reste, sa nature est la même."

[1] It seems the so-called "artificial tubercle" in the rodent animals whose anatomical characters have now been fully revealed by the admirable researches of Dr. Sanderson, Dr. Wilson Fox, and others, is less allied to tubercle (as occurring in acute tuberculosis) than to some more local manifestation, such as that which characterizes "tubercular peritonitis." All these morbid products are, however, as I think, more akin to those of acute cirrhosis. In acute tuberculosis, as it occurs in the human subject, the gray granulations appear to develop almost if not quite simultaneously in meninges, lungs, liver, &c. In acute cirrhosis in the human subject there is often a slight tendency to extension of the process to other organs, and this tendency becomes more marked and constant in the rodent animals, though the spread to other organs is distinctly successive, and seems to take place by actual local contaminations. The frequency of cheesy degenerations in the infiltrating patches of "artificial tubercle" is probably referable in the main to their rapid growth, and the instability of tissue elements which this usually entails.

never absolutely restricted to one tissue. The change originates and is predominant in one, whilst it extends to and may be only more or less slightly developed in the other. The nutrition of the organ, or of parts of it, may be generally deranged, but the stress of the disorder falls in one case principally upon the vascular province of the pulmonary artery, and in another upon that of the bronchial arteries: thus a bronchial or a catarrhal pneumonia may be associated with a certain amount of fibroid induration, and an advancing fibroid change is often mixed up with an increased growth and shedding of epithelial elements from the mucous membrane.

Such being the anatomical nature and mutual relations of these various tissue changes, in what light should we regard the one with which we are now concerned —that which has been spoken of successively under the names of gray tubercular infiltration, chronic pneumonia, and fibroid induration or degeneration? That it is tubercular, or in any way an essential appanage of the tubercular diathesis, may, I think, at once be dismissed from consideration, as there is no evidence to support this view.[1] Is it then an inflammatory change, or one partaking rather of the nature of a degeneration? To Dr. Handfield Jones the merit is due of having first fully pointed out[1] the essential similarity of these indurating processes in various organs of the body (all of which had been previously spoken of as effects of "chronic inflammation"), of having shown that in all alike the essential nature of the change is an hyperplasia or overgrowth of the connective tissue of the part, and for ably insisting that the process by which this was brought about was one totally distinct from what is ordinarily understood by the word inflammation. He held that they were effected, in fact, by a process substantially different —by one which was slow and chronic from the first, and which partook rather of the nature of the process by which an infiltrating new growth spreads.[2] It seems to me, also, that the word inflammation is quite inapplicable to the changes by which these effects are brought about. In inflammation we almost invariably find an accelerated formative process resulting in the production of elements of an unstable composition; such as quickly degenerate and decay—a process of necrobiosis or destruction in fact goes on simultaneously with one of formative increase—whilst in the process which results in the production of fibroid indurations, there is principally an increased formative stimulus by which an overgrowth of connective tissue or lymphatic elements takes place. The necrobiotic process, however, is almost entirely wanting, since the new-formed elements persist as a developing fibroid growth. Thus, whilst the change differs materially from inflammation, so also does it differ from a degeneration. The proper tissues of the part are not merely degenerated and structurally spoiled, they are actually killed, and disappear before a new fibro-nuclear tissue which supplants them. So that we have the increased formative energy of an inflammatory process without its unstable products; and we have the functional degradation characteristic of a degeneration—though this results not from mere spoiling of texture, but rather from the complete substitution of a tissue of a lower grade in the place of that which is proper to the part. Surely in this fibroid hyperplasia, or *fibroid substitution*, as I think we should term it, we have a process strictly intermediate in kind between inflammation on the one hand, and de-

[1] When the above passage was written, I could speak thus confidently; now, however, since the experimental researches of Dr. Wilson Fox, Dr. Sanderson, and others, upon the "Artificial Production of Tubercle," pathological doctrines show signs of undergoing some modification. In the article before referred to, in the Edin. Med. Journ. 1869, Dr. Sanderson's view is most clearly stated. It comes out in this form: "Tubercles are adenoid bodies enlarged: . . . the disease progresses, not by continuous growth, but by the distribution or dispersion of infective material from one point." For the development of "consumption" in man, three things are necessary: 1. A constitutional tendency; 2. A local irritation; and 3. A process of *infection*. Referring to the latter, Dr. Sanderson says: "The word designates the fact that wherever a chronic induration, *due to overcrowded corpusculation*, exists in any organ, it is apt to give rise to similar processes elsewhere." Dr. Sanderson would apply these views even to the mode of extension of "the so-called infiltrated forms of induration" met with in ordinary cases of phthisis; and he would, of course, be compelled to apply it to infiltrating indurations (of cirrhosis processes) generally, because they are almost always characterized by an "overcrowded corpusculation" in the part. Thus the present tendency, with some pathologists, is to consider that *all* infiltrating fibroid indurations may increase by a process of infection, and the logical outcome of their doctrines is the belief that such indurations are tubercular in nature. The chronic inflammations of many writers would thus be transmuted into "tubercular" affections, and the simple nuclear hyperplasia which characterize them in their early stages would be even more likely to be considered as a new "specific product," if it is to receive the name of "adenoid" tissue.

[1] Brit. and For. Rev. 1854. This, as we have seen, is also the opinion which was subsequently expressed by Dr. Wilks.

[2] Loc. cit. p. 345.

generation on the other—it is a sort of neutral ground from which the other two processes may be considered as divergences in opposite directions.[1]

3. *The Mode of Production of Dilatation of the Bronchi, and their Relations to surrounding Indurations of Tissue.*—The opinions expressed as to the mechanism of dilatation of the bronchi have been most various since the subject was first introduced by Laennec. His theory was, that bronchial dilatation was one of the effects of chronic bronchial catarrh—that it was brought about by the accumulation and stagnation of mucus in the inflamed tubes, and that the dilatations, by the pressure they exercised, led to the collapse and consolidation of the surrounding lung tissue. Andral's views[2] were also somewhat unsatisfactory. He recognized three forms of dilatation: one species, with thin walls, he believed was produced after the manner stated by Laennec, whilst two others he attributes to hypertrophy of the bronchial walls, though he does not explain how the modification in texture is to bring about the alteration in calibre of the tubes. Dr. Stokes[3] believed bronchitis to be in all cases the primary cause of the dilatations, inasmuch as this leads to loss of elasticity in the longitudinal contractile fibres of the bronchi, and also to paralysis of the circular muscular fibres. He thought also that the epithelial ciliary action ceased, and thus permitted the accumulation of mucus, which (in conjunction with the other causes mentioned) tended to bring about a dilatation of the tubes, under the straining influence of forced inspirations, during repeated attacks of coughing. Dr. C. J. B. Williams[4] also laid great stress upon the influence of inflammation in bringing about alterations in the texture of the tubes, by which their elasticity and power of resistance was impaired—so that they more easily yielded to pressure during the act of coughing. This was his theory as to the mode of production of the ordinary forms of bronchial dilatation—those which exist without great induration of the surrounding lung texture. Where extreme induration was also present, however, he gave the explanation which has been quoted at the commencement of this section. (See p. 285.)

[1] The phrase "fibroid substitution" will not be applicable to all instances of the kind of change alluded to, since, where it occurs in some of the fibrous membranes, such as the arachnoid, there is no substitution, but only an increase or hyperplasia of the part.

[2] Précis d'Anat. Patholog. tome ii. p. 496.

[3] Diagnosis and Treatment of Diseases of Chest. Dublin, 1837.

[4] Pathol. and Diagn. of Diseases of the Chest, 1840, p. 96.

Very shortly afterwards Sir D. Corrigan[1] published his explanation of the production of bronchial dilatation, as met with in the class of cases to which he gave the name of Cirrhosis of the Lung. This must be given in his own words. He says: "The dilatation of the bronchial tubes is partly owing to the contractile process going on in the tissue of the lung—partly to the expansive action of the parietes of the chest in the act of inspiration. . . . If there were but one bronchial tube with contracting fibro-cellular tissue placed around it, then the contracting tissue would, as in the instance of stricture of the œsophagus or rectum, cause narrowing of the tube; but when there is, as in the lung, a number of bronchial tubes, and the contracting tissue not placed around the tubes, but occupying the intervals between the tubes, then the slow contraction of this tissue will tend to draw the parietes of one tube towards the parietes of another, and necessarily will dilate them." He also says: "In proportion as the contraction of the fibro-cellular tissue obliterates the small air-vesicles, and as these contracting fibres, like so many strings, extending from the root in all directions, tend to contract or draw in the tissue of the lung, obliterating its small air-tubes and its bloodvessels, the larger bronchial tubes dilate to supply the place thus left, until, when the disease has reached its last stage, the tissue of the lung, diminished to a very small size, presents no longer any permeable air-vesicles, but a dense fibro-cellular or fibro-cartilaginous tissue with its fibres radiating in every direction, through the second and third sized bronchial tubes dilated into cells, or ending in *culs de sac*, of every variety of size."

Rokitansky[2] adopted Dr. Stokes's view as to the mode of production of the uncomplicated form of bronchial dilatation: he believes it to be a result of obstructive bronchitis in the ramifications of the bronchi beyond those which become dilated. "It is produced," he says, "by the hindrance which is presented to the free ingress of the inspired air, and is proportional to the difficulty of breathing and the prolonged length of each individual inspiration, and is especially developed in and about the perfectly impermeable bronchial tubes. The parenchyma surrounding this portion of the bronchial system collapses, and this produces a space which becomes filled by the dilating bronchus. The dilatation thus lies entirely, or for the most part, in a collapsed, and apparently compressed, portion of the parenchyma; hence the latter appears to be the primary anomaly,

[1] Loc. cit. p. 270.

[2] Pathol. Anat. (Syd. Soc. Trans.).

and the bronchial dilatation merely a *resulting* and consecutive morbid change." The opinions expressed by Dr. Gairdner[1] were very different, and are as follows: "The conclusion to which I have been led by this survey is, that almost all the so-called bronchial dilatations, and all those presenting the abrupt sacculated character here referred to are in fact the result of ulcerative excavations of the lung communicating with the bronchi." He then adds: "The usual origin of bronchial dilatations is in cavities formed in atrophied lungs, in consequence of bronchitis or tubercle, and afterwards expanded beyond their original dimensions by the inspiratory force." Dr. Peacock[2] thinks Sir D. Corrigan's views unsatisfactory, but he says, in reference to the views of Dr. Williams and Dr. Gairdner: "I believe both to be correct in some cases, and that by one or other of the modes mentioned by these writers all the various forms of so-called dilatation of the bronchial tubes which are observed may be explained." M. Barth[3] believes to a certain extent in the views advanced by Stokes, and also partly in those of Corrigan—to the effect that condensation of tissues usually precedes the bronchiectasis. He also attributes an influence to firm pleuritic adhesions when combined with a shrinking of lung-tissue, and to the pressure exercised by retained and heated air which has been forcibly drawn, through accumulated mucus, into certain bronchi.

Lebert[4] agrees, in the main, with Stokes, though he thinks the weakness of the bronchial walls is ultimately dependent rather upon a disturbance in their innervation than upon an inflammatory state. Quite recently Dr. Grainger Stewart[5] has objected to the theory of Stokes, urging that if bronchiectasis depended simply on bronchitis, it would necessarily be much more frequent than it is. He thinks that Lebert's doctrine is the only one which is not opposed to known facts, and draws the following conclusions from his own observations: "1. That the essential element of bronchiectasis is atrophy of the bronchial wall, that the cause of such atrophy is not yet ascertained, but may perhaps be connected with constitutional peculiarities. 2. That the walls being so thinned and weakened, readily yield to the pressure of air, it may be in deep and sudden inspirations or during violent muscular exertions, certainly in the sudden expiratory effort made while the glottis is closed in the act of coughing. 3. The enfeebled and dilated condition of the bronchi favors the accumulation of the mucus secreted by the bronchial membrane. 4. That the mucus accumulating and undergoing decomposition in the dilatations, irritates the mucous membrane, leads to inflammation, and the formation of villous processes from it, to the formation of increased connective tissue in the walls, to irritation of the cartilages, and frequently to consolidation of the surrounding lung-tissue and pleuritic adhesions, sometimes also to abscess or to limited gangrene." With regard to the primary atrophic change which takes place in the walls of the bronchi, Dr. Stewart says that this is obvious even in the slighter dilatations, in which the mucous membrane is as yet unaffected, and that the atrophy shows itself in the muscular and elastic fibres, which appear granular and indistinct.

Such are the various opinions that have been expressed concerning the mechanism of bronchiectasis, and the relations of this pathological condition to surrounding induration of lung-tissue; and one cannot help being struck with the very opposite views which certain of the writers take as to the interdependence of these two states. This very diversity of opinion, however, seems to indicate that condensation or induration of lung-tissue cannot in all cases be considered as a necessary prelude of bronchiectasis. Those who have formed this opinion must have arrived at their conclusion from an examination of a limited class of cases, since it is a well-known and admitted fact to those who have studied the subject more widely, that in certain cases dilatation of the bronchi exists with scarcely any appreciable alteration of the surrounding lung-tissue. But whilst in some cases it seems certain that adjacent induration either does not exist, or is present to such a limited extent as to be altogether unimportant in an etiological point of view (even if, in these cases, it has not been mechanically produced by the very dilatation with which it coexists), it seems also just as evident that, in a certain class of cases, the bronchial dilatation is to be looked upon as a secondary consequence of induration and contraction of lung-tissue. What the precise means are by which the dilatation is brought about in these cases, we shall consider presently; but that the existence of a disease of the lung-tissue, which entails contraction, is favorable to the occurrence of bronchial dilatation, may be seen, I think, from the facts before mentioned,—to the effect that nineteen or nearly two-thirds of the thirty cases of Cirrhosis I have analyzed, occurred in individuals between the ages of fifteen and forty years, and that, out of these nineteen cases, eleven presented

[1] Monthly Journal of Medicine, vol. xiii. 1851, pp. 248, 249.
[2] Ibid., April, 1855, p. 285.
[3] Loc. cit. p. 517.
[4] Anat. Patholog. tome i. p. 620.
[5] Edinburgh Monthly Journal, 1866.

well-marked dilatation of the bronchi; whilst in forty-three cases of dilatation of the bronchi collected by Barth, only seven —or less than one-sixth of the total number—were met with between these ages, though more than one-half (26 : 43) were in individuals over sixty years of age. The occurrence of Cirrhosis of the Lung, therefore, seems to be favorable to the production of bronchiectasis at such ages when dilatation of the bronchi alone, or as a primary phenomenon, is not prone to occur.

With reference to the occurrence of bronchiectasis in lungs which are not contracted, and have no consolidation of tissue in them, it seems to me that if a primary atrophy of the bronchial walls exists like that which Dr. Grainger Stewart has observed, the order or succession of the phenomena would probably be such as he describes. This mode of origin, also, seems to be the only one capable of accounting for such cases of bronchiectasis as have been met with unexpectedly, in individuals who have not had any long-continued cough or bronchitis: it may, moreover, obtain in the first instance, and be the determining cause of the dilatation in a certain number of those persons who have previously suffered from bronchitis. By reference to such a mode of origin only, does it seem possible to explain some of the anatomical characters of dilated bronchi, such as the occurrence of bridge-like portions of prominent and unatrophied tissue, and the occasional communication between the dilated portions of contiguous tubes. But it seems equally plain that it is not necessary for us, in all cases, to assume the existence of such an atrophy, when we recollect in what a large proportion of cases the individuals in whom bronchiectasis has been met with have suffered from chronic bronchitis and long-continued cough. To explain the occurrence of dilated bronchi in many of these cases, we have only to refer to the views of Dr. Williams and Dr. Stokes, before alluded to; and I would also add, that one important kind of alteration in the walls of the bronchi, induced by chronic inflammation, is the production of a certain amount of fibroid substitution. Then, as in most of the cases of dilatation of portions of the vascular system, more or less of the muscular and elastic tissue of the tubes is replaced by ordinary distensible, though comparatively unelastic, fibrous tissue.[1] A tube thus altered, having once yielded under a powerful inspiratory effort,—or more especially under the powerful expiratory effort, with closed glottis, preceding the act of coughing,—does not regain its normal calibre, and each increment of dilatation successively brought about remains as a persistent abnormality. In those instances of what may be called acute dilatation of the bronchi, met with after attacks of hooping-cough, the inflammatory changes in the walls of the tubes, combined with the powerful inspiratory and expiratory efforts, seem to be the conditions which are most instrumental in bringing about this effect. Then again, the modes of origin suggested by Dr. Gairdner must not be forgotten. There seems every reason to believe that many of the abruptly sacculated cavities which have been described as bronchial dilatations, have really had an ulcerative origin, though their walls may have become perfectly smooth. Cavities thus formed may subsequently be increased in volume by the same means as those which usually suffice to augment the size of the more simple bronchial sacculi.

Although in a certain number of cases little or no alteration of the lung-tissue around the dilatations exists, in many others more or less condensation is met with. This is oftentimes merely a collapse of the adjacent textures, brought about by the pressure of the dilating bronchus; whilst, in other instances, there is an actual induration of tissue, which must be regarded as a consequence of the primarily existing bronchial dilatation. Dr. Grainger Stewart has suggested what may be considered to be a real and feasible explanation of this secondary induration in his fourth conclusion, where he says that influences which suffice to irritate the bronchial wall must, if continuously or intensely applied, affect the structures lying beyond them. In one case, around the dilated bronchi, he found the lung-tissue indurated and pneumonic; and in another case, around cavities which were livid with reddened and inflamed mucous membrane, the lung-substance was consolidated. On microscopical examination of this consolidated lung-tissue, "little trace of air-cells could be made out, and it was mostly composed of fibrous tissue." In other rare cases, the irritation manifests itself in the formation of an abscess, in the centre of which the dilated bronchus is seen; or even—as first pointed out by M. Briquet,[1]—in the establishment

[1] Dr. Stewart says that many of the dilated bronchial tubes present an appearance simulating hypertrophy of their walls, but which is really dependent upon changes in the mucous membrane, by which it becomes granular or villous, and upon the presence of ill-formed connective tissue among the denser elements of the bronchial walls. He adds, "The irritation which causes the inflammatory thickening of the mucous coat may well also account for the spurious hypertrophy of the other."

[1] Archives Générales de Médecine. 1841.

of a limited gangrenous inflammation, involving the walls of the dilated bronchus and the surrounding lung-tissue.

In other instances, where the bronchiectasis is primary, instead of the intervening lung-tissue remaining unaltered, being simply compressed, or undergoing either of the secondary changes just mentioned, it gradually disappears—seemingly as a result of atrophy and slow absorption—so that, in extreme cases, absolutely no intervening tissue may be left between the dilated tubes of the greater part of one lobe of a lung.[1]

We must now come to a consideration of the mode in which dilatation of the bronchi is brought about in Cirrhosis—that is to say, in those cases where induration and contraction of the lung-tissue is the primary occurrence, and where dilatation of the bronchi is an altogether secondary phenomenon, which may occur or may not, according to the presence or absence of other occasional accompaniments of the disease. An analysis of the thirty cases I have tabulated seems to show that dilatation of the bronchi in this disease is of a compensative character, owing to the fact of its being generally most marked in those contracted lungs where the space which would have been left by contraction is not otherwise filled up—either by inshrinking of the thoracic parietes, by elevation of the corresponding half of the diaphragm with proportionate displacement of abdominal organs, or by hypertrophy of the opposite lung and its extension into the diseased side of the thorax. If the space which would have been left by the shrinking lung is not otherwise filled up, then the increased pressure of the inspired air, acting upon bronchi in whose walls more or less fibroid substitution has most likely occurred, tends to dilate some of those which are most favorably situated for undergoing this expansion. It is obvious that something must go towards filling up the space left by the shrinking lung; and if the thoracic parietes are so firm as not to yield easily, or if displacement of the viscera does not take place, then the bronchi must yield and dilate in some of their weakest parts under the continually increasing pressure of the inspired air. It is, however, in great part a mechanical question. In a case where the proper texture of the tubes has become weakened by inflammation or fibroid changes, and where other conditions are favorable, a dilatation may be brought about; whilst in another case, where the lung is equally affected, dilatation of the bronchi may not occur, because, in this particular instance, it may be easier for displacement of viscera or inshrinking of the thoracic parietes to occur in its stead. Of course, this dilatation need not necessarily be situated—and in fact would be less prone to occur—in parts of the lung which had already undergone an extreme amount of induration. So long as the dilatation existed in some part of the organ, the particular region in which it occurred would be altogether immaterial. The weakest part, other things being equal, would most readily undergo dilatation. How far the contractile influence of the fibre-tissue itself may, as suggested by Sir D. Corrigan, directly tend to bring about the dilatation of the bronchi, or be a real cause of their enlargement after a certain amount of dilatation has once been established, seems doubtful. I certainly do not think, however, that this is one of the principal causes of the production of bronchiectasis. If it were really the method by which dilatations of the bronchi had been produced, it might reasonably be expected that they should be most marked precisely in those parts of the lung which had undergone the most notable contraction and condensation. Such a distribution is, however, by no means invariable, and often the arrangement met with is quite the reverse. Taking the view of the case I have proposed, it will be seen that in those instances where dilatation of the bronchi did not exist, or was only very slightly marked, this was explicable from a consideration of other coexisting conditions observed *post mortem*. Thus, in two of the cases in which there was no bronchiectasis, the lung affection was comparatively acute and recent, and no shrinking of the organ had as yet taken place; in the next the amount of lung shrinking was probably not great, as no note was made of its existence: in another the lung was described as "small and solid" on the right side, but then the liver was very large, and the right side of the chest was also flattened; in another the disease was restricted to the lower lobe of one lung on the right side, but then this was universally adherent to the diaphragm, and the upper lobe of the same lung was notably emphysematous; whilst in the last, although the right lung was as small as a closed hand, there was flattening beneath the clavicle, and the right side of the thorax contained a large and displaced heart, in addition to nearly one quart of pleuritic fluid.[1] Of those cases in which the dilatation of bronchi was only slightly marked. in one the diseased lung was universally adherent, and its

[1] There is a good example of this in Guy's Hosp. Museum, 1718[51]. See also Path. Trans. vol. xii. p. 78.

[1] This was the only one of thirty cases, however, in which any pleural fluid was found, or in which there had been any reason to suspect its previous existence.

amount of shrinking was probably not extreme, since it was not specified, whilst there was a most remarkable dilatation of the pulmonary artery throughout the organ; in another, the disease being on the right side, there seemed to have been a falling in of the lower part of the thoracic parietes, whilst the heart was situated entirely in the right side of the thorax, and the enlarged left lung extended under the sternum and partly into the right side; in another, although the amount of contraction of the lung was extreme (the disease being of six years' duration, and having commenced when the boy was only fourteen years old), still the left side was described as being "contracted to an extraordinary degree," both vertically and horizontally; in the last the reason why there was only slight dilatation of the bronchial tubes is not quite so evident, though some of the points which might have explained it have not been distinctly alluded to. Concurrent evidence of this kind strongly tends to support the view now advanced concerning the method of production of the bronchiectasis which may occur in the course of Cirrhosis.

From these considerations as to the mode of production of bronchiectasis generally, and its relation to different states of the surrounding lung tissue, we may venture to draw the following conclusions:—

1. That dilatation of the bronchi may be present, and take place quite independently of alterations in density of the surrounding lung-texture; although such dilatation may be favored by a primary atrophy of the walls of the bronchial tubes, or by the effects of inflammation in weakening them and diminishing their natural elasticity, or by a combination of the two. The actual mode of production, even when these favoring conditions exist, being always the expanding force of powerful inspirations, and more especially the tension occasioned by the expiratory effort, with closed glottis, which immediately precedes the expiratory part of the act of coughing.

2. That in these cases of *primary* bronchiectasis the intervening lung-tissue may be found almost natural, or compressed and airless, though it may subsequently become so far irritated as to be found in a condition of inflammation, of fibroid induration, of purulent softening, or even of gangrene.

3. That in certain other cases the bronchiectasis is compensative, and seems to be *secondary* to a certain amount of collapse of lung-tissue, though its actual production is still aided by the effects of cough and inflammation; or, as in so many of the instances of Cirrhosis of the Lung, the bronchiectasis is secondary to an actual shrinking with fibroid consolidation of the lung-texture—when dilatation of some of the bronchi results, as a physical necessity, if displacement of viscera or inshrinking of thoracic parietes cannot be so easily brought about.

Etiology.—Are we to look upon Cirrhosis of the Lung as a constitutional affection or as one of a strictly local nature? If constitutional, we should have to regard it as one of the local manifestations of a general diathetic condition upon which fibroid degeneration of organs and tissue seems in some cases to depend.[1] The question of the existence or not of such a diathetic condition has been ably discussed by Dr. Handfield Jones,[2] who has shown that not unfrequently we meet with wide-spread degenerations of this kind existing in various organs of the body, which it seems only possible to explain by the assumption of the existence of some particular condition of the blood or diathetic state, favorable to the occurrence of such anomalies of nutrition in many parts of the same organism. Thus, coinciding with a cirrhosis of the liver, we may find a similar condition more or less developed in the kidney, together with opaque thickenings of the capsule of the spleen, fibroid thickenings of the cardiac valves, fibroid degeneration of the parts of the arterial system, opaque thickenings of the arachnoid, &c. Do we in these cases meet with similar changes in the lungs, and is Cirrhosis of this organ to be looked upon as a sequence of the diathetic condition in question? To the first inquiry our answer must certainly be in the affirmative. Fibroid thickening and induration of parts of the lungs is frequently met with in association with similar changes in other parts of the body, as the tables of Dr. Sutton[3] fully prove. An answer to the second question is, however, not quite so easy.

Although in a certain number of cases, and more especially in elderly persons, disseminated fibroid indurations are to be met with, still, in other cases, a notable amount of fibroid substitution may have taken place in one or other organ alone. This latter state of things, so far as I have seen, is more apt to occur in individuals who have not yet passed the

[1] In the paper before alluded to, Dr. Andrew Clark has, since this was written, strongly urged that his cases of "Fibroid Phthisis" are local manifestations of a diathetic condition, characterized by the dissemination of waxy degenerations and fibroid indurations in different organs of the body.

[2] "Fibroid and Allied Degeneration," Brit. and For. Med.-Chir. Rev. 1854.

[3] Med.-Chir. Trans. vol. xlviii.

meridian of life. But fully two-thirds, or perhaps more, of the cases of Cirrhosis of the Lung, are met with in individuals under forty years of age. Moreover, an examination of the post-mortem records of the thirty cases which I have tabulated, lends little or no support to the idea that the induration and shrinking of the lung has been only one manifestation of a general diathetic condition entailing similar changes in other organs. Again, it may be seen from a consideration of the pathology of that form of bronchitis which is set up by the continued inhalation of foreign particles, that a similar fibroid change may be initiated in the lungs, without the agency of any diathetic condition. By the powerful action of a local irritation only, such changes are set up and may be seen in association with the chronic bronchitis of miners and artisans. In these cases the determining cause acts upon both lungs, and the effects are seen in both. Not so, however, with the ordinary cases of Cirrhosis of the Lung: here the fibroid induration, when existing to the marked extent which constitutes Cirrhosis, is almost invariably unilateral (which of itself tends strongly to negative the idea of its being entirely of diathetic origin), and in only three or four out of the total number of cases does there seem to have been any well-marked coexisting fibroid substitution or hyperplasia in other organs. But we do find in more than two-thirds of the cases, old adhesions of a firm and almost cartilaginous consistence uniting the two pleural surfaces on the side affected.

Chronic bronchitis, in fact, when it occasions a dry pleurisy on one side with the gradual formation of adhesions, seems to be the most frequent determining cause of that local overgrowth of fibroid tissue which constitutes the essential feature of the disease. The new growth gradually encroaches upon and replaces the proper lung texture, till at last the whole nutrition of the organ seems to become leavened by this change, and many independent centres of transformation are established. In almost all cases, however, in which thickening of the pleura is produced, the new growth seems to spread inwards from this with greater rapidity than it does from other centres.

In a few cases chronic bronchitis alone seems to have been the determining cause, since no notable adhesions of the pleural surfaces have existed, and the invading new tissue has seemed to start, throughout the organ affected, as direct prolongations from the walls of greatly thickened bronchi.[1] Why in these latter cases the change should be limited to one lung is rather difficult to understand:[1] we can only suppose that this may be due to the unequal incidence of the irritating cause acting alone or else in combination with some obscure, though positive tendency to perpetuate a tissue-change of this kind when it has been once initiated.

But there seems to be still another way in which well-marked Cirrhosis of the Lung may occur, and that too by a process which is usually much more rapid in its progress than when the change originates in the manner I have hitherto described. I refer to those cases in which fibroid induration immediately follows an acute inflammation of the lung—the process which Grisolle, Charcot, and other writers describe as "chronic pneumonia." This is a subject surrounded with doubt and difficulty. I have already said that the name "chronic pneumonia" appears to me to be altogether unsuitable and contradictory as applied to this affection. But, apart altogether from the question of names, there are other difficulties, since —partly owing to the rarity of the occurrence—many physicians are not prepared to admit that such a pathological state is ever the immediate sequence of an acute pneumonia. Several physicians and pathologists, however,—such as Bayle, Sir John Forbes, Addison, Lebert, Grisolle, Charcot, Hughes Bennett, and others—believe in this sequence, and have recorded cases which tend most strongly to support their opinion. Bayle's[2] case of "Chronic Peripneumony, which resembled Phthisis," seems to be one of this kind. Grisolle says that, during his very long experience, he has only seen four examples of the passage of acute into chronic pneumonia. He believes that this sequence is a consequence of neglect, though it may, perhaps, depend even more upon peculiarity and debility of constitution. Huss says it is liable to occur in habitual drunkards, but Grisolle states that such does not seem to be the case in France: neither does he place any more credence in the opinion of Heschel, who, because he found that this complication was rare at Vienna but somewhat more

[1] In very many cases this induration may never reach an extreme degree, and it may affect both lungs pretty equally. It is only in rare cases that it attains an extreme degree in one lung, and so produces the condition of the organ with which we are now concerned.

[1] A somewhat similar difficulty, however, presents itself in the case of simple dilated bronchi, owing to the frequency with which this condition, in association with chronic bronchitis, is met with only in one lung. Here, we must resort principally to the supposition that there is some difference in the texture of the bronchial tubes on the two sides.

[2] Researches on Pulmonary Phthisis, translated by Barrow, 1815, p. 415.

common at Cracow, attributed it to the influence of malaria. In one of Grisolle's patients, who died at the end of the tenth week from the commencement of an acute pneumonia, which he considered to have passed over into the chronic form, the lung affected was found in the following condition: It was almost entirely hepatized; the lower lobe being hard, compact, reddish-gray, and the cut surface being smooth—though granulations appeared when portions were torn. This differed from a state of acute inflammation by the greater hardness and gray color of the part. The whole of the upper lobe was indurated, with the exception of a portion extending rather more than one inch from the summit. The anterior border, which presented the most recent traces of inflammation, was in a state of well-marked gray hepatization, the rest of the lobe being in a condition of red induration, and showing granulations on both its cut and torn surface. This granular appearance may be met with, according to Grisolle, when the malady has only been of two or three months' duration, though after this it gradually disappears. Then with regard to the case (II.) recorded by Dr. Sutton, although a previous history of inflammation of the lung was by no means distinctly made out, Dr. Sutton seems to have been quite convinced that the state of the organs was just such as has been described under the name "red induration," and it does appear quite certain that the change was one of an acute character. So that either the old interpretation must be the correct one (that this "red induration" is the immediate sequence of an acute pneumonia) or else we must accept Dr. Sutton's supposition, that it is possible for an acute fibroid change, of the kind he describes, to occur in a lung not previously diseased. But, in the face of other evidence, the first supposition seems the most probable one, and I look upon the case (III.) recorded by M. Charcot, as affording the strongest support to this view. Whilst believing, therefore, that this sequence may occur in certain cases, it must be clearly borne in mind that it is an occurrence of extreme rarity, supervening only under the influence of exceptional conditions, which as yet may be said to be almost entirely undiscovered.

In fine, then, exposure to cold and wet, leading to the advent of bronchitis, pleurisy, or pneumonia, in certain individuals seems to be the principal determining cause of this disease. It is apparently much more prone to occur in males than in females, though this difference may perhaps be due more to the much greater frequency of exposure of individuals of the male sex than to any inherent inequality in liability to the disease *quâ* sex. And, although met with occasionally in children and in old people, this disease seems much more prone to occur in individuals between the ages of fifteen and forty. But what has just been said with regard to the apparent determining influence of sex, and its subordination to relative amount of exposure to wet and cold, may also hold good with regard to age, since, *cæteris paribus*, individuals between the ages I have mentioned, are more likely to be exposed in this way than persons who are either older or younger.

With regard to the supposed connection between this disease and the rheumatic diathesis, or the predisposing influence of long-continued habits of intemperance, nothing positive can be said; only the extreme rarity with which either of these circumstances has been mentioned would seem to show that neither of them can be considered as essential antecedents of the disease. No casual relationship, either, can be established between syphilis and Cirrhosis of the Lung. Neither does there seem to be any evidence to lead us to imagine that this malady is ever propagated by hereditary transmission: and of course if it be true, as I suppose, that the disease is a local one, set up for the most part in the individual by accidental conditions, this absence of any tendency to hereditary transmission is quite in accordance with what might have been expected.

Cases of Cirrhosis of the Lung.

I. M., æt. 7. Dr. Corrigan, Dublin Journ. of Med., 1838, p. 226.

General History. Influenza three months before, followed by cough and expectoration, with loss of flesh, and occasional *hæmoptysis.*—*Symptoms.* Febrile symptoms for sixteen days, with severe cough, dyspnœa, and hurried respiration. —*Inspection, Percussion, Auscultation, etc.* Right side perceptibly flattened. Bronchial respiration, and distinct bronchophony over flattened portion of chest.—*Autopsy: Right* side, slight pleuritis; lung solid, non-crepitant, grayish-red, tough, and traversed in all directions by thickened white bands of fibro-cellular tissue. Bronchi dilated towards pleura, terminating in spherical sacculi; lining membrane dark red. *Left lung*, healthy.

II. M., æt. 26. Dr. Barlow (recorded by Dr. Sutton), Med.-Chir. Trans., 1865, p. 299.

General History. A well-developed muscular man, of middle height. Always had good health, except for an occasional winter cold. Four months ago appetite began to fail and cough commenced. Pursued his work for one month, and then

gave up, owing to increasing weakness. Afterwards became weaker and weaker, the cough continuing. Three weeks before admission spat phlegm streaked with blood.—*Symptoms.* Oct. 12. Admitted into hospital. During the first ten days cough became easier, and he seemed to gain strength. Appetite variable. Oct. 21. Immediately under right clavicle scarcely any respiration heard, but distant crepitation. Posteriorly over right apex tubular breathing, with moist sounds and whispering bronchophony. Tubular breathing also at right base, with crepitation all down the left side. Oct. 25. Respirations labored, 35; pulse 140, very small and feeble. Profuse perspirations; friction sounds over right base. Died same day.—*Inspection, Percussion, Auscultation, etc.* *Heart* sounds clear and sharp; pulse small and compressible. Skin not particularly hot. Over *right* side, posteriorly, respiration feebler than over left; though, on *left* side, the percussion resonance was also diminished over the base. Vocal resonance markedly increased over right base.—*Autopsy: Right lung.* Signs of recent pleurisy, but no firm adhesions. On section, upper lobe of dark red color, and interlobular tissue appearing increased. Very small quantity of fluid from surface of section, and tissue not breaking down under finger. The whole of lower half of right lung solid, firm, and somewhat tough; of reddish-gray color, and offering some amount of resistance to the knife. Sank in water, and exuded scarcely any fluid when pressed. *Left lung* in a similar condition, except that the consolidation was arranged more in patches. *Bronchial tubes* much congested, but not dilated. *Bronchial glands* much enlarged. *Heart* healthy, except for contraction and puckering of one of columnæ carneæ. *Liver*, normal. *Spleen*, very large and firm. *Kidneys*, large, very firm, and tough. *Intestines*, healthy.

III. M., æt. 61. M. Charcot, De la Pneumonie Chronique, Thèse de Paris, 1860, p. 37.

General History. A hosier; delicate-looking; generally enjoyed good health, but has had a cough for some months, and has grown rather thin.—*Symptoms and Physical Signs.* March 30, 1850. Admitted. Five days ago, rigors, pain in side, and rusty sputa appeared. Had all the signs of pneumonia of whole of *right* lung, with, at first, simple febrile, and afterwards typhoid, symptoms. April 4. General condition improved; muco-purulent, instead of rusty, sputa. April 12–18. Some improvement in general condition: bronchial breathing and dulness continuing in upper part of lung; whilst over the lower lobe, with intense bronchophony and dulness, there was respiratory *silence*, not even bronchial breathing. No ægophony. April 18–29. Local signs continued without change; but return of appetite; feverishness at night, and weakness. April 29. Rigors, frequent respiration, fever, crepitant râle, mixed with bronchial breathing on right side. Epistaxis. Large blister applied. May 8. Better; but still occasional shiverings; eyes injected at night, and cheeks red; but little appetite. Signs of pulmonary induration continuing. May 9. Lower angle of right scapula raised by a *large and deep abscess*, from which, on incision, issued about 15 oz. of pus. Up to June 1st the abscess continued to discharge, though fluid gradually more serous in nature. During this time there were hectic fever and rapid wasting, but no diarrhœa. All the signs of pulmonary induration still continued. Slight cough; expectoration scanty and muco-purulent; no night sweats. During the month of June no alteration. Still losing flesh, but no cough, diarrhœa, or night sweats. July 1–9. Gradually became worse; the hectic persisting, but still none of the last-named symptoms or œdema of legs. On the 9th, the expectoration (being before scanty) became very abundant and somewhat nummulated. On this day the following *Physical Signs* were recorded. On *left* side, resonance good, with puerile respirations. On *right* side, below clavicle, marked dulness; respiratory murmur faint and indistinct. Posteriorly, dulness throughout; over superior lobe, respiratory murmur very indistinct. Vocal resonance over lower lobe, with marked bronchial breathing mixed with large metallic râles, simulating gargouillement. Bronchophony, but not pectoriloquy. But not always the same result: sometimes *complete silence* over whole of upper and lower lobes, and sometimes *tubular breathing mixed with large metallic râles.* No note made as to variations in expectoration at these times. Died on July 19th, much emaciated.—*Autopsy: Right lung*, universally adherent by old and tough adhesions; pleura constituting a fibrous envelope $\frac{1}{8}''$ in thickness. Whole organ $\frac{1}{3}$ smaller than left lung. Tissue heavy, dense; finger cannot penetrate it; on section, resisted scalpel like fibro-cartilage. The three lobes were seen to be firmly united, and the tissue change was the same throughout the whole lung. The surface of section was smooth, non-granular, grayish-blue marbled with black. *Bronchial tubes not at all dilated.* Pale ligamentous partitions of fibrous tissue subdividing lung in all directions, forming a minute network. *No trace of tubercle. Left lung*, large and perfectly healthy throughout. *Heart*, slightly large, flaccid; otherwise healthy. Other viscera carefully examined, and found to be healthy. A fistulous opening existed

near the lower angle of right scapula, leading into the cavity of an old abscess over the third, fourth, fifth, and sixth ribs, whose external surface was in a carious and necrosed condition, whilst the intercostal muscles were partly destroyed. The internal surface of these ribs was quite healthy, and no communication existed between the cavity of the abscess and that of the chest.

IV. M., æt. 30, Dr. J. E. Pollock.

General History. A soldier in India for 5½ years, and afterwards a warehouse porter. Tall, well-made, and pretty well nourished, though he had lost flesh. General health good.—*Symptoms.* Suffering from cough for the last nine months, and streaky *hæmoptysis* for six months. Expectoration profuse, frothy, and mucopurulent. Bowels constipated. Occasional pain in back between the shoulders. Death from rupture of aortic aneurism into left bronchus.—*Inspection, Percussion, Auscultation, etc.* Heart's impulse visible from apex region to second left cartilage. General contraction of whole left side, with flattening in front, and slight depression of shoulder. Dulness not absolute over *left* side; respiration very deficient in sub-clavicular region, with increased vocal resonance over apex posteriorly. On *right* side, increased resonance up to and beyond left of sternum. Respiration normal.—*Autopsy: Left lung*, adherent throughout, contracted; could only be removed together with costal pleura. On section, bronchi greatly dilated, with intervening tissue fibroid and airless. *Right lung* enlarged, covering pericardium. *Heart* healthy; aortic valves thickened, but not incompetent. *Aneurism* of descending part of aortic arch, ruptured.

V. M., æt. 7½. (M. Legendre.) Barth, in Mém. de la Soc. Méd. d'Observat. de Paris, 1856, t. iii.

General History. Parents healthy, was brought up by hand, took violent cold a few days after birth. Three weeks after began to vomit food, which continued for a long time. Cut teeth and walked at usual time. When 3½ or 4 years old, began to expectorate large quantities of purulent matter. Two or three times a day at most, after feeling of anxiety and face becoming red, there were paroxysms of cough, with copious ejections of pus. Felt relieved immediately after this. In intervals neither coughed nor spat. Habitual dyspnœa; skin hot at night, and copious sweats, but no diarrhœa; and appetite always good. From this time to 7th year continued much the same, but grew and was by no means thin. Of moderate size and fatness; skin pale; ends of fingers thick; intelligence good. No injection of face.—*Symptoms.* Oct. 11, 1841. In morning, skin cool, pale; pulse 80; but about 5 o'clock face becomes red, skin hot, pulse 116–120, respiration frequent, and in night abundant sweats. Two or three hours after meal, at 11 A. M., feeling of malaise with anxiety, and in fifteen or twenty minutes the cough comes with floods of expectoration, and often vomiting of food. Fluid thin, purulent, with stale odor; from 6 to 8 oz., though less when night attack also, as often. Dec. 13. Has lost flesh lately; evening attack constant, night sweats copious. Dec. 31. Thinner; bad diarrhœa for two days. Jan. 12. Feebler and thinner, much wasted, slight bed-sores. Breath fetid; ulceration of gums and inner side of right cheek. Appetite less; still diarrhœa. Jan. 14. Gangrene of gums inside of right cheek progressing, and breath very fetid. Jan. 16. Died.—*Inspection Percussion, Auscultation, etc.* Tongue moist, belly big, with tenderness over region of enlarged spleen. *Right* side resonant all over. On *left* under clavicle, as good as right side, but behind completely dull from top to bottom. On right side, and under left clavicle, respiratory murmur normal; but over whole of left back, respiration cavernous, with râles, and also great vocal resonance. Dec. 31. Not having been auscultated for several days in addition to previous signs, there was detected under left clavicle a slight dulness, with bronchial breathing and large mucous râles.—*Autopsy:* Pleural adhesions very slight, principally on left side. *Left lung* not diminished in size. Lower lobe heavy, hard; no appearance of air-vesicles on section, but seen to be converted into a dense, reddish, homogeneous tissue, containing cavities from size of pea to almond filled with expectoration matter. This part—in color, consistence, and density—was like tissue of an enlarged uterus. Bronchi normal, as far as first division; but afterwards *uniformly* dilated throughout. Upper lobe nearly as heavy as lower, but not nearly so dense; and consistent, though airless. Tissue reddish-gray, granular on surface of section, but breaks down less easily than recent hepatization. Bronchi only slightly dilated. Not the least trace of tubercle or gray granulation. *Right lung*, healthy and crepitant, except for a few "gray granulations" scattered through its substance. *Bronchial glands* on left side enlarged, reddish-gray. *Kidneys* and *liver* healthy. *Spleen* large and very consistent. Mucous membrane of intestine healthy.

Symptoms.—The symptoms of this affection present a considerable range of variation in different cases, according to the different modes in which the disease originates, and the amount of change which has been induced, not only in the

diseased lung, but also in the position and size of the heart. Thus one class of cases —and this includes a considerable proportion of the whole—are chronic from the first, appearing to commence obscurely, and being afterwards characterized by the symptoms of chronic bronchitis, with a limitation of the local signs to one lung. The cough, in these cases, dates sometimes back for a period of twenty years or more.

In another class, the affection dates definitely from some acute chest disease—either a bronchitis, a pneumonia, or a pleurisy without notable effusion—and then goes on, from this starting point, in much the same chronic way as when the mode of origin is indistinct. The cases in these two classes may or may not be associated with deviation in the position of the heart, signs of enlargement of its right cavities and dropsy. In a third class, the cases are more acute in their progress, and the morbid change seems to be the immediate sequence of an attack of acute pneumonia. The sufferers included under this last head usually succumb pretty early, and before the disease has attained to its later stages of development. It frequently proves fatal before the end of the first year from the date of the acute pneumonia.

The particular combinations of symptoms in individual cases may be best seen in detail, as they are given in the analytical table. I shall here confine myself to a more general consideration of the different symptoms and signs met with in the disease, and to an estimation of their relative importance.

Although the patient may have many of the physical signs of phthisis, its constitutional symptoms are almost entirely absent. There are no feverish symptoms, no signs of hectic, no copious night-sweats, no disorders of digestion, and the disease for the most part seems altogether of a more stationary and chronic character. No laryngeal symptoms have been noted in any case. Diarrhœa, although an occasional symptom, is less frequent than in ordinary cases of phthisis, and when it exists it may be an accompaniment of blood-poisoning from coexisting gangrene. Once diarrhœa was occasioned by an ulceration of the cæcum, which was rather obscure as to its nature and origin.

Cough is one of the most constant symptoms; it is sometimes present throughout, and undergoes but little variation, though it is often aggravated during the winter months. Where the disease is advanced and there is much dilatation of the bronchi, the cough is often paroxysmal, coming on in violent fits after long intervals of comparative quiet. Such individuals may have violent paroxysms of coughing in the morning, at the end of which the secretion that had accumulated in the dilated bronchi, during the interval between the present and the last fit of coughing, is voided in copious gulps. Vomiting of food may also take place at this time, and one, two, or even three such fits of coughing may, in some cases, occur during the twenty-four hours. The attacks are preceded by a feeling of discomfort and *malaise*, although comparative relief is experienced as soon as the irritation and pent-up secretions have been got rid of.

Where there is little or no dilatation of the bronchi, the *expectoration* is not very abundant, but rather tenacious, and occasionally somewhat nummulated in character. But where the disease includes dilatation of the bronchi, the expectoration is generally copious, muco-purulent, yellowish, or ash-green in color; having a tendency to run together into an almost homogeneous mass, which is often frothy on the surface. Owing to the thin seropurulent nature of the secretion in some cases, the fluid separates, after standing, into three more or less distinct layers—the lowest yellowish, containing most of the solid matter which has settled; a middle stratum of greenish fluid; and an upper frothy stratum, or one composed of mucous and fat granules. In these cases, the amount of fluid excreted daily may reach as much as ten or fifteen ounces. It has often a very stale and nauseous odor, and is sometimes even fetid,[1] though

[1] Upon the presence of what particular substance the fetor depends, different opinions have been held, as may be seen by the following quotation from Dr. Grainger Stewart's paper: "Professor Laycock concludes (Edin. Med. Journ., May, 1865) from experiments and observations made by the late Professor Gregory, Dr. Arthur Gamgee, and himself, that the odor must be due to butyric acid. He also states that Dr. Gregory detected the odor of methylamine in some of the products of the sputa. Professor Bamberger (Würzburg. Mediz. Leitz. 1864) concludes that the characteristic smell of the sputa in bronchiectasis appears to depend upon a variety of odorous matters, among which are the members of the series of acids of the type to which butyric and formic acids belong, ammonia and sulphuretted hydrogen, all of which may proceed from the decomposition of organic substances. He further states that purulent sputa—*e. g.*, that of tubercular patients—sometimes undergoes the same decomposition out of the body, and if long kept, have the same smell as the sputa in question. Dr. Arthur Gamgee (Edin. Med. Journ., March, 1865), from a considerable number of analyses of sputa, concludes that the occurrence of butyric acid cannot at present be proved to have any semeiological value, and that its presence is in no way characteristic of fetid bronchitis, under which term he includes bronchiectasis."

the smell is quite distinct from that of gangrene. On agitating the recent sputum with water, opaque, grayish filaments, of varying diameter, may soon separate and sink to the bottom. These are cases of minute bronchi, which, as first pointed out by Dr. Arthur Gamgee, assume a purplish tint on the application of iodine. They are met with more particularly in the fetid sputa, and, according to Niemeyer, the fine acicular crystals of margaric acid may also be detected by the aid of the microscope in the fetid sputa from dilated bronchi—though they are said not to be encountered in the bronchial secretion in any other lung affection, save that of gangrene. Dr. Grainger Stewart has found these crystals in the dilated cavities after death, but has failed to detect them in the sputa during life.

In more than one-half (17 : 30) of the recorded cases, there has been *hæmoptysis*—sometimes small in quantity, streaking the expectoration, and in others pretty abundant from time to time. Out of the thirty cases there are only four in which there is any mention made of the existence of "tubercle," either in the sound or in the cirrhosed lung. In one of these cases (V.) there was no hæmoptysis at all, in another the hemorrhage seems undoubtedly to have proceeded from the non-cirrhosed but "tubercular" lung; in the third it must, almost certainly, have proceeded from the cirrhosed lung; whilst in the fourth the hemorrhage (which was fatal in this case) seems to have mainly proceeded from the enlarged lung, although there was also a small cavity containing blood in the retracted lung. Thus there were fifteen, or one-half of the total number of cases of cirrhosis, in which hæmoptysis was one of the symptoms, and in which the hemorrhage undoubtedly proceeded from the cirrhosed lung, and in only one of these did "tubercle"[1]—and that in the smallest quantity—coexist with the fibroid change. This is an important fact in connection with the disease, and is in opposition to the view inclined to by Dr. Walshe and Dr. Law, who have both expressed their opinion as to the probability of the hemorrhage, in most cases, proceeding from the non-cirrhosed lung, in connection with the formation of "tubercle." In a small number of cases the patients have complained of pain in the affected side, either localized or indefinite in site.

Dyspnœa, though a constant symptom, is often moderate in degree, even in advanced stages of the disease—so long as the patient remains quiet, and the opposite lung continues to be healthy. It is occasionally more marked as an objective than as a subjective symptom, and is generally much increased after the slightest exertion. With reference to the pulse-respiration ratio, no definite details are given, except as to its condition in the case recorded by Dr. Walshe. Here he says, "It never fell lower than 3 : 1, and was sometimes found at the par of health, 4 : 1; even above this on one occasion—4·7 : 1." The dyspnœa is most marked in cases where there is dilatation of the right side of the heart and dropsy; orthopnœa is then a constant symptom, attended with more or less lividity of lips, face, and even surface of the body generally, whilst there may also be pulsation in both jugular veins. Purpuric spots of hemorrhagic effusion appear on the body occasionally. When an acute attack of bronchitis or pneumonia supervenes, the dyspnœa becomes asphyxial in its intensity, owing to interference with the breathing power of the previously sound lung, and death often speedily ensues.

The patient almost habitually lies on the retracted side; and any attempts to lie on the other cause great increase of dyspnœa and cough, so as to make it impossible to continue in this position.

The pulse is often regular and full, notwithstanding the frequent deviation in position of the heart. The appetite is usually pretty good; and in spite of the chronic nature of the cough, and the almost habitual copious expectoration, the patient does not lose much flesh. Towards the end slight emaciation is common, but extreme emaciation is rare in this disease; when it is uncomplicated by cancerous or other wasting affections.[1]

The mode in which the third class of cases originates has been well described by MM. Grisolle and Charcot. The individuals do not recover from the attack of acute pneumonia as they do in ordinary cases. On this subject the former observer says: "One sees at first the disease decline—in appearance at least; the pain in the chest disappears; the sputa lose their viscosity as well as their hemorrhagic color; the appetite reappears; but notwithstanding this improvement some symptoms obstinately persist; the patient, far from gaining flesh and strength, grows worse and worse in these respects, and one finds, on examination of the chest, that a more or less considerable portion

[1] Really, in all probability, a patch of chronic lobular pneumonia.

[1] Since this was written I have seen and made the autopsy of a man who suffered from an extreme degree of cirrhosis of the left lung, and in whom there also existed an enormous liver, studded throughout with the most typical cancerous nodules. No cancer was found in any other organ except in the bronchial glands, which were completely infiltrated—not even a trace of it could be discovered in either lung.

of the lung still remains impermeable to air—that is to say, percussion reveals dulness for a certain extent, whilst over the same part, on auscultation, bronchial respiration and bronchophony, with sub-crepitant and mucous râles, are heard." But it must be clearly understood that it is not the mere persistence of the local symptoms alone which have any significance, since M. Grisolle has shown that a slow return of the lung to its normal condition is a common, if not an habitual, sequence in a pneumonia whose result is favorable. Feebleness of the vesicular murmur, and a coarse breath-sound, mixed with sub-crepitant râles, are often the only signs of the unfinished resolution; though much more rarely, as M. Charcot says, "tubular breathing, bronchophony, and a more or less marked dulness, have been capable of persisting for two or three months after the complete cure of a pneumonia, and, notwithstanding this, there has not been the least tendency to a relapse, or the least return of febrile symptoms." Cases of this kind, however, which are not those to which we are more especially alluding, may be interpreted, as M. Charcot believes, by supposing that the new consolidating "materials have not been re-absorbed, and have remained for a time in the tissue of the lung, without the coexistence of any inflammatory action." But, in the cases where an inflammation of the lung is about to terminate in what MM. Grisolle and Charcot term "chronic pneumonia" (or, as we prefer to say, in a fibroid induration leading to cirrhosis), although the general symptoms occasionally subside for a brief period, they soon reappear. The symptoms, then, have more or less of a hectic character from the first; or there may be a preliminary and short reappearance of the symptoms of the acute condition—in other words, a relapse, of short duration. Gradually the hectic symptoms become more marked; every evening the skin becomes hot and the face flushed; sometimes night-sweats are profuse, and at others they are absent altogether; nutrition soon becomes impaired and the patients lose flesh, whilst cough and dyspnœa continue. Œdema of the lower extremities may supervene, and the patient, already wasting, may be still further lowered by the setting in of an obstinate diarrhœa. The resemblance of the general symptoms to those of phthisis is often most striking.[1] When the individual does not perish in the course of a few months from gradual exhaustion or from uncontrollable diarrhœa, the symptoms gradually diminish and the disease lapses into the chronic state.

PHYSICAL SIGNS.—*Retraction or shrinking of the thorax*, to a greater or less extent, on the side of the affected lung, is very frequent after the disease has existed for a certain time. It is, however, not commonly met with till after the lapse of about eighteen months. In two only out of seven cases, which proved fatal at or before this period, was there any flattening of the chest. One of these (I.) was that of a child only seven years old, at which age of course the flexible parietes would readily follow the shrinking lung; whilst in the other case (IV.), that of an adult, although the disease only presented symptoms for nine months, it seems to have made rapid progress, and there was obvious shrinking of the chest on the affected side, and even slight lowering of the shoulder. In the great majority of the individuals who live longer than eighteen months after the commencement of the disease, some amount of retraction of the chest is observed, either general or sub-clavicular; and in almost all, there is a proportionate amount of immobility on the retracted side. Moreover, in those cases where contraction cannot be detected, comparative immobility may be easily established. The flattening and retraction is an almost purely physical process dependent upon the shrinking of the lung within; and its amount depends principally upon the degree of rigidity of the thoracic parietes at the onset of the malady, and upon the rapidity of its course. If the lung-shrinking goes on pretty rapidly and the patient is young, the amount of contraction may be enormous—as actually occurred in Dr. Mayne's case, where the disease had existed for six years, and had commenced when the patient was only fourteen years old. The more the neighboring viscera are pulled into the space gradually vacated by the shrinking lung—the more the opposite lung enlarges—and the more the actual amount of lung-shrinking is diminished by the formation of dilated bronchial cavities—the less will be the amount of contraction or flattening of the thoracic parietes; all these conditions must be considered together, as they have a sort of complemental relationship to one another.

On *percussion* over the affected side—where the disease is well marked—we do not get a merely dull sound, but rather a more or less marked, high-pitched, tubular note, with firm, wood-like resistance under the finger. Over portions of the surface corresponding with large dilated bronchi, the note may present a well-

[1] The symptoms even of "galloping phthisis" may be imitated, where the disease is more rapid in its progress, and when it becomes associated with acute pleurisy, as in the case of M. Monneret, recorded by Charcot. (Loc. cit. p. 27.)

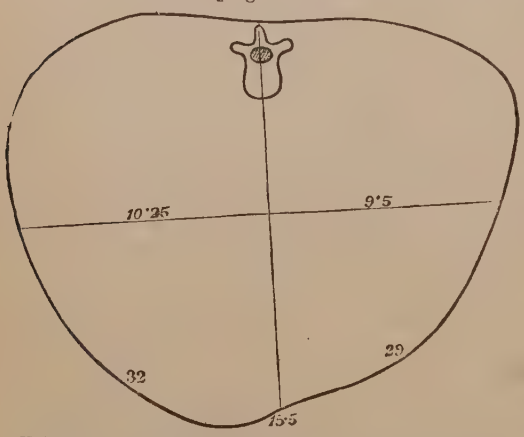
marked amphoric or tympanitic sound. The dulness is sometimes as distinct anteriorly as it is posteriorly; but occasionally the anterior area of dulness is diminished owing to the overlapping of the sound but hypertrophied lung, which extends into the diseased side of the thorax. In some extreme cases the percussion note may be good over almost the whole of the affected side in front, whilst it is absolutely dull with characters of resistance posteriorly.[1] On the opposite side of the chest the percussion note is almost always clearer than usual, and more like that which is met with when the subjacent lung is emphysematous.

On *auscultation* the normal respiratory murmur is either altogether absent or heard only over limited areas; whilst at other parts the respiration is high-pitched and bronchial, with cavernous and amphoric characters here and there. These sounds may be of the dry character; but, more frequently, there are moist rhonchi of various kinds—sometimes smallish, but mostly of the large bubbling kind and of a metallic character—such as constitute what is frequently described as *gargouillement*. The loud bubbling râles may be so abundant as to drown almost every other sound. Vocal resonance may be either diffused and bronchophonic, or various degrees of pectoriloquy may exist. Vocal fremitus is generally much increased over the dull parts, and this, together with the great sense of resistance on percussion, has, when it exists to a marked extent, considerable diagnostic value. The signs indicative of cavities may exist most plainly under the clavicle, towards the middle of the lung, at the base—or, perhaps, in all of these situations at the same time. It can scarcely be said, positively, that they are more frequent in one situation than in the other. Sometimes such signs, however, may be absent altogether. The breath-sounds over the opposite enlarged lung mostly deviate from the condition of health, only by being louder and more puerile than natural. When an intercurrent attack of bronchitis or pneumonia sets in, the character of the respiration on this side will, of course, undergo a corresponding modification.

The *position of the heart* often deviates much from that which is normal. The amount of displacement, of course, depends in great part upon the amount of lung-shrinking; but it is generally more considerable when the disease is in the right lung than when it is in the left. When the right lung is affected, the whole heart seems to be drawn over bodily into the right side of the thorax, so that its impulse may be felt only to the right of the sternum, whilst its apex impinges under the nipple. An amount of displacement so considerable as this has been encountered several times. In one case, in which the disease was on the left side, the heart's impulse was perceived close under the left clavicle; but in other instances the organ seems only to have been slightly drawn up, and the area of impulse, therefore, only slightly raised. Though its position is altered, the heart mostly beats with regularity, and no bruits seem to be produced by the displacement.[1]

More or less dilatation and hypertrophy of the right side of the heart has occurred in one-third of the cases. The deviation in position of the heart would often make it difficult to establish this by percussion and auscultation, but in three of the cases there were the signs of a loud tricuspid regurgitant murmur, associated with pulsation in the jugular veins and more or less dropsy. And in almost all the cases where the right side of the heart was found to be enlarged after death, there had been dropsy during life—either anasarca alone, or anasarca and ascites combined in a few of the cases. Dropsy existed in more than one-third (12 : 30) of the cases; so that it was present (mostly in the form of anasarca of the lower extremities) in a few cases where there was no dilatation of the right heart, and in

[Fig. 42.



Unilateral retraction of chest; consequent upon cirrhosis of left lung in a girl of fourteen years. The figures indicate antero-posterior and transverse diameters, and semi-circumferences of right and left half of chest (Dr. Gee).]

[1] This was most notably so in two cases recorded by M. Barth.

[1] Dr. Andrew Clark, however, states that "a low-pitched systolic bruit is commonly heard over the pulmonary artery."

which it depended upon disease of the kidneys or other coexisting conditions.

In the *acute form* of the disease, answering to what has been called "chronic pneumonia" (which may affect the whole of one lung, or only one lobe[1]), the physical signs are almost identical with those of the early stage of the chronic form, before much contraction of the lung has taken place. Thus, there is absolute dulness over the diseased part, with a considerable sense of resistance to the finger, whilst the vocal fremitus is much intensified. On auscultation over the dull part, bronchial or tubular breathing is heard; the latter being sometimes so loud as to be of a cavernous character even where no cavities exist. Râles are generally heard also — often loud, large, and metallic, though they are sometimes smaller or even absent. The vocal resonance is mostly bronchophonic. Occasionally, however, there may be a *complete absence of all breath-sounds*, either healthy or morbid, and of vocal resonance, whilst the percussion sound is quite dull—a combination which occurred in a case observed by M. Requin and quoted by Grisolle, and which led to its being mistaken for one of pleuritic effusion. In another case (III.) which has been recorded by M. Charcot, remarkable alterations were observed on different days. At one time, over the whole extent of a lung which was diseased throughout, there was a complete silence—no sound or râle of any kind; whilst at other times, on the contrary, there was loud and universal tubular breathing mixed with metallic râles. Unfortunately no observations were made as to the state of the expectoration at these times—either as to its quantity or quality.[2] Contraction of the chest-walls is of course not met with until the diseased lung has undergone a certain amount of shrinking, and by that time, if the patient survives so long, the intensity of the general symptoms has diminished, and the condition comes to resemble that of a person who is suffering from the more chronic form of the disease.

DIAGNOSIS.—The diagnosis of this affection in certain well-marked cases can be made almost with complete certainty, though in other instances only with great difficulty. The diseases with which it is most likely to be confounded are chronic pleurisy with retraction of the side, cancerous infiltration of one lung, certain forms of "tubercular" phthisis, simple general collapse of one lung, and simple or primary bronchiectasis.

In chronic pleurisy with retraction of the side, according to Dr. Walshe, the ribs are twisted downwards and inwards, the spine is curved, and the shoulder is drawn down; which effects are not produced by Cirrhosis alone. The lowering of the shoulder was, however, distinctly produced in one case of Cirrhosis occurring in a youth, with whom the amount of chest contraction was extreme; and it also existed to a slight extent in another case. Cirrhosis being so frequently complicated with dilatation of bronchi, is more frequently associated with physical signs of the hollow class; though the bronchial symptoms are not always most severe in cases where there is the greatest amount of contraction of the chest-walls. The heart is generally much more displaced by Cirrhosis than by chronic pleurisy. Then the frequency of hæmoptysis in Cirrhosis, with its non-occurrence in chronic pleurisy, must be borne in mind; and also, the greater frequency of enlargement of the right side of the heart with dropsy in the former affection. As Dr. Peacock has suggested, it will be well also to bear in mind the possibility of confounding Cirrhosis of the Lung with contraction of the organ succeeding an empyema which is evacuating itself through the bronchi, by means of a fistulous communication between them and the pleura.

Cancerous infiltration of one lung also causes retraction of the side, though, according to Dr. Walshe, the retracted ribs are not altered in axis, and the tendency seems to be to draw in an upward direction rather than latterly, so that when the disease occurs in the right side, the liver may be much elevated, though the displacement of the heart is much less than is met with in Cirrhosis. Occasionally, however, as we have seen, the same upward traction occurs in Cirrhosis. In both there is frequently cough, expectoration, failure of nutrition, and often hæmoptysis. The existence of well-marked signs of cavities of a stationary character, such as are due to dilated bronchi in Cirrhosis, would be absent almost universally in cancer. The condition of cachexia is generally more marked, however, in cancer, as well as the amount of intrathoracic pain; and the disease is often more rapid in its progress, its duration being sometimes much less, and never exceeding two and a half years. As Dr. Walshe points out, also, cancer of the lung is

[1] In this latter case the lower lobe of the right lung is said to be the most frequent seat of the disease.

[2] Charcot says: "Cela eut été cependant fort intéressant; car dans la pneumonie aigue, où l'absence de tout bruit respiratoire, normal ou anormal, s'observe quelquefois, ce phénomène paraît en général dépendre de l'obstruction des tuyaux bronchiques des parties hépatisées par une grande quantité de liquide visqueux ou par un bouchon d'exudation concrète." A similar temporary silence has been occasionally observed in cases of bronchiectasis.

generally associated with a mediastinal tumor of the same nature, so that not only may the morbid percussion note extend across the middle line, but there is apt to be greater dyspnœa, with lividity of face, and other pressure signs—such as dilatation of the superficial veins, and œdema of the thoracic parietes. In cases of cancer of the lung, moreover, cancerous tumors may exist in other parts of the body, and towards the last the cancerous cachexia often becomes extreme. This affection could, therefore, as a rule, only be confounded with the more acute forms of Cirrhosis.

"Tubercular" disease of the lung, presenting such characters as would render it liable to be confounded with Cirrhosis, is only encountered with extreme rarity. The characters of the latter disease which are most opposed to those of the more ordinary forms of phthisis are the signs indicative of an almost absolute freedom from morbid deposit in one lung, combined with the gravest amount of implication of the other—producing, perhaps, not only retraction of the side, but also cavities, and more or less complete impermeability of the lung-tissue between them. Then with local signs of so pronounced a character on one side (whilst the other lung appears to remain intact), we not only have no laryngeal disease, but there is a comparative absence of the constitutional symptoms peculiar to phthisis: so that there is an utter disproportion between the gravity of the local and the constitutional signs, and at the same time the disease presents a comparatively stationary character. Cirrhosis also frequently exists in previously strong individuals with well-formed chests; and, in one-third of the cases, there have been signs of hypertrophy and dilatation of the right heart, associated with dropsy. Only the contraction of an enormous tubercular cavern could produce such an amount of displacement of the heart as we frequently meet with in Cirrhosis; and that such an amount of disease and disorganization of one lung as this implies, should have existed without the least implication of the other, is contradictory to all experience as to the nature of ordinary phthisical affections. Any great contraction occurring in a "tubercular" lung is almost certain to be due to a considerable admixture of fibroid substitution with the other morbid product, so that the points of diagnosis just considered may be said to be those distinguishing the pure fibroid from the mixed fibroid and "tubercular"—or rather fibroid and pneumonic—forms of phthisis.[1]

Simple general collapse of one lung is a condition of extraordinary rarity, which, as Dr. Walshe says, could only result from the pressure of an aneurism or a tumor upon the main bronchus. In such a case, in addition to the signs of the tumor which might exist, there would in all probability, be a dull, toneless sound on percussion, instead of resonance of a wooden or even tubular character, whilst the respiration would be simply weak instead of bronchial, with more or less signs of cavities.

Simple primary bronchiectasis of one lung may exist, and then be followed by more or less fibroid induration of tissue.[1] Many of the signs and symptoms of this disease would be similar to those of Cirrhosis; only, in the early stages, the signs of cavities would be marked, whilst those indicating consolidation of the intervening lung-tissue would be comparatively slight. The signs of retraction of the chest and displacement of heart are almost or completely wanting.

Prognosis.—In almost all cases, the individuals suffering from this disease are ultimately carried off by an acute affection of the hitherto sound lung. An attack of bronchitis or a pneumonia supervenes, or a mixture of these two conditions, and the breathing power becomes so seriously interfered with, that the patient rapidly dies in an asphyxiated condition. Death may take place, also, from gangrene in the cirrhosed lung; or a copious effusion of blood proceeding from an ulcerating cavern in the lung may prove fatal—although usually the amount of blood lost in this way is not extreme.

In those cases hitherto styled "chronic pneumonia," and in which an extreme amount of fibroid induration follows an attack of acute pneumonia, the patient is apt to die in a state of marasmus, or from uncontrollable diarrhœa, before the local disease has attained its maximum—that is to say, before much contraction of the lung has occurred, or many bronchial caverns have been formed.

Death may also take place, however, when the disease is fully established, without the advent of acute inflammation

[1] With reference to the presence or absence of diarrhœa, it will be well to bear in mind the following remarks by Dr. Andrew Clark, which are in perfect accordance with my own experience. Dr. Clark says (Trans. of Clinic Soc. vol. i. p. 188): "Experience has peremptorily taught the writer, that the occurrence of ulceration of the bowels in the course of chronic disease of the lungs is not conclusive as to its tubercular nature. Deposits in and ulcerations of the intestinal glands may occur in almost any form of chronic disease to which the lung is liable."

[1] In rare instances, as before stated, owing to the amount of secondary induration and contraction, some of these may actually develop into cases where the cirrhosis becomes the most prominent feature.

in the opposite lung, but gradually, owing to the mere exhausting influence of the disease—when it is associated with marked bronchiectasis, and when the amount of purulent fluid daily expectorated is extreme. The occurrence of dilatation of the right side of the heart to such an extent as to produce tricuspid regurgitation, is, of course, a most grave complication.

In other cases, the patient is cut off by some acute or chronic coexisting malady, such as disease of the brain, cancer of the stomach, or uncontrollable diarrhœa from ulceration of the cæcum—diseases which actually proved fatal in a few of the cases included in my list.

Although the ultimate prognosis in this disease is most grave, still, if the sound lung can be maintained in its condition of health, the fatal termination may be warded off for some time, and the individual may live for years after the disease has been fully established.

TREATMENT.—The indications in this disease are to pay prompt attention to the very earliest signs of bronchitis, or pneumonia in the non-cirrhosed lung, so as, if possible, at once to arrest its progress. The patient's life should, moreover, be so regulated that, whilst exposed to wet and cold as little as possible, he may be brought under the influence of habits which are best calculated to promote the general health. The development of the non-cirrhosed lung should be favored by such carefully regulated exercise as can be indulged in without distressing the heart's action or causing much dyspnœa. Plenty of time should be spent in the open air; the diet should be good, simple, and nourishing; and the functions of the skin should be stimulated by the daily use of baths and dry friction. Whilst these general measures are being adopted, their action may be supplemented, when necessary, by various medicines. The dilute mineral acids or salts of iron, combined with bitter infusions, or iron and quinine, may be had recourse to; whilst in some cases, cod-liver oil, either alone or combined with iron, will be of much use.

In cases where diarrhœa sets in, every effort must be made to arrest this by the careful administration of dilute sulphuric acid, or by opiates and the various vegetable astringents; and, in like manner, where dilatation of the bronchi is well marked, and the daily flux from these is excessive, we must endeavor to check the copious flow by the administration of astringents combined with balsamic remedies (such as tolu, copaiba, or turpentine), and an application of counter-irritants externally. Where necessary, also, we must endeavor to bring about a regular and periodical evacuation of the dilated bronchi; so as to prevent decomposition of the retained secretion within the tubes, which is liable to produce general distress, and may also entail local gangrene. For this purpose Niemeyer strongly recommends the inhalation of turpentine two or three times a day. About half a drachm of the spirits of turpentine is to be placed in a bottle of hot water, and by means of some suitable addition to the neck of the bottle its vapor is to be inhaled. In this way the amount of secretion not only is diminished, but violent fits of coughing are induced in from ten to fifteen minutes, which are accompanied by an evacuation of the contents of the dilated bronchi. Niemeyer says he has seen great amelioration thus induced in the symptoms of patients whose condition had been previously most distressing.

With regard to the possibility of bringing about an actual disappearance of the new fibre-tissue, and a reappearance of the lung-tissue which it has supplanted, this seems a result beyond our most sanguine expectations, and one to which we are scarcely likely to attain. But, whilst the disease is still advancing, we may hope and ought to endeavor to prevent the spread of the morbid change to previously healthy portions of lung-tissue. This desirable result will be best brought about, not only by the means before alluded to, which are destined to bring the patient's general health up to the highest possible standard; but will, perhaps, be also encouraged by the use of iodide of potassium internally, in conjunction with counter-irritation to the affected side, and the free inunction of iodine locally. As Dr. Walshe suggests, a trial might also be made of some of the natural iodurated water, such as those of Kreuznach or Woodhall. The amount of influence which the iodides have in checking the over-growth of fibre-tissue seems in some cases to be most marked, and in a disease of so grave a character as this we are bound to try the influence of remedies which may have a favorable action, so long as they exercise no deleterious effect.

APNEUMATOSIS.

By Graily Hewitt, M.D., F.R.C.P.

Definition.—Apneumatosis is that condition of the lung-tissue characterized by the return of certain air-cells to a quasi-fetal state; the portions of lung so affected have once been physiologically active and efficient in promoting the respiratory change in the blood circulating through them, and have ceased to be so.

History.—The older observers of the diseases of children record the great frequency with which they found after death certain parts of the lungs solidified. The death was in such cases attributed to this alteration of the lungs; and as it resembled, in many of the outward appearances observed, the solidification found in the lungs of adults, and which had received the name of "pneumonia," they naturally enough gave the two conditions the same name. Only within a comparatively recent period has it been established that the two conditions are essentially different.

One circumstance, however, was observed as peculiar. The consolidation was always in the cases of young children seen to be abruptly separated from the adjoining sound lung, to be mapped out as it were by the lobular divisions of the lungs. Hence it was called "lobular pneumonia." It was said that in the case of infants and young children the pneumonia was *lobular*. The mortality from the disease so-called was found always to be very considerable, and hence children were considered to be *par excellence* predisposed to pneumonia.

Valleix[1] has given an admirable account of this lesion. Unable, however, to reconcile the facts observed with the theory that the lesion in question was true pneumonia, he thus expresses himself: "La forme particulière de cette hépatisation ma paraît donc inexplicable dans l'état actuel de la science." Before Valleix, Gerhard and Rufz and De la Berge had described in a very suggestive manner the peculiarities attending this alteration of the lungs in young children, both in respect to its essential characters, and to the circumstances under which it was found to be present. Earlier still, Leger pointed it out as peculiar pneumonia under the term "latent."

[1] Clinique des Maladies des Enfants nouveau-nés, p. 197; Paris, 1838.

Seifert[1] recognized the nature of the so-called lobular pneumonia so far as its mode of production was concerned, giving it the name of "bronchio-pneumonie," and he pointed out the counterpart of the lesion in that kind of pneumonia seen in adults which Laennec termed "peripneumonie des agonisants," and Piorry "pneumonie hypostatique." There is reason to believe, however, that some of the cases alluded to by him were really cases in which the lung had never been expanded at all at the affected parts, but remained from the day of birth in the state described and truly explained by Jörg as *atelectasis*.

Barthez and Rilliet[2] first, in 1838, distinguished between "lobular" and "lobar" pneumonia, laying down the principle that lobular pneumonia was always a secondary affection connected with bronchitis.

MM. Legendre and Bailly,[3] however, have the merit of first pointing out the essential nature of the condition known as lobular pneumonia. They used the very simple expedient of artificially inflating the lungs after death, and observed the effect of the inflation on the portions consolidated and in a state of lobular pneumonia. The result was, that the apparently hepatized parts swelled out, became filled with air, and were, as it seemed, suddenly converted into healthy-looking lung-tissue. The lung so inflated was found to possess all the physical characters of lung in a normal condition, and it was evident to these observers that the essential difference between the pseudo-hepatized and the sound lung only consisted in this,—that in the former case the air was withdrawn from the air-cells, the tissue of the lung itself not being necessarily altered. They were led to this result by observing how closely the lobules so collapsed resembled in external characters those of the lungs of the fœtus which has never respired; hence they replaced by the term "état fœtal" the old designation "lobular pneumonia." The results of this discovery have been most important in enabling us by a simple and easily applied test to ascertain whether

[1] Die Bronchio-Pneumonie der Neugeborenen; Berlin, 1837.

[2] Traité des Maladies des Enfants.

[3] Archives de Médecine, 1844, p. 157.

the condition of the lung present in a particular case is one only of collapse, or whether it is due to a change of another character altogether.

MM. Legendre and Bailly showed that in the cases in which their "état fœtal" was present, there was no true inflammation of the lungs present such as would entitle them to be considered as cases of true pneumonia; pneumonia, as it is generally understood, being constituted by a breaking-down or softening of the lung substance, whereas in cases of lobular pneumonia the lung-tissue is quite firm, and, with certain exceptions, not easily broken down under the finger. The peculiar limitation of the morbid change to certain lobules, the change beginning and ending abruptly, and not shading off gradually, also evidently gave to it a character altogether distinctive, and such as is not found in true pneumonia. Reference more in detail will, however, be made to these several points further on.

The subsequent history of "lobular pneumonia" will include an account of the more or less complete adoption by recent writers of the views of MM. Legendre and Bailly. By West, Gairdner, and Jenner, the terms "bronchial" or "pulmonary collapse" are used instead of "état fœtal." The term "Apneumatosis" was first employed by Fuchs[1] in an excellent treatise on the subject, and its adoption was recommended by myself in a paper read before the Royal Medical and Chirurgical Society of London.[2] The old term "lobular pneumonia" being calculated to give very erroneous ideas of the condition to which it is applied, a new term was necessary. "Pulmonary collapse" was not sufficiently distinctive, the word "collapse" being already in use, and very properly, in another sense, to indicate the spontaneous collapse of the lungs, which always occurs to a greater or less degree on opening the thorax after death. In the absence of a better, the word Apneumatosis (ἀ, *privative*, and πνευματῶσις, *a condition of being filled with air*) was considered the most appropriate, expressing, as it does, precisely the condition which is present, without involving any theory as to its cause or mode of origin.

Apneumatosis is not simply an anatomical alteration; it must be regarded as a diseased condition of the lung manifesting itself by a variety of symptoms and signs, producing certain important effects on the system at large, and very frequently proving fatal. Its importance fully justifies its being considered separately from bronchitis, with which it is always, or almost always, associated. The bronchitis of early childhood is, when fatal, almost invariably attended with Apneumatosis.

PATHOLOGICAL ANATOMY.—A description of the physical characters of those portions of the lungs affected with Apneumatosis will in reality include more or less completely a description of those lesions variously designated by authorities up to the present time as *Disseminated Lobular Pneumonia*, *Carnification*, *Pulmonary Collapse*, "*état fœtal*," *Marginal Pneumonia*, *Catarrhal Pneumonia* (Rokitansky), *Bronchio-Pneumonie* (Seifert), together with some of those described as *Atelectasis*, all these terms applying to one condition which is somewhat modified in certain cases.

The account given by Legendre and Bailly of the physical characters presented by the lung so affected is remarkably true to nature, and our own observations, as well as those of others, confirm the accuracy of the facts stated by the authors in question.

There is no material difference between Apneumatosis and Atelectasis, anatomically speaking, and it would be exceedingly difficult, judging from the mere physical characters present, to distinguish between them.

In the first place, the lobules affected are remarkably distinct; abruptly separated from adjoining healthy lobules, generally depressed below the surface of the healthy lobules; for the most part they are found at the margins of the lobes,—those portions of the lobes most distant from the root of the bronchial tree. The distribution of the affected lobules is evidently connected with their relation to the divisions of the bronchial tubes, and is such as to preclude the idea of the affection having spread by simple contiguity. The lobules supplied by one particular bronchial tube all present the physical characters of the lesion, whilst the lobules supplied by the closely contiguous bronchial tube may be perfectly healthy. The number of lobes affected is of course subject to great variety: it occasionally happens that the whole of one particular lobe is affected, but this is not very frequently observed, and it most commonly happens that nearly all the lobes present a greater or less number of apneumatic patches, although it is somewhat rare to find apneumatic portions present in all the lobes.[1] Certain parts of the lungs present this lesion with remarkable frequency. These are, first, the lower margins of the lower lobes of both lungs, the tongue-like prolongation of the upper left lobe, and the middle lobe of the right lung. Next in order come the posterior surfaces of the lower and of the

[1] Die Bronchitis der Kinder; Leipzig, 1849.

[2] See Proceedings of Roy. Med. and Chir Soc. No. I.

[1] See Valleix, op. cit. p. 62 et seq.

upper lobes. The order of occurrence here laid down is rarely departed from, a very interesting circumstance, and one which will again be alluded to in considering the essential nature and mode of production of the lesion in question. The lobules situated at the periphery of the lung are thus the first affected, and in slight cases the alteration is confined to them.

In many cases the apneumatic patches are symmetrically placed on the corresponding parts of each lung. This is especially the case with the posterior surfaces of the lower lobes, where we have frequently observed a line concave superiorly passing across this aspect of the lobe on each side, and forming the upper boundary of an apneumatic portion of the lung almost identical in form and equal in superficies on the two sides.

It generally happens, when Apneumatosis of the kind to which the description given above would apply is present, that the healthier parts of the lungs are pitted and depressed at certain parts, and these depressed portions have a rather darker color than usual. The little pits are caused by the partial collapse of the air-cells there situate, and, although slight in degree in particular lobules, the aggregate number of air-cells thus rendered useless may be very considerable. Apneumatosis thus slight in degree disappears completely on inflation, and between this and the more complete form involving the whole of several adjacent lobules, many gradations may be witnessed.

To the naked eye the apneumatic patches appear like islets of a darkish-red color abruptly separated from the lighter and more healthy lobules, and having a sharp determinate outline. On a more minute inspection fine whitish streaks are evident on the surface, dividing the affected portions into compartments. These indicate the boundaries of the small lobules affected, and it is thus evident that no inflammatory destruction of the lung substance has taken place. With reference to the color of the affected patches, nothing is more variable. The lung of the young child is naturally of a light-pink color, and the various shades of darkish-red presented by the apneumatic lobules contrast in a striking manner with the lighter and more healthy portions which lie close to them. The typical color is a darkish-red, with a shade of violet. The intensity of the color present often depends on the degree to which the lobules are congested. Sometimes the color is a lightish-red, like that of a piece of anæmic muscle, but at other times it is a deep purple; and between these two extremes many varieties of color are observed.

The depth to which the lung tissue is affected is in an almost direct ratio to the degree in which the lung is seen to be apneumatic superficially. Section of the part shows the same definite limitation of the Apneumatosis which is evident externally; the shape of the portion of lung involved in the change is determined by the outline of the lobules to which certain bronchi are distributed, and each apneumatic portion has thus a more or less pyramidal form, the base of the pyramid being towards the periphery. The apneumatic lobules are generally depressed below the level of the adjacent part of the lobe. This is not always the case however. When these lobules are less in bulk than usual, they have a somewhat lighter color than in the other condition of things. Thus the apneumatic portions which are of a deep violet color have usually the normal bulk, and may even exceed it. This circumstance is connected with the greater or less quantity of blood contained in the vessels of the part.

The *consistence* of the apneumatic lobules is open to some variation. This is in like manner connected with the quantity of blood within the vessels of the part, and also with the length of time the lesion has existed. The paler, less bulky lobules have a loose texture exactly resembling that of a piece of flesh: the anterior tongue of the left upper lobe often presents this peculiarity. The more congested and darker parts have, on the contrary, a very firm consistence, much resembling that of a piece of liver, and resist pressure much more effectually than the looser portions. In all cases the apneumatic lobules are found to sink in water. The section is very smooth and even, and when the part is much congested it resembles that of a firm clot of blood. The bronchi cut through often contain mucous fluid in considerable quantity. Where the lobe is thin, as in the anterior tongue of the upper lobe, the lobules are in many cases felt quite distinctly between the fingers. This is due to the fact that the air-canals within the individual lobules are filled and distended with mucous secretion, which everywhere extends into the air-cells. This peculiar feel is lost on cutting through the lobules in question, for the fluid then escapes out of the cut vessels. This extremely distended state of the air-channels is chiefly found in those parts in which less congestion is evident.

Inflatability.—The most important circumstance, however, in reference to the apneumatic lobules is the absence of air. No air-cells are visible on the pleural surface, none on the surface of the section. The difference presented between two parts of the same lobe, one of which is apneumatic and the other healthy, is in this respect most remarkable. All around the affected lobule the air-cells are most

distinct and evident: none are visible where the Apneumatosis exists. These lobules are, in fact, as a rule completely destitute of air; the opposite walls of the air-cells are in apposition; the finer air-tubes are either filled with fluid or in the same condition as the air-cells themselves —*i. e.* collapsed. As a consequence of this non-aërated state of the lobules, they are found to be absolutely non-crepitant on pressure. If a blow-pipe be introduced into the bronchus leading to the collapsed portion, and air be then forcibly injected, an instantaneous change takes place in the color, appearance, and physical characters of the apneumatic lobules: they swell out, become of a light rose-red, air-cells are at once visible on the surface, and the affected lobules come to so far resemble the adjoining healthy ones, that they are not to be distinguished from them. As has been already stated, this effect of insufflation was first pointed out by MM. Legendre and Bailly. Now the effect of this insufflation is not always the same. It is not always entirely successful, some portions of the affected lobules resisting this forcible refilling of the air-cells. As a rule, the operation is performed easily and with the use of very little force, and it always partially succeeds. The portions which resist the attempt to introduce air are those which are the most congested, and in these small portions remain uninflated, whatever may be the force used by the lungs of the operator. This point it is important to remark upon, for there is reason to believe that a want of success in the operation of inflation in cases of the kind alluded to, has induced some observers to doubt the correctness of the general statement with reference to the nature of the lesion now under discussion. The fact is, however, that where the Apneumatosis and considerable congestion coexist, the pressure of the contents of the bloodvessels effectually prevents the re-distension of the air-tubes, and air cannot be made to pass into the ultimate air-cells. A difficulty of the same kind occurs when the air-tubes are much distended with fluid secretion, but here it is hardly ever practically productive of much opposition to the operation of inflation. After inflation has been performed the dilated air-cells have a tendency to collapse again in a short time if the bronchus leading to them be not tied.

The operation of inflation is to a certain point a test of the presence of Apneumatosis as distinguished from pneumonia. In Apneumatosis, as has been already explained, it generally succeeds. In pneumonia, however, the lung-tissue cannot be restored to its *natural appearance* by inflation. It is not often that an opportunity presents itself of verifying this statement so far as the pneumonia of children is concerned, it being very rare to meet with true hepatization at this period of life, but in two or three cases which have come under our notice this verification has been completely effected. Lastly, the pleura is almost invariably found to be healthy in cases of Apneumatosis, uncomplicated with diathetic disease; in pneumonia it is just as rare to find it free from disease.

The physical characters of Atelectasis as distinguished from those of Apneumatosis.— The two conditions are almost identical, anatomically speaking, and in some instances, indeed, the history and other particulars of the case must be examined in order to decide the matter at issue. Some go so far as to say that Apneumatosis and atelectasis are one and the same thing, that the lobules presenting the characters described above as those of Apneumatosis have never been expanded at all, and have been in the condition in which they are found.[1] The grounds on which this statement is made are very insufficient, and will not bear examination. The following fact is quite sufficient to settle the contested point: a child, previously healthy, is seized with a catarrhal affection of the air-tubes; up to that time there has been evidence that the act of respiration has been habitually performed in a regular manner; after a few days the child dies, and a large portion of all the lobes is found collapsed; the physical signs and symptoms, during the last few days, having indicated, step by step, the progressive and increasing disease of the lungs. A few instances, perfect in every particular, it is not a difficult matter to collect. With such an amount of lung implicated in the lesion it is difficult to conceive that symptoms could have been previously absent. A weakly child affected with atelectasis is easily the prey to bronchitis, and this latter affection is so fatal because in such a case it is so often followed by Apneumatosis. This seems to be the proper way of stating the relation of the two lesions, atelectasis and Apneumatosis, one to the other.

When the lung has undergone mechanical compression, and thus become hardened, reduced in bulk, as in cases of pleuritic effusion, &c., its physical characters to a certain degree resemble those of Apneumatosis. It seems desirable to restrict to this condition the term *carnification*. Carnified lung is firmer and denser than is the case in Apneumatosis, and differs from it in the essential particular that it is not susceptible of inflation; added to this, the peculiar circumstances

[1] Such appears to be the opinion of Friedleben. "Ueber die Pneumonie der Kinder," in Archiv, für physiologische Heilkunde, 1847.

under which it is found are sufficient to establish its true identity. In order to prevent unnecessary confusion we have hitherto described only Apneumatosis of a typical character, or rather Apneumatosis in which the condition of the air-cells present is one of simple collapse. The air-cells are destitute of air, their walls are in apposition. In certain cases, however, on the surface of the apneumatic portions are seen little elevated oval or rounded spots of a yellowish-white color, resembling at first sight tubercular masses. These little cavities are situated immediately under the pleura, communicating freely with the bronchial tubes. They are the "granulations purulentes" of Fauvel. They are for the most part, according to our own experience, found in portions of lung affected with Apneumatosis, but they may be found in other situations, and are not therefore perhaps so entirely a part of this affection as to justify their being considered fully in this place. It will suffice here to say that in chronic cases in which Apneumatosis is present the little cavities in question are rarely absent. They are often described as "bronchial abscesses," "vesicular bronchitis," &c.

Etiology. — Apneumatosis is a mechanical effect of the presence of certain morbid conditions of the air-tubes, these morbid conditions appearing to be particularly efficacious in the production of Apneumatosis during infancy and early childhood.

Catarrhal inflammation of the bronchi, either existing *per se*, or forming a part of other diseases, is a very common affection in early childhood, and Apneumatosis is one of its effects, the presence of a mucous secretion in the finer air-tubes preventing the due aëration of the lobules to which they lead. The connection between the two circumstances, excessive secretion and collapse of the air-cells, is one which is supported by considerations the result of experimental and pathological inquiry.

It appears that any obstruction of the bronchial tubes is sufficient to produce after a time the appearances of Apneumatosis in the distal lobules. The experiments of Mendelssohn and Traube, described by Fuchs,[1] are especially interesting as demonstrating this fact.

In one of these experiments tracheotomy was performed on a dog, and a shot introduced which was afterwards found in the left bronchus. In two days death took place, and the appearances found were as follows:—The right lung was emphysematous, enlarged; the left lung was collapsed, its lower lobe was in great part congested, devoid of air, and also the upper lobe in certain parts, near which lay emphysematous patches. Inflation distended the whole lung. In other experiments, a like effect was produced by the introduction of a ball of paper, certain portions of the lungs becoming hard, condensed, and no air-cells being visible on the surface.

The resemblance between these cases and those of children affected with Apneumatosis due to the obstruction produced by the bronchial secretion is, as Fuchs remarks, at once apparent. The relation of bronchial obstruction to pulmonary cohesion, also indicated by Legendre and Bailly, has been more completely developed by Dr. Gairdner,[1] so far as the mechanism of the process is concerned, with whose acute and original remarks our own almost completely agree. Dr. Gairdner has demonstated the nature of certain lesions of the lungs, met with in adults, and identified them with Apneumatosis. "Bronchitic" collapse, as he describes it, is therefore not peculiar to children, although very much more common in them than in adults. Dr. West gives to the theory of the connection between bronchial obstruction, produced by secretion, and "pulmonary collapse," his entire support, in common with Bailly and Legendre, laying also some stress on the imperfect inspiratory power of weakly infants as an additional predisposing element in the production of the lesion in question. Gairdner's satisfactory and lucid explanation of the *rationale* of the process by which Apneumatosis, or collapse of the lung, is produced, is as follows:—

Commenting on the experiments of Mendelssohn and Traube, before alluded to, he says, "It is clear, therefore, from experiment, as well as from pathological observation, that the most usual and most direct effect of obstruction, or of diminished calibre of the bronchi, however caused, is not accumulation" (as Laennec had contended), "but diminution in quantity of the air beyond the obstructed point." The author then shows that another mechanical condition which comes into play in producing collapse from obstruction is to be found in the form of the tubes; these diminishing in size as we approach the periphery of the lung; consequently, if the calibre of a tube be nearly filled at one point by a plug of mucus, the effect of inspiration, propelling it towards the air-cells, will be to completely close the tube, when it arrives at a part the calibre of which is less than that which it originally occupied. The plug of mucus will thus act as a ball-valve, and at every

[1] Loc. cit. p. 61 et seq.

[1] On the Pathological State of the Lung connected with Bronchitis and Bronchial Obstruction: Edin. Monthly Journal, 1850–51.

expiration a portion of air will be expelled, which, in inspiration, is not replaced. In the end, the lobule to which the bronchus in question leads, contains no air at all, and the condition to which it is reduced is one of Apneumatosis. Fuchs, in the work referred to, and also quoted by Gairdner, accounts for the disappearance of the air from the lobules, by supposing it to be absorbed by the bloodvessels, having been first shut in and confined by the presence of mucus in the tubes, these latter having, moreover, their calibre diminished by the thickening of the mucous membrane always present. Dr. Gairdner's explanation is rather too much dependent on the supposition that the bronchi contain a tenacious, viscid material; this may be the case in adults, but in the case of children an examination of a considerable number of cases has convinced us that the bronchial tubes are rarely found to contain mucus having the characters of tenacity and viscidity: in almost all cases indeed, the mucus readily flowed out of the vessels when cut across, and had the consistence of thin pus. Here of course Dr. Gairdner's explanation also holds good, but it is only necessary to add that tenacity and viscidity of the contained mucus is not an indispensable element in the explanation in question.

With reference to the opinion of Fuchs as to the cause of the disappearance of the air, it is probable that it is in part true; the fact of the disappearance is sufficiently accounted for by a combination of the theories of both of the authors. If such absorption take place, it is natural to suppose that the oxygen will disappear first, and be replaced by carbonic acid; this latter product being readily dissolved in fluid will also finally be carried away, together with the nitrogen. The dark coloring of the apneumatic portions Fuchs attributes to the excessive quantity of carbonic acid present.

The inability to cough and expectorate is another circumstance to which Dr. Gairdner alludes as a cause of bronchitic collapse. It appears to us, however, that this is rather to be looked upon as a consequence than as a cause of the collapse, at least at the commencement. The efficiency of the cough in expelling mucus from the tubes is dependent on the presence of air in that part of the tubes beyond the obstruction. Each lobule is a miniature lung, and the sudden expulsion of the air from the lobules drives the obstructing agent before it. As long, therefore, as the air-cells contain air, so long will the cough aid in the expulsion of mucus from that part of the lung. When the Apneumatosis has been produced in certain lobules, those lobules are in great part unaffected by the cough, and there is no expulsion of mucus from the air-tubes with which they are supplied. The Apneumatosis is thus *perpetuated* by the inability to cough and expectorate, but it is not produced by it except in a secondary manner. The fatal result of cases in which Apneumatosis occurs is probably connected with the absence of expectoration, and the imperfect character of the cough.[1]

It is evident that the condition here supposed to be effective in the production of Apneumatosis is only the last step of the process. Why, it will be inquired, is Apneumatosis so especially common in young children, while it is so rarely observed in adults? In the first place, it must be answered that Apneumatosis is not so rare in adults as has been imagined, which fact is shown by a perusal of Dr. Gairdner's papers just alluded to; his statement being in great part, indeed, founded on observations made in adults. But, on the other hand, it cannot be denied that Apneumatosis is comparatively much more common in early life, and there must accordingly be certain powerful predisposing circumstances leading to this result, favoring circumstances or conditions, without which Apneumatosis would not more readily occur in the one than in the other.

These *predisposing circumstances* it may be well to consider a little more closely. Whatever tends to lessen the intensity of the inspiratory effort, and thus to impair its efficiency, will certainly favor the occurrence of Apneumatosis. The introduction of air into the air-cells is the result of a mechanical process, the walls of the chest are separated, and the diameters of the chest increased by the action of certain muscles: the lungs follow the walls of the chest, and increase in bulk, and air is driven in to fill up the vacuum which would otherwise exist within the chest. The principle, indeed, precisely resembles that of the pump. Now, in order that a pump may act efficiently, a rigid state of the walls of the tube which the piston traverses is necessary; the atmospheric pressure would otherwise produce collapse of these walls. In like manner it is necessary that the parietes of the chest be sufficiently rigid to prevent their being driven inwards by the pressure of the atmosphere from without during the process of inspiration. The walls of the chest in the child are very far from presenting that firmness and resistance which is observed in the adult; the result of this is that at certain situations the ribs fall inwards during the act of inspiration, and at the corresponding part of the lungs little expansion of the pulmonary tissue occurs.[2] This collapse of the thoracic walls may sometimes be observed in infants who are breathing vigorously when

[1] Dr. Stokes. [2] Rees.

the air-tubes are everywhere quite patent. The diaphragm, which is the chief inspiratory muscle in early life, also tends to draw in the chest-walls at the points of the ribs to which it is attached, if those walls do not present a sufficient degree of rigidity. The point at which the chest-walls most readily give way is at the junction of the cartilages with the ribs, and the ribs which more especially exhibit this want of power to resist the atmospheric pressure are those just above and below the nipple, the fourth to the seventh inclusive. Not unfrequently a groove may be observed passing downwards at the junction of the cartilages with the ribs on each side, marking the degree to which these parts have given way. Rickets is a frequent source of this, rendering the bones more pliant than they should be. Sir William Jenner has particularly demonstrated the great influence of rickets in producing this result. Another circumstance which acts in a somewhat different way, is congenital or induced general weakness. In this case, the muscles which elevate and draw asunder the ribs are not powerful enough to withstand the opposing force of the diaphragm; the ribs here may be rigid enough, but the muscles are incapable of retaining them separated and elevated, while the diaphragm acts. A combination of the conditions here mentioned—viz., deficient rigidity of the bones or framework of the thorax, and deficient power of the muscles—will obviously have a very considerable influence in diminishing the efficiency of the inspiratory act.

But under ordinary circumstances nature provides a remedy for these defects. If the chest-walls give way at one point, and the diameter of the thorax be thus diminished in that situation, it is increased in a corresponding degree at another situation. It is only when to the mechanical defects here pointed out others are added that serious diminution of the oxygenation process results. We have hitherto supposed the channels by which the air is admitted to the air-cells to be free. If any obstruction arise in the bronchial tubes, the mechanical defects first described enhance in a very considerable degree the difficulty which the child experiences in performing an efficient inspiratory act. The already defective apparatus is impeded in its action, and the quantity of air inspired is proportionately small. Catarrhal inflammation of the air-tubes is generally the origin of the obstruction in question. It produces, in the first place, a swelling of the mucous membrane, and secondly, a secretion of fluid; the one diminishing the calibre of the air-tube, the other obstructing it. Unless the child possess sufficient strength to overcome this obstruction (a strength often wanting) by exercising a greater effort than usual, Apneumatosis of certain parts of the lung will be produced in the manner previously described. The fact that, on the one hand, the small air-tubes are proportionately less in the child than in the adult (Fuchs), and on the other, that bronchial inflammation is so exceedingly common in childhood, will present conditions highly favorable for the production of the lesion, coupled, as they often are, with the partly inherent defective mechism of the inspiratory act at this period of life.

The researches of Hutchinson and others have shown that the act of inspiration is one-third less powerful than that of expiration. Under the morbid conditions just pointed out the disadvantage under which the inspiration labors is increased, while the efficiency of the expiratory effort is but little impaired. All the conditions mentioned are such as render the inspiration more difficult, and tend to prevent the passage of air into the air-cells. Inspiration being entirely dependent on muscular effort, is directly influenced by the degree in which that effort can be exercised, subject to certain modifications already pointed out; whilst the expiratory act being in part the result of the reaction of the elastic tissue of the lung, is much less liable to alteration of this kind. This, then, is another circumstance facilitating the removal of air from the air-cells when the tubes contain an undue quantity of fluid, the obstruction interfering with the inspiratory, but not to a corresponding degree with the expiratory, effort.

A condition which somewhat interferes with the inspiratory act is undue distension of the abdominal cavity, from whatever cause.[1] The diaphragm cannot descend to the full extent necessary, and less air than usual enters the chest. In common with most of the other conditions named this distension of the abdomen will not be effective in the production of Apneumatosis, unless coexisting with obstruction in the air-tubes themselves. The practice which often prevails of binding up the abdomen of the infant tightly must act in precisely the same way, and if the child be attacked with bronchial catarrh it is not difficult to conceive that the mechanism of the inspiratory act may be so impaired, under this combination of evils, as to favor the occurrence of Apneumatosis.

Certain affections of the air-tubes more readily than others produce obstruction and consequent Apneumatosis. Infants having portions of their lungs in a state of atelectasis are more liable to suffer from Apneumatosis than those in whom

[1] This point has not escaped the notice of Dr. Gairdner (loc. cit.).

the lungs have been fully aërated at birth: atelectasis is therefore a predisposing circumstance.

Apneumatosis is not by any means frequently observed, in such a degree at least as to prove fatal, after the age of five or six years; it is very common, however, before this period, and in general terms its frequency may be said to be inversely as the age. The first few months of the infant's life are those in which the lung most readily returns to the quasi-fœtal state, loses its gaseous contents, and becomes apneumatic. As the muscular power becomes greater, and the framework of the thorax becomes firmer and more consolidated, Apneumatosis less commonly occurs. The mortality from affections described in the Registrar-General's Reports as pneumonia, hooping-cough, bronchitis, and influenza, in the first year of life, is a rough index of the comparative frequency with which Apneumatosis occurs at this period of life. The result of examination of a large number of cases of children dying from bronchitic and allied affections during the first year of life, was, that with hardly an exception Apneumatosis was present in all, other complications being in many cases also noticed. I am inclined to speak less positively of the state of the lungs present in children dying of such affections after the age of about five years, opportunities being much more rarely afforded of studying the post-mortem changes after this period.

In round numbers the deaths during the first five years of life, and set down in the Registrar-General's Reports under the heads Hooping-cough, Influenza, Bronchitis, and Pneumonia, amount to 25 per cent. of the total mortality at those ages; between the ages of five and ten years, they amount to 10 per cent. of the total mortality; between the ages of ten and fifteen, to 5 per cent. After the second year the mortality from these diseases gradually diminishes: the inference to be drawn is, that the frequency with which Apneumatosis occurs is subject to a corresponding diminution.

The effects produced on the system generally by the presence of Apneumatosis.—Children in whom the lungs are extensively affected with Apneumatosis die of a slow asphyxia, and the manner in which this effect is produced is sufficiently obvious. No respiration, in the mechanical or physiological sense of the word, can take place in the lobules which are collapsed; these portions have become absolutely useless so far as the oxygenation of the blood is concerned; the effect is the same as if the size of the lung had been reduced in a corresponding ratio by complete removal of these portions. It has been shown that the degree to which lobes may be affected is often very considerable in the aggregate; as much as half of the entire lungs has been found to be involved in some cases. The fact that the surface still available for respiration is thus diminished explains the symptoms observed in such cases—the quickened movements of the chest, the distress, and dyspnœa. It is a curious circumstance, and one which of all others should have prevented the older observers from deciding as to the purely inflammatory nature of the lesion in question, that in cases of Apneumatosis a stage soon sets in characterized by great pallidity of the surface, bloodlessness of the integument, and excessive debility. The surface becomes cold and the decarbonization of the blood is thus shown to be reduced to a minimum. The condition of a child in an advanced state of Apneumatosis in fact bears a great resemblance to that of one of the cold-blooded animals. The asphyxia comes on very slowly and gradually, the system apparently accommodating itself to the lowered respiratory function, less blood circulates through the lung, and less in the system generally. All organs suffer; the energy of the muscles is impaired; they no longer contract with force and vigor. Further portions of the lungs become apneumatic from this very circumstance, and when this has reached its extreme limit the patient dies. In the outset there is no congestion in the skin, face, &c., but the asphyxia afterwards observed is of a more chronic, and apparently less congestive form.

The circulation is necessarily greatly affected. The blood ceases to circulate in the lobules deprived of air. The cessation does not take place immediately, but after the lapse of a certain time. The first effect of collapse of the air-cells on the circulation in the lobules affected is to retard the flow of blood—to produce congestion. The blood which at first flows through the part more slowly than usual soon ceases to flow at all. What then becomes of it? Dr. Richardson's experiments have shown that blood will remain for some little time fluid, if preserved from contact with air at rest within the body, but after a time it coagulates. Thus then a second effect, and one occurring later, is coagulation of the blood in the apneumatic lobules. The presence of these clots within the bloodvessels of the lobules, and their various conditions as regards consistence, density, color, &c., explain the difference observed in individual cases, in the appearance of the section of apneumatic lobules. Fuchs[1] describes after Stilling, the changes which the clot ("der thrombus") found within the vessel undergoes as follows: At first it lies free

[1] Loc. cit. p. 75.

within the vessel, but after a time varying in the smaller vessels from two to three days; in the larger, from five to six days, it becomes adherent to the walls of the vessels. Later still it becomes whiter and more dense and contracted, resembling the walls of the vessel in appearance; finally the vessel becomes obliterated, this termination taking place in the small vessels in 20–22 days, in the larger in 30–40 days. The difficulty occasionally experienced in inflating apneumatic lobules is attributed by Fuchs to the contraction which the lung-tissue has undergone as a consequence of the process thus described. The changes which take place in the bloodvessels must after a certain time be an insuperable obstacle to the restoration of the function of the parts involved. An effect of the retardation of the current will be distension of the bloodvessels, and the bulk of the lobules reduced by collapse of the air-cells is still preserved by this distension. Various dynamical effects may thus result. The forcible inspiratory efforts may even produce such distension of the bloodvessels as to render the lobule in question larger than usual. This accounts for the increased size of the apneumatic lobules which, as before stated, is sometimes observed. A further remarkable dynamic effect is the unnatural distension of air-cells in other adjacent portions of the lung; emphysema is in fact almost invariably present in cases of Apneumatosis. Large patches of lung present air-vesicles greatly increased in size.

Symptoms.—The symptoms observable in cases of Apneumatosis are quite peculiar, and more reliance can be placed upon them as indicating the presence of the lesion in question than on the physical signs, unless large portions of certain lobules are affected. When the lungs are extensively affected, the state in which the child is found is generally as follows: There is great prostration and debility, restlessness, and inability to sleep. The temperature of the skin and extremities rapidly falls, and the skin is either very pale or of a dusky hue, the lips have a bluish cast, the eyes are sunken, the skin hangs in folds on the attenuated and wasted limbs, and the child appears prematurely aged, having lost the infantine expression peculiar to a healthy child. The pulse is very quick and often hardly to be felt. There is a constant cry, this being of a whining character, and often very feeble. The respiratory function undergoes important changes, manifest in the altered characters observed. The distinctive feature of the respiration is its *shallowness*, it being very evident that very little air enters and escapes from the chest at each successive movement of the walls. The respiratory movements are much quickened; in a child a year old, the number of respirations in a minute may be as high as seventy or even eighty, and if younger than this higher still. The *rhythm* of the movement is altogether changed, being what is called "expiratory," the interval occurring between inspiration and expiration instead of between expiration and inspiration. This is not pathognomonic of the presence of Apneumatosis, for it may be observed in other cases, but it always coexists with the lesion in question. The dyspnœa in fact is extreme, though not accompanied with that degree of lividity of the face and evident distress usually a concomitant of intense dyspnœa. It is evident also that the dyspnœa is not dependent upon pain in the chest as is the case in pleurisy; the child gives no sign of that kind of suffering which is observed when inflammation of the pleura is present; the suffering is of another character altogether. The cough is very distinctive. In bad cases it can hardly be called a cough at all; the little patient is perpetually making feeble expiratory efforts which produce no effect in evacuating the contents of the tubes, and if the thorax be uncovered, it will be seen that little or no diminution of its bulk takes place during these ineffectual attempts to free the bronchi from the obstructing mucus. These attempts are moreover generally followed by a cry, an expression of impatience at the inadequate result obtained. Nothing can be more significant than the character of the cough, the inefficient nature of which is explained by the fact that there is a deficiency of air in certain parts of the lungs; for as already pointed out each lobule is a miniature lung, and the presence of air is necessary for the production of that jerking expulsive effect constituting a cough. The dyspnœa present in these cases is usually attributed to the presence of mucus in the tubes, but this is not the whole truth; that mucus would be expelled if there were sufficient air behind it, and the patient had, so to speak, the usual control over that air, and could thus drive it out. The dyspnœa observed in bronchitis alone is of a different character, more suffocative, and more productive of congestion; there is more heat of skin and fever present also; but these febrile symptoms disappear in great part when the lungs become extensively apneumatic. The *physical examination* of the chest affords information of a very valuable character. The yielding nature of the thoracic walls in infancy has been spoken of as predisposing to the occurrence of Apneumatosis. That the chest-walls do actually give way during life we have practical proof on watching the movements of the chest during respiration in a child whose lungs are extensively apneu-

matic. The younger the child the more readily does this take place. During inspiration the lower part of the chest is strongly retracted, and the diameter of the chest diminished at this situation, the converse of what is observed in health. Not only do the firmer parietes of the chest thus fall in, following the tractile influence of the diaphragm, but the intercostal spaces become much more manifest, sinking in during the act of inspiration. Conversely, during expiration the same parts may be seen to move outwards to a slight extent. The retraction of the chest-walls during inspiration may be observed when Apneumatosis is not present in consequence of unnatural mobility of the parts, a circumstance previously alluded to, but it is, nevertheless, a sign of considerable importance. The change in the shape and contour of the chest produced by Apneumatosis has been already described.

The results of percussion and auscultation in the young child are in all cases less to be depended on than in the case of the adult. Where the Apneumatosis is extensive, the percussion sound is dull and attended with some degree of resistance; but as it generally happens that the lobules affected are more or less intermixed with others which are healthy, or which even contain a greater amount of air than usual, this dulness on percussion often escapes detection in cases where the aggregate amount of Apneumatosis is considerable. Emphysema, as before stated, is constantly combined with Apneumatosis. The presence of these emphysematous patches will interfere with the results of percussion practised immediately over them in a manner sufficiently obvious. When the whole of one lobe is affected, or when, as it frequently occurred in cases coming under our own observation, the greater part of the lower lobe on either side has lost its gaseous contents, the dulness on percussion has been very marked, and the width of the surface presenting this dulness has increased from day to day under observation. Generally speaking, then, the presence of dulness on percussion is a positive sign, but its absence is, for the reasons just stated, not a negative one. It is to be looked for at the basis of the chest posteriorly, and next in order of frequency at the same position anteriorly.

The respiratory murmur disappears over those parts of lung affected with Apneumatosis, if the disease be widely spread. On the whole, however, it is rare to meet with entire absence of respiratory sound on auscultation, some sounds being still transmitted from deeper parts. We have observed its complete absence more especially in the case of very young infants. The more usual circumstance is that the breath-sound is, when not masked by rhonchi, somewhat bronchial in character, the solidified lung transmitting the sound from the larger air-tubes. It is somewhat rare, however, to meet with cases in which rhonchi, due to the passage of air through mucus, are not audible. With reference to these rhonchi, the most striking character they possess is a degree of coarseness and roughness, not often noticed in the case of the adult. Rhonchal fremitus is only present in the early stage. The true crepitant rhonchus, which is in the adult the chief distinctive sign of the presence of pneumonia, is not heard. Authors have generally accounted for the absence of this pneumonic crepitus in young children, supposed by them to be the subject of "pneumonia," by concluding that the peculiarities of the structure of the child's lung prevented its development; but the fact is, there being no pneumonia, there is, therefore, no crepitus. It is unnecessary further to describe the various kinds of rhonchi which are found to be present in these cases. They depend on the bronchitis present. An important circumstance is the rapidity with which these changes from the normal condition may take place. A large surface of the lung may become solid, causing dulness on percussion and loss of respiratory murmur in twenty-four hours; the limits within which the alterations are observed may also change in as short a time as this. Valleix observes that a dulness of all the posterior part of the right and of the lower third of the posterior surface of the chest may supervene in the space of twenty-nine hours, no sign of this dulness having been present the day before.[1] This is, perhaps, more especially the case in very young infants, for in older children the lung requires to be longer subjected to the necessary process in order that large portions may become apneumatic. The changeableness of the character of the sounds conveyed to the ear by the stethoscope, is of course produced by and follows the alterations in the lung-tissue here alluded to.

The peculiarity of the child's voice interferes with any observations on the intensity of the resonance as felt by the hand, the vocal fremitus.

Such are the symptoms and signs observed in cases where the Apneumatosis is tolerably extensive and well marked. In cases where it is inconsiderable in amount, and scattered over different parts of the lobes, the physical signs may be wholly inadequate to determine its presence, and the general symptoms then afford more information. Cases, indeed, not unfrequently occur in which death having taken place, the Apneumatosis is

[1] Loc. cit. p. 128.

found to be considerable, but having the characters here alluded to, no dulness on percussion, no positive sign of solidification having been detected during life.

The *course*, *duration*, and *mode of termination* of the disease must necessarily vary in different cases. The disease is generally fatal, when involving the lungs to a considerable degree. A child, badly fed, living in a close, confined apartment, breathing constantly a vitiated air, may, if attacked by bronchitis, die in consequence of the Apneumatosis, resulting therefrom, in a short space of time, but the time will vary in different cases. If the child be affected with atelectasis to begin with, the disease is more quickly fatal, but if previously strong and tolerably healthy, its duration is proportionately prolonged. Hooping-cough is exceedingly fatal to very young children, because the bronchitis which accompanies it so readily gives rise to Apneumatosis;[1] but it is well known that it is amongst the children of the poorer classes only that the disease occasions so great a mortality, where, in fact, the predisposing causes before alluded to are allowed to come into operation. The hygienic conditions being favorable, Apneumatosis both less readily occurs, and, when produced, is less likely to prove fatal, than when this is not the case. Unless interfered with, the natural course of the malady is from bad to worse: from the nature of things, the disease tends to intensify itself, and from day to day the affection increases by involving more of the lung substance. As the disease extends, the patient becomes very feeble, unable to cough, or expel the mucus from the tubes, and the quantity of blood in the system seems to undergo a diminution. This is proved by the result of post-mortem examination in chronic cases, and is made evident during life by the pallid, bleached appearance of the patient. After suffering under the symptoms for, it may be, two or three weeks, the death takes place by what is, in reality, a slow asphyxia. The course of the disease may be more rapid, as is sometimes the case in infants who have previously enjoyed a better state of health. These are seized with a severe attack of bronchitis, pervading the smaller as well as the larger tubes, and large portions of the lungs suddenly, or comparatively so at least, become apneumatic and deeply congested; death then rapidly supervenes, the asphyxia being more suffocative and acute in character than in the former case. In both cases, recovery may of course be the result, although the lungs are a long time before their functional activity is completely restored; the seeds of future mischief are some of them left behind, and may subsequently induce a return of the disease: chronic emphysema is a very frequent result of Apneumatosis.

That large portions of lung substance may, within a very short space of time, return to the healthy state, which a short time before had been obviously apneumatic, has been with us matter of observation, and the same circumstance has been noticed by others. The effect of judicious treatment, in restoring clearness of percussion sound and respiratory murmur, is occasionally indeed very marked, and is of itself a sufficient evidence that the dulness which before existed was not due to true pneumonic consolidation of the lung. The cure is often impeded, may often be prevented by the emphysema which coexists; for although the child may have the power of inspiring forcibly restored, the thorax being already filled by the emphysematous distension of certain of the air-cells, no expansion of the apneumatic lobules occurs.

The Prognosis, in a particular case, is favorable if the affection be recent, occurring in a tolerably healthy child, and when the muscular power is not greatly reduced: the hygienic and other conditions in which the patient may be placed, are very important features in the case, as regards the prognosis. In infants, Apneumatosis occurring in connection with hooping-cough is especially fatal; few recover from it when placed, as are the children of the lower orders in large towns, under unfavorable hygienic conditions.

Diagnosis.—Dulness on percussion and bronchial respiration are of most value, where they are present; under other circumstances the altered character of the respiratory movements, the retraction of the chest-walls, combined with the general condition of the patient, and the history of the case, are data on which a diagnosis may be arrived at with tolerable facility.

The diagnostic signs of atelectasis cannot be entered on here. In reference to the other conditions with which Apneumatosis may be confounded, and which it is necessary therefore to distinguish, a few remarks will suffice. *True pneumonia* is very rare in early infancy; the presumption in a particular case will be, therefore, that this condition is not present. The absence of the continued and persistent heat of skin, the absence of the pneumonic crepitus, afford negative evidence tending to the same conclusion. It will

[1] See the author's essay "On Pathology of Hooping-cough" (Churchill, 1855), containing the results of the examination of the lungs after death in nineteen fatal cases of this disease.

be more difficult, however, to distinguish between a case of true pneumonia, in which the inflammatory acute stage has passed away, leaving consolidation of the lung, and one in which Apneumatosis is present. Another condition—*extensive deposit of miliary tubercle* in the substance of the lungs—might present symptoms and physical signs somewhat resembling those observed in the case of Apneumatosis. The history of the case would, however, show that symptoms, as cough, wasting, &c., had been observed for some time previously; and the general condition of the patient, together with this circumstance, could hardly fail to lead to a correct conclusion as to the nature of the case. It may be remarked, however, by the way, that Apneumatosis, as a complication, is often discovered after death in tubercular disease of the lungs. In cases of *pleurisy*, with effusion, there would be dulness on percussion over the lower part of the base of the thorax, together with absence of breath-sound on auscultation, both of which physical signs are present in cases of Apneumatosis; it is to be distinguished from the latter condition, by the greater intensity and width of the dulness on percussion, by the more complete absence of respiratory murmur, observed in the former case. Moreover, in cases of Apneumatosis, it is generally found that the dulness is not limited to one side, as is more frequently the case in pleurisy.

TREATMENT. — Patients affected with Apneumatosis have lost for all functional purposes large portions of the lungs; it is our business to endeavor to restore these portions to their functional activity, and to prevent others from falling into a similar condition. Clear indications for treatment will be found on examining the class of causes, effective in the production of Apneumatosis. As every circumstance which tends to lower the muscular and vital power of the patient favors the production of Apneumatosis, it is very obvious that we are not likely to improve matters by the exhibition of medicines having a lowering character, or by the abstraction of blood, in a case where the child is already too feeble. Setting aside for a moment the consideration of the bronchitis itself, which is or has been present in a particular case, there seems to be no good reason for the employment of depletive or depressing remedies in the treatment of Apneumatosis. There are many reasons against this procedure. The older observers carried their principles into practice: they considered that they had to treat pneumonia, and they treated it accordingly. It is no less incumbent on us to adopt a treatment precisely the reverse.

We are decidedly of opinion that, as a general rule, when an infant is the subject of Apneumatosis, depletion, local or otherwise, is not admissible. The same must be said of the internal administration of tartar-emetic in repeated doses.

One of the chief difficulties to be encountered is the impediment offered to the entry of air, by the presence of mucus, which the child is unable to expel. A primary object is then to assist the respiratory efforts of the patient, at the same time that we endeavor to diminish the excessive secretion of mucus in the air-tubes. Counter-irritation is a valuable means to this end, the degree of which must be adapted to the strength of the patient and the duration of the disease. Mustard poultices are very useful; they can be frequently repeated, and do not produce prostration. Blisters are objectionable from their weakening tendency. We have found frictions of the chest to be followed by markedly good effects, when performed in the following manner:—The hand, lubricated with sweet oil, is to be rubbed tolerably briskly over the whole surface of the chest for ten minutes or a quarter of an hour together, two or three times a day. The result obtained is twofold, a counter-irritant effect is produced, the blood being drawn to the surface and the internal congestion thus diminished, and the movements of the chest are very much facilitated. The movements of the walls of the chest, which the pressure of the hand produced, also aids in the expulsion of the matters blocking up the air-tubes. The warm bath, producing increased action of the skin, is occasionally of service, but is less suited to cases of Apneumatosis than at the outset of an attack of bronchitis; its operation, if continued, or too often repeated, is too weakening. Nothing is more effective in removing the contents of the air-tubes than an emetic, for which purpose ipecacuanha seems to be the best; eight to ten grains of the powder is a proper dose for an infant a year old. Effective, however, as is the emetic in question, it is not to be administered rashly, or under certain circumstances. If the patient be very weak and the disease of some days' duration, the emetic may be unsafe. When not contra-indicated, it may be given once, but is not to be repeated. If it acts efficiently, the object in view is attained, and most patients will not bear its repetition unless after the lapse of a certain time. A little ipecacuanha wine (about ten drops), given in a little syrup, every four or six hours, has the effect of promoting expectoration. The state of the bowels must not be neglected, but mild aperients only are admissible. The food must be extremely simple, but at the same time nourishing. The breast milk for an infant, milk and water for an older child, are quite suffi-

cient in ordinary cases. The case is, however, different when the lungs are extensively affected. Then all our efforts must be directed to the maintenance of the vital powers. Emetics are not safe, even mild expectorants may be improper. Small doses of aromatic spirit of ammonia, or steel wine, or at a later period, the syrup of the phosphate of iron, must be given, and together with the milk diet a little port-wine and water, or brandy-and-water, and weak beef-tea. In dieting young children it is too often forgotten that concentrated food is not well digested, and rich cream and strong beef-tea in many cases act as irritant poisons if taken into the stomach of an infant; great care must be taken to dilute the food given, so that it may be easily digested, or it will do considerably more harm than good.

BRONCHITIS.

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Definition.—An affection of the mucous membrane lining the bronchial tubes, varying from mere hyperæmia of limited extent, to an intense and widely-distributed inflammation, which may involve the deeper structures. It usually gives rise to an increased and altered secretion, containing abundant cells, but in some cases a plastic exudation is thrown out into the tubes. Hence there are two chief forms of Bronchitis, named the Catarrhal and Plastic or Croupous, each occurring as an acute and chronic affection.

Synonyms.—Bronchial Catarrh; Catarrhus Pituitosus; Catarrhus Suffocativus; Angina Bronchialis; Erysipelas Pulmonis; Peri-pneumonia Notha; Bronchite (French); Bronchialentzündung (German).

Acute Catarrhal Bronchitis. Acute Bronchial Catarrh.

Natural History. — Causes. — I. *Predisposing.*—These are due partly to the individual, partly to surrounding external conditions. The following include the most important:—

1. *Age.*—There is no age at which Bronchitis does not occur, but it is far more commonly met with at the extremes of life. It is a very frequent complaint among children, especially during the first two years of life, while dentition is going on, and persons of advanced years are also exceedingly subject to it. The occurrence in children of various affections which tend to have Bronchitis as a complication, and in old persons, of chronic pulmonary, cardiac, and other diseases, will to some extent account for this; while, in addition, they possess less vital power to resist the ordinary exciting causes. The table below shows the rate of mortality at the various ages, during the year 1868, as contained in the Registrar-General's Reports, but it only gives an approximate idea of the relative frequency, as Bronchitis is so much more fatal among the old and young.

MALES.

Under one year	3849	Ten years	40	Fifty-five years	2430
One year	1585	Fifteen years	52	Sixty-five years	3002
Two years	562	Twenty years	85	Seventy-five years	1956
Three years	289	Twenty-five years	331	Eighty-five years	300
Four years	139	Thirty-five years	728	Above	10
Five years	207	Forty-five years	1369		

FEMALES.

Under one year	2969	Ten years	47	Fifty-five years	2316
One year	1585	Fifteen years	61	Sixty-five years	3218
Two years	594	Twenty years	93	Seventy-five years	2158
Three years	243	Twenty-five years	327	Eighty-five years	440
Four years	161	Thirty-five years	615	Above	20
Five years	211	Forty-five years	1267		

2. *Sex* does not seem to influence the number of cases materially. In the year 1868, 16,934 deaths were recorded among males, as compared with 16,324 among females; and it will be seen from the tables that the first year of life gives the greatest difference. Probably men have bronchitic attacks more frequently than women during the adult years, being more exposed to cold, &c.

3. *Habits.*—Unquestionably those who indulge in luxurious and enervating habits, and who wrap themselves immoderately, or live in rooms of a high temperature, produce a relaxing and depressing effect upon the system, and render themselves more obnoxious to slight external influences. The excessive care which many children receive in these respects is certainly injurious; while, on the other hand, their resisting power may be increased by a judicious process of inuring them to various atmospheric changes.

4. *Temperament.*—It is said that those of a sanguineous and lymphatic temperament are more liable to be attacked, but I am not aware of any positive facts bearing out this statement.

5. *State of General Health.*—A constitutionally weak state of the system, or debility resulting from any cause, such as deficient and improper food, or severe illness, predisposes to Bronchitis; while the existence of any positive constitutional disease, such as tuberculosis, rickets, Bright's disease, gout, diabetes, cancer, &c., is still more favorable for its occurrence.

6. *Condition of the Lungs and Bronchi.*—The presence of any deposit in connection with the lungs, as tubercle or cancer, as well as the existence of certain chronic affections, especially emphysema and dilated bronchi, necessarily favors the setting up of Bronchitis. If the mucous membrane has been once attacked, it is rendered more susceptible, and this susceptibility is increased with each attack; hence it is not at all uncommon for a person to suffer every year when the cold weather sets in.

7. *State of the Heart and Circulation.*—Any heart disease that interferes with the return of the blood through the bronchial veins, or anything that causes extra pressure upon the circulation in the bronchial arteries, has a considerable predisposing influence as regards catarrh, and may even excite it. In the manner last mentioned, abundant ascites is said to act by exerting pressure upon the aorta below the origin of the bronchial arteries, and thus throwing an extra strain upon them.

8. *Occupation.*—The occupations which seem to be specially favorable to Bronchitis, are those which involve much exposure to cold and wet, or sudden and marked changes of temperature, and those which lead to the inhalation of irritating particles floating in the atmosphere, such as cotton, steel, charcoal, &c.

9. *Social Position.*—Those among the poorer ranks of society are, for several reasons, very liable to Bronchitis. A large number of cases occur among hospital and dispensary patients.

10. *Climate.*—Bronchitis is very much more common in climates characterized by considerable moisture of the atmosphere, combined with low temperature; and especially where there are sudden and marked variations in temperature. The same observation applies to individual districts; those that are bleak and damp being rarely free from bronchitic cases. It is an exceedingly prevalent disease in this country, and stands very high as a cause of death. In 1867, 40,373 deaths occurred from Bronchitis, being in the proportion of 1902 to every million persons living, and of 86,554 in every million deaths. In 1868, the number of deaths was 33,258, giving a proportion of 69,765 per million deaths. The mean rate of mortality for 15 years, from 1850 to 1864, was 1344·4 in every million living. It occurs in different districts with very variable frequency. The following statistical summing up gives, approximately, the proportion of deaths from Bronchitis to the number of inhabitants in the different districts during the year 1868:—

London, 1 in 442·3; South Eastern Counties, 1 in 805·01; South Midland Counties, 1 in 834·7; Eastern Counties, 1 in 987·5; South Western Counties, 1 in 844·8; West Midland Counties, 1 in 665·03; North Midland Counties, 1 in 876·2; North Western Counties (Cheshire and Lancashire), 1 in 379·5; Yorkshire, 1 in 541·5; Northern Counties, 1 in 774·8; Monmouthshire and Wales, 1 in 955·4.

11. The foregoing statistics prove that Bronchitis is much more prevalent in large towns and cities than in country places, and the reasons for this will be obvious. The same remark applies to the fact that the poorer districts of cities and towns furnish by far the greater number of cases. Places where extensive manufactures are carried on, loading the atmosphere with various irritating materials, have also always a considerable proportion of cases.

12. *Season.*—By far the largest number of cases is met with during the colder months of the year, extending usually from the end of autumn, through the winter, into early spring. Much, however, will depend on the kind of weather that is experienced. The number of cases was considerably less in the year 1868 than in 1867, on account of the comparative mildness of the weather; a sudden change in the weather is very likely to bring with it numerous bronchitic at-

tacks, and the prevalence of north-easterly or easterly winds has a similar influence.

II. *Exciting.*—1. In the great majority of instances, cold, in some form or other, acts as the immediate exciting cause of Acute Bronchitis. It may produce its effects in various ways: thus, an attack may arise from the breathing of cold air, especially if at the same time loaded with moisture, and particularly if there has been a sudden change from a warm and dry atmosphere; emerging from a warm room into a cold atmosphere, particularly when in a state of perspiration, and sitting in a cold draught, contribute numerous cases. Wearing an insufficient amount of clothing in cold weather, and exposing the upper part of the body; neglecting to change damp clothes, or having wet feet; sleeping in damp beds, &c., are all frequent causes. Infants who drivel constantly and profusely, so that the garments covering the chest are always moist, are said to be very subject to Bronchitis. In most of the instances where the cause cannot be traced, it is probable that the patient has "taken cold" in some way or other. The modes in which cold produces its injurious effects appear to be, first, by causing local irritation of the bronchial mucous membrane, and disturbing its circulation and nutrition; secondly, by acting upon the system at large in some way or other not understood, the Bronchitis being only a part of a general disturbance.

2. On the other hand, sudden great heat after cold, *e. g.* passing from the night air into a very hot room, is said sometimes to cause Bronchitis, but this is difficult to substantiate.

3. Another important exciting cause is the *direct action* of various irritants upon the mucous membrane lining the air-passages. This may arise from certain conditions of the atmosphere inhaled, such as a very high or low temperature, or from its containing any irritant gas or vapor, *e. g.* sulphurous anhydride, chlorine, ammonia, &c.; or having certain minute particles floating in it, such as dust, steel-filings, charcoal, cotton, flour, &c., and in the same category may be included those cases of Bronchitis that result from inhaling certain vegetable substances, viz., the powder of ipecacuanha and the emanations from hay. "London fogs" undoubtedly act in this way, and, it is said, also miasmatic productions. The blood remaining in the tubes after hemorrhage, and unhealthy secretions from cavities in the lungs, &c., coming into contact with the mucous membrane, may excite inflammations.

4. Certain morbid conditions of the blood are very prone to give rise to Bronchitis. To this is attributable that form which complicates certain febrile affections, especially typhoid fever and measles, and, less commonly, scarlatina, smallpox, hooping-cough, diphtheria, typhus fever, &c. It is particularly liable to occur in the eruptive fevers, if the eruption comes out imperfectly, or suddenly recedes. Neglect of proper precautions during convalescence from these affections, is very apt to lead to dangerous Bronchitis. The poison of syphilis, as well as that of gout and rheumatism, also produces this affection, and it is particularly prone to occur in the last two diseases if sudden metastasis takes place. The state of the blood must also account for those cases that are said to result from the rapid disappearance of the eruption of erysipelas, the suppression of long-continued discharges, whether natural or morbid, and the too rapid cure of an old-standing skin disease. Iodine taken internally sometimes causes bronchial catarrh, evidently due to its presence in the blood.

5. Various deposits in the lung may not only predispose to, but actually excite inflammation of the mucous membrane. It is constantly met with more or less when tubercle or cancer is present, and is then prone to be localized.

6. In connection with influenza, Bronchitis occurs epidemically, without our being able to trace it to any special cause. At certain times of the year a large number of persons are often simultaneously attacked, so that the complaint may almost be said to be epidemic, but this is due to obvious atmospheric conditions already alluded to.

Symptomatology.—The clinical history of Acute Bronchitis varies considerably under different circumstances, and an attack may range from a slight "cold in the chest," to one inducing suffocation and gravely affecting the system at large. The chief reasons for these variations are to be found in the age, general condition, and health of the patient, the previous state of the lungs, the extent of mucous membrane involved, and the immediate cause of the disease.

In practice, the following forms are met with:—

I. Acute Primary or Idiopathic Bronchitis, the result of "cold," there being no previous evident lung affection:—
   1. Involving the larger and middle-sized tubes only, and not extending into the smaller tubes.
   2. Implicating the smaller tubes—"Capillary Bronchitis."

II. Secondary Bronchitis:—
   1. In connection with the exanthemata.
   2. In certain blood-diseases.
   3. After chronic lung and heart affections.

III. Mechanical:—
1. Hay-asthma, &c.
2. That resulting from mineral and other irritant particles.

IV. Epidemic.

The primary forms it will be necessary to describe at some length, but the others will call for only a few remarks, pointing out in what respects they differ: whereas Epidemic Bronchitis it will not be requisite to allude to again, as it belongs to Influenza.

1. Acute Idiopathic Bronchitis, not extending beyond the middle-sized tubes.

Invasion.—This is almost always characterized by the occurrence of symptoms of so-called "catarrh," in consequence of the mucous membrane lining the nasal cavities and their communicating sinuses being affected, and, frequently, the conjunctivæ. There is an irritating watery flow from the nose and eyes, and a feeling of fulness, heat, and soreness in these parts, with frequent sneezing fits. Frontal headache exists, due to the state of the frontal sinuses. The upper and back part of the throat often feels sore and rough, and frequent attempts are made to clear it from mucus. There is generally uneasiness over the larynx, and the voice is more or less hoarse and husky, indicating that the mucous membrane here is also implicated. Not uncommonly the catarrh seems to spread regularly downwards along the respiratory tract, beginning in the nose. In some instances the larynx is alone involved at first, while in others the bronchial mucous membrane seems to suffer from the outset, the upper part of the tract escaping; but this rarely happens in the form now under consideration. Along with these local symptoms there are others of a general character, almost always present more or less. The patient feels chilly, or there may be even rigors in a sensitive person, but they are never of marked intensity, and several occur at irregular intervals, not a single prolonged fit of shivering. Their severity is usually in proportion to the extent of the inflammation. In the intervals between them the patient feels hot, but the temperature is not raised, as evidenced by the thermometer, or only slightly. The pulse is often somewhat increased in frequency. The limbs and joints, or even the body generally, are affected with pains of an aching, contused character, and there is a general sense of fatigue, languor, and want of energy, the patient experiencing a disinclination for any occupation, mental or physical. He is heavy and drowsy, but sleep is often restless and uneasy. There is frequently a furred tongue, anorexia, and constipation, evidencing that the alimentary canal also suffers. In nervous, irritable persons, and in the older children, slight delirium is said to be present sometimes; while in younger children, especially during the period of dentition, and in those who are weakly, a fit of convulsions may usher in the attack.

After the initiatory symptoms have lasted a brief but variable time, those characteristic of the bronchial inflammation set in. They may be very slight, or tolerably severe, and are "local" and "general."

Local.—Various unpleasant or painful sensations are experienced behind the sternum, especially towards its upper part, and in the supra-sternal notch. These are, more or less heat, sometimes reaching to actual burning, and a sense of soreness or rawness, which may amount to considerable pain—as a rule, however, it is not severe, when the patient is quiet. A deep inspiration aggravates these feelings in a variable degree, while the act of coughing gives rise to much positive pain, of a raw, aching, burning, or tearing character. This is not only complained of behind the sternum, but also radiates towards the sides, as if in the course of the primary bronchial divisions. If the cough is severe and frequent, a feeling of soreness or aching is soon felt all over the chest, but especially towards its sides, and the base where the abdominal muscles are attached. A very unpleasant irritation or tickling is also experienced above and behind the sternum, which excites the cough. Tenderness over the sternum is often present, the skin feeling sore on percussion. These sensations vary much in intensity, and may merely amount to a diffused feeling of slight heat and uneasiness over the front of the chest, but most marked behind the sternum.

Dyspnœa is not a prominent symptom, but the frequency of the respirations is often somewhat increased, and the pulse-respiration ratio may be more or less altered. The act of breathing is labored in many cases, and there is always a sense of oppression, weight, and tightness about the chest, especially towards its upper part.

Cough is one of the earliest and most striking symptoms. It is loud, and usually a little hoarse at first, owing to the larynx being affected; otherwise it is free from hoarseness. It comes on in paroxysms, either spontaneously, or from any slight irritation, as inhaling cold air. These last a variable time, and cannot be suppressed. They increase in frequency as the disease advances, and often become very violent, especially after a sleep, and on first lying down at night. There is no expectoration at the outset, the cough being hard and dry, but afterwards each

fit ends with expectoration. It is evidently due at first to the abnormally irritable condition of the mucous membrane, and subsequently to the presence of excessive and altered secretion in contact with it, which is itself probably of an irritating nature at first. The *expectoration* varies in its characters at different periods of the case. At the beginning it is small in quantity, thin and watery in appearance, almost transparent, but frothy, and has a saltish taste. The changes it undergoes are: increase in quantity to a variable degree; diminution in transparency, becoming at last almost or quite opaque; increase in consistence and viscidity; diminution in frothiness; loss of taste; and change in colors. Thus, it generally passes through stages of viscid, semi-transparent, slightly yellowish or grayish, frothy mucus, to a muco-purulent or purulent-looking substance, nearly opaque, of a grayish-yellow, yellowish, or a greenish-yellow color, and but slightly aërated. It usually runs together into one mass, but a distinct, nummulated form of sputum is sometimes met with, which is thoroughly opaque. Its tenacity and adhesiveness may be so great as to make it stick closely to the vessel containing it, and to admit of its being drawn out into threads. Sometimes it is quite ropy and gelatinous. A few streaks of blood may be seen, especially at the early period. Should an extension of the inflammation take place, this is indicated by the expectoration once more assuming its early characters in part. As the sputa become altered they are more easily expelled, especially from the larger tubes, and hence the cough abates and is much less painful. *Microscopical characters.* — In the early stage, pavement, columnar, and ciliated epithelial cells are seen, with a few imperfectly formed cells. Later there are abundant young cells, discharged from the surface of the mucous membrane, many resembling the so-called exudation corpuscles, and at last pus cells. Molecular and granular matter is seen in quantity; a few blood disks may be present, and occasionally amorphous, fibrinous coagula. Crystals of oxalates, &c., are sometimes visible.

General.—In the slighter cases there are no notable signs of general indisposition, but if the attack is at all severe, the system gives indications of being affected. More or less febrile reaction occurs, the pulse becoming frequent, but rarely above 100; at the same time in a healthy person being strong and full. The skin feels hot, but not acridly; and it may soon be moist. The actual temperature is never very high, but it follows the ordinary rule of increasing in the evening. If the fever precedes the bronchitic symptoms, it is said to be notably more severe. Slight rigors may continue throughout the attack. The tongue is generally more or less furred, but moist; and there is some thirst, with loss of appetite. The bowels are mostly confined. Vomiting may occur, especially after a severe fit of coughing. The urine presents the ordinary febrile characters in a varying degree: the urea and pigments are increased, but the chloride of sodium may be notably diminished. There may be heat during micturition, probably from slight catarrh of the urethral mucous membrane. A sense of languor and weakness continues throughout the case, and there may be considerable depression, quite independent of, or out of proportion to, the febrile state.

A favorable case of this description may run its course in three or five days, or may last two or three weeks, according to the number and size of tubes involved, the depth of the inflammation, and the state of the patient. The fever, if any existed, soon abates, and the local symptoms gradually subside, the cough, however, often holding on for some time, especially in the mornings, on account of the secretions having accumulated. These cases do not always end in recovery. In very old patients, and in those weakened by disease or want, fever of an adynamic type is apt to be present from the first, or to follow sthenic fever, especially if this has been severe. Then there is great debility, a quick, feeble pulse, a dry, brown tongue, and low delirium. Or it may happen that the patient is unable to expel the secretion formed in the tubes, which therefore collects and tends to pass into the smaller tubes, thus possibly causing inflammation in them, or blocking them up, and leading to slow suffocation. In young infants, even a very little bronchial catarrh may lead to serious results, especially if they are feeble and ill-nourished, or are the subjects of rickets. They are unable to expectorate, and thus the fluids accumulate, and a large tube, or a number of tubes, become blocked up, collapse of portions of the lung resulting from this. Under any of these circumstances a fatal result may ensue. In a comparatively few instances this form of Bronchitis remains as a chronic affection, particularly if it implicates the deeper structures of the tubes.

2. ACUTE BRONCHITIS, involving the minute tubes. *Capillary Bronchitis.*—This is a very dangerous condition, even in a healthy and robust adult; but it is peculiarly grave when children, old people, or very debilitated persons are the subjects of it, among whom it occurs with considerable frequency, in the order in which they are mentioned. This results partly from the great interference with the

blood-aëration that it involves, partly from the accompanying fever, which has a strong tendency to become adynamic. In the majority of cases it is preceded by symptoms of inflammation in the larger tubes, or the whole tract may be more or less involved simultaneously or very rapidly. In some instances the smaller tubes seem to be alone affected from the first. The early symptoms may be those already described, or well-marked rigors, severe headache, and sickness may usher in the disease. There may be only slight or very considerable pain behind the sternum, but it is absent if the capillary tubes are alone implicated. Children and aged persons often do not appear to suffer any particular pain. There is always, however, much aching and soreness about the base of the chest and epigastrium, owing to the severe spasmodic contractions of the expiratory muscles during the fits of coughing. This is aggravated during each paroxysm, and patients frequently sit up or bend forwards while they cough, in order to release their abdominal muscles, at the same time pressing their sides, so as to give them support. *Dyspnœa* always attracts attention, but its degree varies materially. It may be limited to accelerated and somewhat laborious breathing, with a feeling of constriction and oppression across the chest; or the respirations may be extremely frequent and hurried, attended with violent efforts during inspiration, and an urgent craving for air. There may be constant or paroxysmal orthopnœa, the latter supposed to be due either to spasm of the bronchial tubes, or to the sudden blocking-up of a large tube with secretion. The absolute frequency of the respirations may rise to 50 or more, and being increased out of proportion to the pulse, the normal ratio is disturbed, being sometimes 2·5 to 1. Wheezing and whistling sounds are often present, audible at a distance, and attending both inspiration and expiration. *Cough* occurs almost continuously, but it also comes on in extremely violent, prolonged, and distressing paroxysms, during which the face becomes turgidly red or purple, the veins swell, and the arteries throb. *Expectoration* is effected with much difficulty, owing to the secretion being exceedingly tenacious and sticky, and having to be expelled from the smaller tubes, while the muscular fibres of the bronchi, which normally assist expectoration, are probably paralyzed in many cases. The sputa are scanty at first, but soon increase greatly in quantity, becoming chiefly muco-purulent, yellowish-green, or bright green and opaque; or extremely viscid, glutinous, and ropy: they may partly retain the form of the smaller tubes, and minute cylindrical casts, consisting of fibrinous exudation, may be present, or irregular particles of the same substance. Some frothy, lighter mucus from the larger tubes is mixed, more or less, with the above. Children do not expectorate, or rather they swallow what they bring up, but some of it may be obtained for examination by wiping the base of the tongue with a handkerchief after a fit of coughing.

The *constitutional* symptoms are always severe. At first there is ordinarily considerable fever, which, in the case of healthy adults and plethoric children, is of the sthenic type, but in the aged and feeble is prone to be asthenic from the outset, or speedily to assume this character. The pulse is frequent, quick, and generally full. The skin is hot, but may be dry or moist. The temperature may reach 103·5° Fahr. in the evening, when it is often 2° in excess of the morning. Flushing of the face, and headache, increased by the cough, are commonly present. Pains are complained of in the trunk and limbs, and there is a feeling of great weakness and exhaustion. Wasting occurs in proportion to the fever and to the interference with sleep, which is generally great. Loaded tongue, anorexia, constipation, are usually marked symptoms, and there may be much sickness. The urine, in addition to being febrile, is sometimes slightly albuminous temporarily, and it is said a trace of sugar is occasionally present. Chloride of sodium may be almost totally deficient.

The symptoms, both local and general, may, after reaching a certain point, subside, and gradual recovery take place; but in the majority of cases this favorable result does not occur. Indications of more or less imperfect aëration of the blood are observed in almost every instance, owing to the impaired respiratory process; but in many, especially children, this constitutes the main source of danger, and leads to a fatal issue. Gradual suffocation is brought about, and the blood becomes charged with carbonic anhydride, while its oxygen is proportionately deficient; and hence the various organs essential to life are supplied with blood which cannot maintain their functions. When this happens the face assumes at first a turgid, bloated, and more or less red, dusky, or livid appearance, but it soon becomes generally pale, while the lips, tip of the nose, malar prominences, and external ears deepen in their lividity, which contrasts strongly with the surrounding pallor. The veins of the head and neck swell. The surface generally is also cyanotic in a variable degree, particularly the fingers and toes, this appearance being very marked under the nails. The feet and hands may swell from œdema, which may extend even to the trunk. The temperature rapidly falls, especially that of

the extremities. Cold, clammy sweats break out about the face and upper part of the body, and then spread universally. Rapid exhaustion of the vital powers follows, and the patient allows his head to sink on the pillow or droop in any direction. The pulse becomes greatly accelerated, weak, small, and compressible, and at last often irregular. Intense thirst is complained of. Cerebral symptoms set in early; the mind wanders, and in many cases a persistent desire to get out of bed is manifested. I have seen this well marked in some adult cases. There is at first perpetual restlessness, with a deeply anxious expression of countenance, and great dread; but these conditions soon change, and the patient becomes more and more indifferent, with dull and heavy eyes, then falling into a drowsy state, out of which for a while he starts suddenly, but which gradually deepens into permanent stupor, and finally complete coma, which precedes death. Convulsions may occur before the final coma. The cough ceases after a time, the power as well as the desire of expectorating being lost. Breathing becomes much quieter, but very hurried and shallow. As a consequence, the secretions gradually fill up the air-tubes, and thus are produced rhonchal sounds, audible at some distance, which change into gurgling as the fluids rise into the larger tubes. The expired air is cool.

The urine is greatly diminished in quantity, and may be totally suppressed.

Death sometimes occurs suddenly, before the brain is much involved, owing to the blocking up of a large bronchus with secretion; which is most liable to happen in young children.

Instead of the symptoms just described, those characteristic of adynamia may arise, especially in the aged or feeble, and where the fever has been excessive. The tissues are rapidly consumed, and the blood loaded with the resulting impurities. The tongue becomes dry and brownish, with a red tip and margins, or a thick dark fur may form upon it behind. The pulse is very frequent and small, often irregular and uncountable. Low, wandering delirium sets in, succeeded by coma. Profuse, clammy sweats break out, the extremities becoming cold. There are no marked cyanotic symptoms at first, but owing to the condition of the sensorium the need of expectoration is not felt, and thus the secretions accumulate in the tubes, this being aided by paralysis of the muscular fibres in the walls of the bronchi, which finally leads to slow suffocation.

In many fatal cases, two classes of symptoms above described appear to be combined more or less. Certain complications may occur greatly increasing the danger, the chief being lobular or more extensive pulmonary collapse, acute emphysema, lobular or lobar pneumonia, congestion ending in œdema, and pleurisy.

The term "Peripneumonia Notha" is applied rather vaguely to some cases of Bronchitis. With some it is synonymous with Capillary Bronchitis; but it seems more appropriately to refer to the disease occurring in an old or enfeebled subject, after some chronic malady, with febrile symptoms at first, but signs of adynamia, and deficient aëration of the blood setting in early

3. Bronchitis occurring in connection with the Exanthemata.—Some of these are never free from a certain amount of bronchial catarrh, more especially typhoid fever and measles, and it may constitute the chief source of danger. It is very apt to come on insidiously without pain or difficulty of breathing, and scarcely any notable cough or expectoration. In short, physical signs may alone indicate the existence of the catarrh. On the other hand, the attack may be exceedingly severe, and mask for a time the nature of the fever. In measles constantly, and in scarlatina usually, coryza exists at the outset, but in the other fevers it is commonly absent. The Bronchitis may come on early or late in the case. Should it be extensive, or the patient be much weakened, it is a serious complication, and may rapidly lead to a fatal result. It is important to bear in mind the non-occurrence of subjective symptoms, and that it is necessary to employ physical examination of the chest at frequent intervals.

4. Bronchitis in connection with Blood Diseases.—In some instances it may be considered as truly secondary, depending immediately upon the poisoned state of the blood; but in others this only acts as a strong predisposing cause. Here again the disease is prone to come on insidiously, without any marked symptoms, and also to last a long time, often becoming chronic. The expectorated matters are said to contain some of the poisonous materials which accumulate in the blood, such as sugar in diabetes, urea in Bright's disease, uric acid in gout, &c.

5. Bronchitis in connection with Chronic Lung and Heart Diseases.—When occurring as the result of deposits in the lungs, especially tubercle, Bronchitis is very commonly localized to their immediate neighborhood, and hence is often confined to the apex. It is not preceded by coryza, and there are usually no marked symptoms. Should there have been previous chronic Bronchitis, espe-

cially with emphysema, upon which an acute attack has supervened, dyspnœa is always considerable, and is liable to become extremely urgent, with early and grave cyanotic signs, particularly if the heart is also affected. Œdema of the extremities, or even of the trunk, readily occurs. Pain is frequently absent, but the cough is distressing and severe. In cases of emphysema, the expectoration is at first very frothy, as well as abundant. Even a slight amount of acute Bronchitis, superadded to extensive chronic catarrh with emphysema, brings with it much danger.

6. MECHANICAL BRONCHITIS.—The various irritating substances, such as charcoal, &c., when inspired, at first give rise to slight but repeated attacks of acute catarrh, without coryza, not attended with pain or fever, but having an exceedingly irritable and frequent cough, without much expectoration, which contains some of the particles inhaled. The condition soon becomes chronic, and will call for a few further remarks when Chronic Bronchitis is treated of.

Under this head it will be necessary to notice briefly those cases in which bronchitic symptoms are brought on by the inhalation of certain vegetable matters, the most important being "hay-asthma," or "hay-fever." The symptoms of bronchial irritation are prominent. There are frequent and severe paroxysms of coughing, but there is generally no expectoration, or at most, a small quantity of clear, thin, watery mucus. Breathing is much oppressed, and there is often considerable soreness behind the sternum. Marked coryza occurs, and other indications that the whole tract of the respiratory mucous membrane is involved; much general languor and want of energy is experienced, but fever is absent. Only a few, possessing a special idiosyncrasy, are liable to this complaint, and they are attacked on the slightest exposure to the exciting cause, and sometimes apparently even without this; hence they usually suffer every hay season. The symptoms come on suddenly, and are severe almost from the outset; they may last from two to six weeks or more.

Ipecacuanha produces very similar effects, and I am acquainted with a case which recently occurred, in which a severe attack resulted from smelling for a moment a bottle containing ipecacuanha powder, as an experiment, the patient having previously suffered in a similar way.

Physical Signs:—1. *Inspection.* (*a*) *Form and size of chest* rarely altered, but if the lungs are greatly distended, the chest may be somewhat enlarged, but equally so throughout. (*b*) *Movements* more frequent and more rapid than in health, in proportion to the amount of dyspnœa. Expiration is evidently difficult and ineffectual, and hence protracted. In most cases the abdominal movements are in excess of the thoracic, but if there is extensive accumulation in the tubes, the upper costal movements become considerably the more marked, and elevation is often in excess of expansion. Much, however, will depend on age, sex, the extent of the tubes involved, &c. In children, particularly if they are subjects of rickets, signs of more or less imperfect inspiration are commonly observed. The epigastrium, ensiform cartilage, and contiguous rib cartilages sink in during each inspiratory act, the lower ribs are drawn in laterally, and the supra-clavicular regions become deeply hollow. Niemeyer mentions another sign of the same condition, viz. "prominence of the supra- and infra-clavicular regions, with feeble respiratory movements."

2. *Palpation.*—In addition to the signs mentioned under "Inspection," palpation reveals usually "rhonchal fremitus," of variable quality and extent. It may be felt over a large area, without a large number of tubes being necessarily involved; but should it continue thus for some days, it indicates widely-spread Bronchitis. The presence of this fremitus shows that some of the more superficial tubes are affected. It generally accompanies both inspiration and expiration, but it is often more marked during one or other act. A cough may cause it to disappear, or alter its position. Stokes states that it is more marked in females, and over the lower and middle part of the chest. It may be felt only in front, and over the upper part of the chest. This sign is of great importance in the physical examination of very young infants. Vocal fremitus varies widely, and cannot be relied on. Tussive fremitus is often well marked.

3. *Percussion.*—In most cases the area and amount of pulmonary resonance are not obviously altered. It not unfrequently happens, however, especially in children, that owing to the air-vesicles and small tubes being permanently distended in consequence of obstruction, the resonance is in excess, both in extent and degree, and is not diminished after expiration in the normal proportion. Rarely, a certain amount of deficiency in tone may be noticed over the base of the lungs posteriorly, owing to great accumulation of secretion, congestion with œdema, or lobular collapse; and the same may be observed in other parts of the chest, if collapse has resulted from obstruction of a large tube, or even extensively should the main bronchus be pressed upon by enlarged glands, which is said to happen

sometimes. In infants, a sound resembling the "*bruit de pot fêle*" may often be produced by sharp percussion, especially during expiration, variable in its site.

4. *Auscultation.* (*a*) *Respiratory Sounds.* — These vary considerably in different parts of the chest. Where the tubes are free the sounds are loud and exaggerated, and this is usually the case towards the upper part of the thorax. Over the affected regions they are weak, and may become totally suppressed, owing to the narrowing or complete closure of the tubes by thickened membrane and secretion; or temporarily, from spasm of the muscular fibres. Their quality is always harsh and coarse, and expiration is prolonged. In the early stage the sounds seem dry, but later on certain rhonchi are mingled with them, by which they may be completely masked.

(*b*) *Adventitious Sounds.* — These include the various "rhonchi" produced by the air passing through tubes containing fluid, or diminished in calibre by thickened mucous membrane or spasm. They vary with the nature and quantity of the fluids, the size of the tubes in which they are originated, &c., and are divided into "dry" and "moist." The former comprise the "*sonorous*," which are very low-pitched and grave in tone, resembling the sound of snoring generally, but sometimes of rubbing or other quality, often heard extensively, and giving an impression of superficialness in their origin; and the "*sibilant*," which are high-pitched, and may be musical, hissing, or whistling. If the sibilant rhonchi are extensively heard, it indicates that the smaller tubes are affected. Occasionally "clicking" sounds of dry character are observed. The "moist" rhonchi are all more or less bubbling, being caused by the passage of air through fluid. They vary much in size, quality, and pitch, according to the quantity and consistence of the fluid and the dimensions of the tubes in which they are produced, and the varieties are named "mucous," "submucous," "subcrepitant," &c. Occasionally they have a "rattling" character.

It will be readily understood that these rhonchi are variously combined, and are heard in different parts of the chest, according to the seat, extent, and stage of the Bronchitis. Generally they exist on both sides, though not to the same degree, but may be localized to a part of one lung. At first the "dry" may alone be present, but the "moist" are soon added, and frequently both forms are perceptible from the first. The "moist" are usually most marked behind and towards the base of the lungs. All kinds are liable to change their sites, as well as to disappear for a time, sometimes suddenly, either from the secretions having been driven out of the tubes, or because these have become thoroughly blocked up. A strong cough will often disperse many of them. These remarks are especially true with regard to the sonorous and sibilant rhonchi.

When Capillary Bronchitis is present, abundant and very minute bubbling rhonich are heard towards the lower part of both lungs, accompanying inspiration and expiration, and completely hiding the breath-sounds; while higher up they are larger, and the respiratory sounds are perceived, altered in quality. This may be partly the result of gravitation, but very extensive and minute rhonchi indicate that the smaller tubes are themselves implicated.

(*c*) The action of the heart sometimes causes rhonchal sounds.

Vocal resonance is not materially altered in either direction. The cough is generally very loud, and gives rise to a number of rhonchal sounds.

5. *Position of Organs.*—As a rule this is normal, but if the lungs are greatly distended the diaphragm is depressed, and with it the liver and spleen somewhat. The heart is said to be pushed downwards and to the right. In some cases which have recently fallen under my notice in the post-mortem room, the heart was so placed that its right border lay almost horizontally on the diaphragm, and its apex was outside the left nipple-line, occupying a similar position to that described by Niemeyer as occurring in emphysema.

Duration and Termination. — In the milder forms the duration varies from four or five days to three weeks or more, but a case is usually convalescent under nine to twelve days. In fatal cases of Capillary Bronchitis death generally occurs in a few days, but it is difficult to lay down any certain average. Walshe gives from the sixth to the eighth day for children, from the tenth to the twelfth for adults. Convalescence is not thoroughly established for some time in cases that recover, but generally begins under three weeks. The clinical terminations are: (*a*) complete recovery, (*b*) death, (*c*) transition into the chronic state. Relapse may occur, or an extension of the Bronchitis; but this is not common. As already mentioned, it is an affection very liable to recur. It should be mentioned that it may leave behind it permanent emphysema, or may be the foundation of certain forms of phthisis. Niemeyer believes that extensive acute bronchial catarrh is the most common cause of "galloping consumption."

Diagnosis.—The characteristic symptoms of ordinary Bronchitis are the various sensations behind the sternum, a

greater or less sense of oppression, often amounting to dyspnœa, with wheezing, cough, and expectoration, having the characters already described. The previous catarrh, as well as the general symptoms, with slight but repeated rigors, and absence, or comparatively small degree, of fever, are also important. The more significant physical signs include absence of dulness, or any material alteration in the vocal fremitus or resonance; the characters of the breath-sounds, but especially the presence of the various rhonchi, as indicated by palpation and auscultation. In the majority of cases there is no difficulty in arriving at a proper diagnosis, but doubt may arise in some instances. It will be necessary to notice briefly the special diagnosis of Bronchitis from certain other affections.

It cannot be decided in the earlier stage of hooping-cough, whether the case is not one of Bronchitis. Subsequently the paroxysmal nature of the attacks—with the peculiar cough and expectoration, often followed by vomiting—is sufficiently characteristic of hooping-cough. However, it may be complicated with Bronchitis, which is then revealed by its physical signs.

In some children the breathing of Bronchitis may at first somewhat resemble that of croup, the cough being at the same time hard and ringing or husky, and the voice affected. The evidences of Bronchitis in such a case are, the presence of catarrh; breathing less affected, and not truly stridulous, but wheezing; fever absent or slight; the cough is soon moist, and expectoration may be obtained by wiping the base of the tongue, which does not contain any shreds of membrane. Physical examination also shows the existence of rhonchi, &c.

Laryngitis in the adult is distinguished by its own special symptoms, which are localized in this part, and by the absence of the chest-symptoms and physical signs of Bronchitis.

Pneumonia occurring in the adult is usually easily diagnosed from Capillary Bronchitis by attention to the following points. There is a single, prolonged, and severe introductory rigor, followed by intense fever, with a burningly hot and dry skin. A sharp pain is experienced in the side, and the cough is less marked, being usually attended with rusty expectoration. The pulse-respiration ratio is more disturbed, but the sense of dyspnœa is less, and there are no cyanotic appearances. Physical examination discloses dulness, increased vocal fremitus and resonance, crepitant rhonchus, and bronchial or tubular breathing, in pneumonia, usually limited to one base. When an acute attack supervenes upon Chronic Bronchitis, it may give rise to dulness at one base, and respiration may become high-pitched, bronchial, or even diffused blowing, but it is never actually tubular, and vocal resonance is not of a metallic and sniffling character. In such a case attention must also be paid to the symptoms. From lobular pneumonia occurring in children the diagnosis is often difficult. In this affection frequently no dulness can be observed, or it may be present in Bronchitis from collapse. In the latter the moist rhonchi are much more diffused, and of larger size; at first they are generally limited to, and throughout are most marked at the bases, whereas in lobular pneumonia they are scattered irregularly. Tubular breathing is not heard in Bronchitis. There is less fever, and the skin is not acridly hot, being often moist. The respirations are more frequent in lobular pneumonia, but the sense of dyspnœa is much less as well as the asphyxial appearances and general anxiety.

The symptoms and physical signs of pleurisy are so totally different from those of Bronchitis, that it does not appear necessary to say anything as to their diagnosis.

When a child is attacked with bronchial symptoms, it is sometimes difficult to determine at first whether they constitute the entire ailment, or are associated with one of the exanthemata. The amount of fever as evidenced by the thermometer, and the special symptoms premonitory of the various fevers, must be the guides until the eruption appears. The same applies to typhoid fever, at any age, which may be at the early period masked by the bronchial catarrh if its ordinary symptoms are not prominent. The thermometer will prove of great value in any such cases.

From the various forms of acute phthisis, Capillary Bronchitis may be distinguished by the following characters. The fever is much less, and consequently the temperature is considerably lower; the pulse-respiration ratio is less perverted; signs of asphyxia set in; there is free expectoration of a muco-purulent character. There are abundant dry and moist rhonchi, the latter being most marked below. In one form of acute phthisis there are signs of consolidation, followed by those of cavities. In the true tubercular miliary form there are hardly any physical signs except scattered rhonchi, which are most abundant towards the upper part of the lung. The dyspnœa is very great, and there is violent fever.

PROGNOSIS AND MORTALITY.—As will be seen from the tables given when considering the causes of Bronchitis, this is a disease attended with much danger, especially if it be extensive. Its prognosis must be guided by the following circum-

stances: 1. *Age.* The mortality is far greater among children, especially young infants, and the aged, than in adults. 2. *State of Health.* The danger will be increased in proportion as this is below par, and particularly if there is any positive disease present, either acute or chronic. 3. *Extent of inflammation.* If both lungs are widely affected with Capillary Bronchitis, the prognosis is grave, even in healthy adults. 4. *Previous state of the lungs and heart.* Any chronic disease of these organs will seriously aggravate the danger, but especially extensive emphysema, with dilatation of the right cavities of the heart. 5. *Special symptoms.* Those of evil import are—excessive expectoration, of thick and viscid character, and brought up with difficulty; suppression of cough, with accumulation of secretion in the tubes; very frequent and rapid breathing, with signs of asphyxia; quiet and shallow breathing in an otherwise bad case; evidences of imperfect inspiration in children; very frequent and feeble pulse; adynamic symptoms; the head being kept on a low level from the first in a grave case. 6. *Presence of complications.* Those that add generally to the gravity of the case are—collapse; pneumonia, lobar or lobular; congestion with œdema; acute emphysema; pleurisy; gastric or intestinal catarrh. 7. *Epidemic character.* 8. *Time and method of treatment.* The sooner appropriate care and treatment are adopted, the more likely is a case to be brought to a favorable issue.

Pathology. — Bronchitis is in most cases a catarrhal inflammation of the mucous membrane lining the bronchial tubes, and is often associated with a similar condition in the trachea. The membrane becomes hyperæmic, and, as a result of this, excessive fluid is soon poured out into the tubes, as well as into the substance of the tissues. Nutrition is perverted, and an excessive formation of cells takes place on the surface of the membrane; these are thrown off in a more or less imperfect state, and, mingling with the fluid, give rise to the various corpuscles seen in the expectoration, to which this principally owes its increasing opacity. In many cases it is a purely local complaint, the result of direct irritation; in others it is but a part of some general condition of the system, produced under the influence of "cold" and other agencies, in which the mucous membranes are very liable to suffer more or less extensively. Again, in some instances it appears to be an attempt on the part of the membrane to assist in throwing off some morbid material contained in the blood, which is attended with congestion. With regard to what is termed "Capillary Bronchitis," in many instances undoubtedly this term is properly applied. There is an actual inflammatory state of the smaller tubes, which may either extend from the larger tubes, or originate there in the first instance, or, I believe, may in some cases be caused by the irritation of secretions formed in the larger tubes, running back into the smaller. But in other instances there are no evidences of any inflammation in the capillary tubes, and it seems probable that there is merely a collection of fluid in these tubes, which has flowed down from those of larger calibre, in consequence of a want of power to expectorate. This would be aided by gravitation, as well as by the destruction of the ciliated epithelium, and, after a while, by paralysis of the muscular fibres in the walls of the bronchi. The fluids thus accumulating, added to that normally forming in the tubes, which might be somewhat increased from congestion, would account for the serious symptoms in these cases. The fever which may accompany Bronchitis is not usually due to the inflammation, but is a part of the general state. None is present if the affection is local and limited. The asphyxial symptoms are easily explained by the obstruction in the air-tubes, and consequent interference with the due aëration of the blood. In proportion as the vital powers are below par will be the tendency of the combined fever and imperfect respiratory process to lead to a fatal result.

Morbid Anatomy.—On opening the thorax of a person who has died from extensive Bronchitis, the lungs do not collapse, but remain distended, or may even bulge out: this is caused by the air being unable to escape through the obstructed tubes, and even pressure cannot materially diminish their bulk. The degree of this distension will, of course, vary with the number and size of the tubes affected. The mucous membrane presents various forms of redness, and generally all are seen more or less in the same case. Thus, it may be arborescent, capiliform, mottled, streaked, in points, or diffused, but it is not uniformly spread over a large surface as a rule. In tint it may vary from a bright, vivid pink-red, to a somewhat dark, venous hue, the latter being observed in the later stages. It is sometimes scarlet, and has a velvety appearance. The redness does not ordinarily extend beyond the fourth or fifth divisions, often not beyond the second or third, but it is said that even the finest ramifications may exhibit it. It is generally more marked towards the upper part of the lung. Possibly the action of the elastic and muscular fibres in the walls of the bronchi may diminish it after death.

Where the tubes bifurcate, it is often well marked. Thickening and opacity of the membrane are also observed, to a greater or less degree, from distension of the vessels and infiltration into its substance; from this cause, as well as frequently from the presence of exudation in the submucous tissue, the tubes are reduced in calibre, but unequally, and the surface of the membrane appears uneven. The more minute tubes may be completely closed up; and this is especially apt to happen in young children. The tissue of the membrane is relaxed and softened; often it cannot be stripped off for any length. Patchy abrasions of the epithelium are frequent, giving sometimes an appearance of slight ulceration, but this is never observed in children (Vogel). In the very early stage abnormal dryness is observed, or a very small quantity of transparent tenacious substance covers the surface. Soon excessive secretion is formed, and various materials are found in the tubes, corresponding to the different stages of the expectoration. They may be so abundant as to extend from the finest ramifications up even to the trachea, completely filling all the canals. In appearance the contents of the tubes resemble frothy mucus, or a muco-purulent, or even purulent-looking fluid; the degree of viscidity and adhesion to the surface of the membrane varies, but is usually marked. More or less blood may be present. Sometimes a fibrinous-looking material is seen attached to the surface, lying loose in flakes or masses, or even forming complete casts of the smaller tubes, which may be hollow or solid. The microscope reveals epithelium scales, perfect and ciliated in the early stage, but afterwards imperfect, small and somewhat oval in shape; so-called mucus and exudation corpuscles; large pus-corpuscles, containing numerous granules; sometimes blood disks; granular material in abundance. More cells are observed in proportion to the opacity of the fluid. It is in the lower and more dependent parts of the lungs that the secretions are found in largest quantity. In some cases, especially in children, small yellow spots are visible near the surface, due to accumulation in the air-cells and minute tubes.

Along with the Bronchitis, and as the result of it, lobular collapse is very commonly observed, as was first pointed out by Dr. Gairdner. This condition is particularly frequent in young children. If a large tube is blocked up, more extensive collapse is present. In some cases the bronchial tubes are slightly but uniformly dilated, and acute emphysema is said to occur, but it is a question whether in these cases the air-vesicles are usually actually distended beyond their normal size in deep inspiration, and therefore whether the term emphysema can be properly applied to this condition. Lobular pneumonia is occasionally present, and may be preceded by collapse. Ordinary lobar pneumonia is rare. There may be more or less congestion of the lungs with œdema, or these organs may be natural in hue, or even paler than normal. The bronchial glands are often large, red, and softened.

The blood is dark, and the venous system, with the right side of the heart, overloaded.

Of course the morbid appearances characteristic of Bronchitis will vary according to its extent. Both lungs are usually involved, but seldom to the same degree; nor is one lung affected uniformly throughout. Different conditions are seen in different parts of the same lung. The membrane lining the smaller tubes suffers less than that of the larger, but more secretion is sustained in the former. In cases of death from other causes, more or less Bronchitis is often present.

TREATMENT.—No case of Bronchitis, however slight, should be neglected, because a little care and appropriate treatment at the outset may soon put an end to an attack which otherwise might become very serious or even lead to a fatal result. It must be remembered also that a catarrh, if overlooked at first, may lay the foundation for certain favorable chronic affections. The treatment will necessarily vary much according to a variety of circumstances, and therefore no uniform method can be laid down. I shall first consider the mode of dealing with an ordinary case resulting from cold, and afterwards notice any modifying conditions which may appear to call for remark.

It is always well, if possible, to make the patient keep to the house, or even to one room, maintained at a uniform temperature of 64° to 66° Fahr., if the weather is at all unfavorable; but under any circumstances damp night air must be avoided. Lying up thus for two or three days, will often cure a catarrh. It is customary as well as, I believe, useful in these cases, to endeavor to bring about a free action of the skin. For this purpose a copious warm drink may be given before going to bed, such as hot milk, mulled claret, warm elder wine, or even some strong alcoholic stimulant, such as hot spirit and water. A warm foot-bath should be used, and some mustard and salt may be added to the water. A large quantity of bedclothes should be put on the bed, and the patient should sleep between blankets. Finally a full dose of Dover's powder may be administered, or a diaphoretic and saline draught. Some recommend a hot-air or vapor bath, and a Turkish bath has certainly often the effect of curing a cold.

Wrapping the body in wet sheets is employed by some in order to procure free perspiration, and does not seem to be attended with danger. It is generally advisable to apply a large mustard poultice over the front of the chest, and to allow steam to be inhaled for a few minutes.

If the case is a severe one from the first, and attended with fever, or if it is not checked by the above treatment, more active measures will be required.

Venesection has been practised extensively in this disease. In most cases it certainly is not required, while in those of a more serious type it is extremely rare to meet with the combination of conditions which warrant the taking of blood from the arm. These conditions are said to be where the inflammation is marked and extensive, occurring in a robust and healthy young or middle-aged adult, and accompanied with severe sthenic fever. It can be safely affirmed that venesection is scarcely ever called for, at all events in town districts. *Local bleeding* by leeches or cupping may certainly be employed with advantage in some cases, but great discrimination is necessary even in the use of these modes of removing blood, and in the great majority of instances they can well be dispensed with.

If leeches are used, they should be applied over the front of the chest, or sometimes at the base posteriorly. Their number must vary according to circumstances, but certainly more than from five to ten are seldom advisable. In plethoric children, the blood removed by two or three leeches sometimes relieves great dyspnœa very effectually. Cupping may be performed either in front or behind, if thought necessary, to the extent of from three to six ounces.

It is certainly improper to adopt, as the ordinary practice, any mode of removing blood in cases of Bronchitis; it is far safer to act on another principle. In any doubtful case the patient will stand a better chance of recovery if no blood is taken away. Free dry cupping over the chest, both front and back, is often of much service, relieving the oppression and dyspnœa, and it is quite devoid of danger.

An *Emetic*, in the form of tartar emetic, or ipecacuanha, is made use of by some at the outset, especially in children. Though extremely valuable in certain conditions, it appears to me that it may well be dispensed with at this time. The bowels may be freely opened by some aperient, varying according to the age and condition of the patient, and throughout the case mild purgatives must be used as required.

[There is a strong and reasonable objection in the minds of many practitioners, to the use of tartarized antimony as an emetic with young children. With infants under two years of age, I believe it ought never to be so employed; and rarely, with them, even as a sedative in minute doses. Very small quantities of it will sometimes, in children, produce alarming depression.—H.]

Among medicinal substances, *tartar emetic* ranks as one of the most important during the early stage of Bronchitis. The dose must be regulated by circumstances, but from a third to half a grain every four hours is usually sufficient for an adult. It may be given in a saline draught, containing liquor ammoniæ acetatis, and its effects should be carefully watched.

Tincture of digitalis is also employed by some at this period of the case, and often with marked benefit. *Calomel* with *opium* is recommended if either of the above cannot be taken from any cause, but it seems to me of very doubtful efficacy, and might be often injurious. As the case progresses, and secretions form in the bronchial tubes, the main indications which medicines have to fulfil are the following: 1. To assist expectoration. 2. To alleviate cough, due regard being had to the proper discharge of the secretions. 3. To diminish the quantity of the expectoration. 4. To allay spasm of the tubes, if present. These are carried out by the administration of various expectorants, sedatives, narcotics, and antispasmodics, in different combinations, along with diaphoretics or demulcents. The chief expectorants are ipecacuanha wine, tincture or oxymel of squills, compound tincture of camphor, and, later on, sesquicarbonate of ammonia or chloride of ammonium, senega, serpentary, galbanum, ammoniacum, tincture of benzoin and balsam copaiba. The sedatives and narcotics principally used are hyoscyamus, conium, opium, or morphia, and hydrocyanic acid. The most important antispasmodics include sulphuric ether, ethereal tincture of lobelia, and spirits of chloroform. These various medicines must be employed as they are required, and no rules can be laid down as to their precise use; but it may be stated that the less stimulating expectorants should be given at first, and narcotics, especially opium, must be used with very great caution if expectoration is difficult, and the secretions tend to accumulate.

Local Applications.—Sinapisms are beneficial even from the first, and may be repeated over different parts of the chest. Hot applications are also of much value in the early period, especially hot moist flannels, which may be sprinkled with turpentine, or linseed-meal poultices, which should be large, applied very hot, changed frequently, and continued for some time. The latter are particularly valuable in children. Blisters are called

or after expectoration sets in and the acute symptoms have subsided. One of good size may be placed over the front of the chest, or some recommend the interscapular region behind as the best place for a blister, because more fluid will be drawn there, but the discomfort caused is a great objection. In children the blister may be left on only for two or three hours, and afterwards a linseed-meal poultice applied.

If the affection is tending to become chronic, other forms of counter-irritation may be employed, as the application of croton oil liniment, or acetic acid.

Inhalations.—In the early stage inhalations of simple steam are decidedly useful in many cases, especially if the larynx is in an irritable condition, and giving rise to constant cough. Later on medicated inhalations are of service under certain circumstances, such as those of conium, sulphuric ether, or chloroform, if there is much spasm; those of tar, or creosote, if the expectoration continues very abundant.

Diet and Regimen.—It is quite unnecessary to keep patients on too low a diet, and they may have a fair quantity of beef-tea and milk from the first. With regard to stimulants, it is impossible to lay down any positive rules. Ordinarily they are not required, and might be injurious; but if there are any indications for their administration, such as a tendency to adynamia or asphyxia, they should be given without delay, and their effects watched. Any case that is at all severe should be absolutely confined to the bedroom, kept at a temperature of 66° to 68° Fahr., the air being moistened by steam from a kettle kept constantly boiling; at the same time the room should be occasionally well ventilated, the patient being protected from draughts. Warm clothing must be worn, including flannel, with a sufficient amount of bedclothes. The head should be kept high, and cough should be encouraged, if there appear to be any indication that the secretions are not properly discharged; on the other hand an irritable cough may sometimes be subdued by an effort of the will.

In treating children, emetics are useful at the outset, if the attack is severe. For ordinary cases, ipecacuanha wine constitutes a valuable medicine. They should be encouraged to cough, if old enough, and means should be used to make them breathe freely and expectorate; sleep must not be too prolonged or deep. The throat should be cleared occasionally from mucus by means of the finger; and if the physical signs show that fluids are accumulating in the tubes, an emetic ought to be given, the best being sulphate of zinc. Narcotics must be used very cautiously in children. They should drink freely. Attention must be paid to their constitutional state, such as rickets, tuberculosis; and if either of these exist, all depressing treatment must be carefully avoided. The quality and quantity of the milk should be looked to.

In aged persons and those who are debilitated or are suffering from any acute or chronic disease, depletion in any form is inadmissible. Antimony can only be given very cautiously, and, in most cases, stimulants and stimulant-expectorants are required from the first. Wine or brandy, with plenty of strong beef-tea and other nourishment, must be administered, in quantities according to the requirements of the case. Sesquicarbonate or muriate of ammonia, cinchona, camphor, sulphuric ether, spirits of chloroform, senega, squill, galbanum, ammoniacum, are the medicines called for.

In most cases of Capillary Bronchitis the stimulant treatment is decidedly that which yields most favorable results. If the Bronchitis originates from any constitutional condition, such as gout or tuberculosis, the treatment appropriate for these affections must be employed.

When an acute attack supervenes upon Chronic Bronchitis, free dry cupping, with flying blisters over the chest, are very serviceable. If asphyxial symptoms set in, the stimulant treatment must be persevered in. Chlorate of potash may be given frequently as a drink; artificial respiration and galvanism along the course of the vagus nerve are recommended in extreme cases, and the former may be carried out in children persistently whenever there are signs of danger. A warm bath, with cold affusion while in it, is also advocated by some as an effectual mode of combating asphyxia. If there is a great amount of fever, and there seems to be danger from this cause, quinine is advisable, in doses of from two to three grains every three or four hours.

During convalescence, tonics, such as quinine, mineral acids, iron, may be added to the other medicines. The clothing should be warm, and a pitch-plaster may be worn on the chest. Cold and damp must be avoided for some time, and the patient should not neglect proper precautions until perfectly convalescent.

In those who are subject to Bronchitis, prophylactic measures are called for. All causes likely to bring it on must be avoided; if possible, a change to a warm climate during the cold season should be enjoined. Cold sponging is useful, especially for children, who should be properly clothed, but not immoderately. Constitutional treatment may be called for.

With regard to the treatment of hay-asthma, during the attack small doses of dilute hydrocyanic acid, with tincture of lobelia and other antispasmodics, fre-

quently repeated, seem to act best. Inhalations of creosote are also recommended, or of an atmosphere containing a small proportion of chlorine. In the intervals general tonic measures are useful, such as quinine and iron, with cold bathing. Arsenic and nux-vomica are also employed. Dr. Reynolds has found much benefit from the systematic inhalation of chloroform, from a handkerchief, four or six times a day.[1] In the spring any one subject to this affection should go to the sea-side or take a voyage.

Chronic Bronchitis. Chronic Bronchial Catarrh.

Causes.—Much that has been said on this head with regard to Acute Bronchitis applies to the chronic form also. In fact, it usually is the result of the acute affection, remaining sometimes even after a single attack, especially if the deeper structures of the tubes have become involved, but in the great majority of cases being due to several repeated attacks. The complaint may be chronic from the first, coming on slowly; and this is particularly the case in old people. It is commonly associated with any chronic lung disease which may exist, but especially emphysema or dilated bronchi and the various forms of phthisis. In the latter affection much of the expectoration arises from a chronic catarrh of the mucous membrane. The presence of certain heart diseases, interfering with the circulation, frequently leads to Chronic Bronchitis, especially some forms of it. Various blood affections, notably gout, predispose to it greatly. The inhalation of the different irritating particles, already alluded to, soon causes the affection to become permanent and chronic. Old people suffer in a much larger proportion than those who are younger, but the complaint may be present even in children, particularly after measles and hooping-cough, or where tuberculosis exists. It frequently exists in connection with chronic alcoholism.

Symptomatology.—The local symptoms of Chronic Bronchitis are in kind similar to those attending the acute form, viz. cough, usually accompanied with expectoration, more or less oppression in the chest or dyspnœa, and frequently uneasiness behind the sternum, which, however, is never considerable, and may be absent. These differ materially as to their degree in different cases, and they are variously combined, while certain conditions of the lungs and heart modify them, so that it becomes necessary to classify them into certain groups. The constitution may not be affected in the least, or, on the other hand, may suffer severely.

It will be necessary to describe three chief forms, which are mainly distinguished by the quantity and characters of the expectoration attending each variety, and the qualities of the cough.

1. The first group about to be considered includes the ordinary cases of Chronic Bronchitis, which present innumerable grades of severity, both in the intensity of the symptoms and in their degree of persistence, and the same case will often exhibit these grades during its progress.

At first the patient is only attacked during the winter, having what is termed "winter cough," and being perfectly well during the warmer months. Afterwards the attacks become more frequent, until finally the complaint becomes permanent, but is always aggravated whenever cold weather sets in. The cough may not be severe, only occurring in comparatively slight paroxysms, at considerable intervals; or it is more or less constant, but also increased in paroxysms, which are often very violent and distressing. They are worse at night usually, on first going to bed, but they are especially severe in the mornings, on account of the secretions having accumulated. In bad cases, sleep is much disturbed by fits of coughing during the night.

The expectoration may be brought up without much difficulty in the less advanced cases, but later on it is discharged only with great trouble, as well on account of its own characters as the state of the bronchial tubes, in the smaller of which it is chiefly formed. It varies in quantity greatly, being sometimes inconsiderable, at other times exceedingly abundant. In the slighter cases, it consists of yellowish-white or grayish masses of muco-purulent matter chiefly, containing a number of young, imperfect cells. In others it may be partly in the form of viscid, tenacious, grayish mucus, but the greater portion of it is muco-purulent or purulent in appearance, usually of a yellowish-white color, but often greenish-yellow, and sometimes bright or dark-green. As a rule, it is not much aërated, and often not at all; hence it may sink in water, either partly or entirely, but often floats. The different masses may remain distinct, but usually run more or less together. True "nummulated" masses of large size are occasionally seen. All the forms of expectoration tend towards opacity, and it is not uncommonly thoroughly opaque; an exceedingly disagreeable odor is often present in bad cases, which may amount to extreme fetor. This is supposed to be due to some chem-

[1] See Lancet, "On the Value of Chloroform Inhalation in certain Classes of Spasm."

ical change, and butyric and other acids have been detected, as in a case of Dr. Laycock's. Minute microscopic sloughs of the mucous membrane have also been considered as its cause. Streaks of blood may be observed, especially if the cough is very violent and expectoration difficult, and still more if heart-disease exists. With the aid of the microscope, abundant imperfect epithelium-cells are seen with pus-cells and granular matter. Blood-corpuscles are frequently visible also.

There may be not the least uneasiness or pain behind the sternum, or it may be present at first, and afterwards cease. Generally a certain amount exists, and particularly a sense of soreness after severe cough. In severe cases, breathing is somewhat short on exertion, and during the fits of coughing the respirations are increased in number, but evident dyspnœa does not exist, unless there is emphysema.

The constitution does not suffer in the milder attacks, and the general condition is unimpaired; but in permanent and extensive Chronic Bronchitis the system is gravely affected, on account of the interference with sleep, abundant expectoration, and other circumstances. The flesh wastes, and emaciation may become marked, but it does not go beyond a certain point, and then remains stationary. The strength is reduced in proportion to the wasting. A slight degree of fever often sets in towards the evening, followed by copious sweats at early morning, and there may be marked hectic fever. This increases the loss of flesh materially. The digestive organs usually suffer to a greater or less extent, as evidenced by a furred tongue, deficient appetite, and constipation. If the system is much implicated, the patient is quite unable to follow any employment.

2. A very characteristic class of cases is that which is described under the terms "dry catarrh" ("*catarrh sec*" of Laennec), or "dry bronchial irritation." In this form of Bronchitis very little secretion is produced, and that principally in the smaller tubes. The mucous membrane is in an exceedingly irritable state, and hence violent, prolonged, and very distressing fits of coughing come on, during which the face becomes turgid and red, and the veins swell out, the smaller vessels at last remaining permanently distended. There is, as a rule, no expectoration at the close of the paroxysm, but sometimes a small mass of tough, viscid, semi-transparent grayish mucus is discharged, compared to boiled starch or pearl, or a little thin watery fluid. Much soreness is frequently experienced in the chest after a spell of coughing. There is persistent shortness of breath, which may amount to extreme dyspnœa at times, should a large bronchus be blocked up, or acute catarrh set in. In some instances spasm of the bronchial tubes evidently exists. A feeling of constriction about the chest is always present. Vomiting may occur during a paroxysm of cough. Febrile symptoms are usually entirely absent, but there may be an occasional slight rise of temperature; as a rule the general condition is unaffected. This form of Bronchitis is liable to lead to emphysema, and is commonly associated with this condition in variable degree. It is frequently met with in gouty people, and is said to be prevalent at seaside places, and to come on after the cure of chronic cutaneous diseases, and in those weakened by excesses.

3. The third variety is named "bronchorrhœa," which, as its name indicates, is distinguished chiefly by the abundance of the expectoration, but also partly by its characters. It often occurs in old, feeble persons, after several attacks of acute Bronchitis, particularly when there is some heart affection present obstructing the circulation. The cough comes on in paroxysms, which are often spasmodic and severe, but may be slight compared with the quantity of expectoration. A fit may set in every day, or even several times a day, and it ends with, and is relieved by, profuse expectoration, which is almost clear, transparent, thin, and watery; or thick, ropy, and glutinous, compared to unboiled white of egg mixed with water. It is a little frothy on the surface, but the general mass contains no air. The quantity discharged may be very great, sometimes amounting to four or five pints in the twenty-four hours, and frequently a quarter or half a pint is expelled within an hour; and the amount of fluid poured out into the tubes may be so excessive as to cause fatal exhaustion or asphyxia, especially in aged individuals who are unable to expectorate. During the paroxysms there is considerable dyspnœa, but at other times this is not much observed. The strength fails and the flesh wastes in severe cases, but the constitution may not suffer for years if the expectoration is limited, and it may even relieve the local symptoms produced by certain forms of cardiac disease, which lead to congestion and inflammation of the lungs.

With regard to the form of disease produced by irritant particles, all that it seems necessary to add here is, that after a while the symptoms of bronchitic irritation become chronic, with a constant dryish cough, and subsequently consolidation takes place, which leads to destruction of the pulmonary tissue, and thus cavities are ultimately produced. At first there are no general symptoms, but afterwards emaciation and exhaustion set in.

Usually the course of these cases is very chronic, but it may be tolerably rapid.

It must be borne in mind that very rarely does Chronic Bronchitis exist in an uncomplicated form, and its symptoms will be materially modified by co-existing states of the lungs and heart, and also by the constitutional condition of the patient.

Physical Signs.—It is difficult precisely to define what physical signs are associated with Chronic Bronchitis as its direct results, because there are so many other morbid conditions generally added to it.

1. *Inspection.*—In ordinary cases there is nothing abnormal in the form and size of the chest, but it may be equally and generally enlarged, especially in dry catarrh, in which it is drawn up and made more convex; but here, probably, more or less emphysema exists. The movements are deficient in bad cases, especially that of expansion, and expiration is seen to be prolonged and labored.

2. *Palpation* reveals rhonchal fremitus over various parts of the chest, subject to changes in amount and site. The vocal fremitus is not obviously altered.

3. *Percussion.*—In most instances of confirmed Chronic Bronchitis, it will be found that the resonance is increased in extent and degree on account of co-existing emphysema. Similar conditions to those mentioned under the acute form may cause temporary and localized dulness.

4. *Auscultation.*—The breath-sounds are much weakened usually, but vary in different parts of the chest. After a free expectoration they may be heard extensively. Their quality is always harsh and coarse, and sometimes markedly so. In unaffected parts they are exaggerated. Expiration-sound is much prolonged in long-established cases. Rhonchi of various kinds are heard, but the "dry" are most abundant. The "bubbling" rhonchi are of large, coarse character, and are often temporarily absent. They are altered by cough and deep inspiration, as in the acute affection. It will be readily understood that these rhonchi will vary in the different kinds of Chronic Bronchitis, according to the quantity and consistence of the fluids contained in the tubes. In bronchorrhœa the bubbles give the idea of being produced in a thinner fluid than that of the ordinary form, while in dry catarrh they are necessarily absent. Vocal resonance is very variable. It may be bronchophonic, normal, deficient, or absent.

5. *Displacement of Organs.*—Owing to the emphysema accompanying Bronchitis, there is usually more or less displacement, particularly of the heart.

Diagnosis.—There is scarcely ever any difficulty in diagnosing the presence of Chronic Bronchitis, but this is frequently experienced in determining with what conditions it is associated. Where there is much emaciation, with abundant purulent expectoration, it may simulate certain forms of phthisis, but the comparative degree and rapidity of wasting, absence of or only slight fever as a rule, want of hæmoptysis, and other symptoms present in phthisis, as well as the physical signs, will distinguish them. It is only when there are dilated bronchi, that usually much difficulty is felt in arriving at a correct diagnosis, and these cases are considered elsewhere.

Prognosis.—No case of Chronic Bronchitis ought to be looked upon as unimportant or treated lightly. Though it does not of itself often cause death yet in proportion to its extent does it become dangerous, as an acute attack may set in at any moment; and however slight this may be, the danger in such a case becomes considerable, on account of the difficulty in expelling the secretions from the tubes. At the same time it is a serious affection, because it tends to become more diffused, and also to give rise to certain important and incurable sequelæ. These are chiefly emphysema, dilated bronchi, and collapse. Many pathologists believe, also, that by extending into the air-vesicles it may be the immediate cause of a form of phthisis; in fact, what they consider the ordinary form of pulmonary consumption. Others are of opinion that by causing irritation it leads to a deposit of true tubercle. Much will depend upon the variety of the disease which is present, the amount of expectoration, its effect upon the constitution, the age of the patient, the state of the lungs and heart, and other circumstances. It is said that complete and permanent recovery may take place in the young, if they are taken to a proper climate and treated carefully. In almost all cases it is incurable, when once it is well established.

Pathology and Morbid Anatomy.—Many cases of Chronic Bronchitis are simply due to congestion, usually passive, sometimes more or less active, of the bronchial mucous membrane; others present a so-called inflammatory condition of the membrane, also involving the deeper structures after a while. Hence there is permanent hyperæmia, with perverted nutrition and excessive cell-formation. The morbid appearances met with are usually as follows: The mucous membrane is discolored, being of a more or less dull red, often of a deeply venous hue; a dirty grayish or brownish color may mingle with the redness. It is usually in patches, but may be diffused extensively. The minute vessels are dilated, and frequently varicose. Swelling

and increased consistence of the membrane are usually marked characters; hence reduction in the calibre of the tubes, and an uneven surface. The submucous tissue in time becomes infiltrated, contracted, and indurated, thus in some parts completely closing up the smaller tubes, while the larger tubes tend to dilate diffusely, or even saccularly; a fibroid material is produced, which may increase, and at last fibroid phthisis be established. The elastic and muscular coats of the tubes become hypertrophied, but their elasticity is lost. The cartilages are prone to be the seat of calcareous deposit. Owing to the thickening and induration of their walls, the tubes gape when cut across, and many appear enlarged. Epithelial abrasions are common and diffuse, or follicular ulceration is said to be observed occasionally, especially in tubercular phthisis. The contents of the tubes correspond to the matters expectorated. There is often a large quantity of yellowish purulent-looking fluid, which may completely fill the smaller bronchi. Usually frothy mucus exists in the larger ones. In dry catarrh the membrane is much swollen, and has upon it a small amount of tenacious, glairy, semi-transparent mucus. Emphysema is constantly present to a greater or less extent.

Treatment.—Much harm unquestionably results from the indiscriminate treatment of cases of Chronic Bronchitis by expectorants and narcotics, which is often practised. There is no disease in which a careful study of each individual case is more required than this, in order to take proper measures for its relief. Of course the primary object that should be kept in view is the cure of the complaint; but, failing this, it is very important to prevent it from extending, and hence early and persistent treatment is called for, not merely with the aid of medicinal agents, but also with regard to general management and hygienic measures. In advanced cases, all that can be done is to relieve certain symptoms, and to ward off various dangerous complications.

In dealing with any particular instance of the disease, the following points should be taken into consideration: 1. Whether there is any obvious cause, either external to the individual or depending upon some internal condition, which keeps up a state of congestion of the bronchial mucous membrane, and consequent catarrh. 2. The constitutional state of the patient, and the degree to which the system has become affected. 3. As regards the immediate symptoms, the treatment must depend upon (*a*) the quantity of secretion formed, and the degree of difficulty which is experienced in its discharge; (*b*) the condition of the mucous membrane; and (*c*) whether there is any spasmodic element present in connection with the muscular fibres of the bronchial tubes.

With regard to the immediate cause of the affection, if it is known to result from any irritant inhalation, removal from exposure to this is the first thing called for. The same applies to the atmosphere of any particular district which may appear to disagree; a change to some other atmosphere is necessary, as will be pointed out when the subject of climate is considered. Certain cardiac affections seem to keep up bronchitic symptoms, by inducing congestion of the mucous membrane; when any such is present, treatment directed against it may afford much relief, especially the administration of tincture of digitalis in moderate doses, which may be combined with such other remedies as the case requires. This drug is especially recommended in the variety named "bronchorrhœa."

Various constitutional conditions are associated with Chronic Bronchitis, and these demand careful attention. If plethora exists, this must be reduced by appropriate diet and general management, and the use of watery purgatives. On the other hand, an anæmic state of the blood must be rectified by the different preparations of iron, which are frequently of much value. In many instances a gouty diathesis is present, especially when "dry catarrh" is the form of the affection assumed; if such be the case, colchicum with alkalies often proves of much service. Alkalies are also useful if there is a rheumatic tendency, as well as sulphur, certain mineral waters, and other remedies employed in rheumatism. Iodide of potassium is said to afford much relief in certain cases, and probably those accompanied with rheumatism would be most benefited. When the complaint occurs in children, in connection with rickets or tuberculosis, the treatment requisite for these diatheses must be thoroughly and perseveringly carried out. In the great majority of cases of Chronic Bronchitis, it will be found that a general tonic plan of treatment is attended with most success. A course of quinine, or mineral acids with decoction of cinchona or some bitter infusion, often proves of great service. The quinine may be combined with sulphate of iron, or some other chalybeate preparation. Mineral nervine tonics, such as sulphate or oxide of zinc, are also of use in some cases. It is especially in those instances where there is excessive expectoration, and consequent loss of flesh and strength, that tonics are valuable; and here also cod-liver oil is of essential service, given in small doses at first, which may be gradually increased. Bronchorrhœa is also much benefited by tonics, especially the different preparations of

iron. A course of mercury is said to have a very favorable influence over some cases of Chronic Bronchitis.

The *Symptomatic treatment* is often attended with much difficulty, and remedies have to be variously combined, and frequently changed, in order to afford relief. It will be necessary to consider briefly the main indications. The secretions may be formed in excessive quantity, and then the indication is to limit their formation. For this purpose various inhalations are of much importance. Among these, tar and creosote, or naphtha with steam, rank high. The vapor of iodine, chlorine gas, muriate of ammonia, the different balsams, and resins, are also used with success as dry inhalations. They should be employed freely diluted, and their effects carefully watched; but when properly administered they certainly often prove efficacious. [In some cases, presenting great irritability of the bronchial tubes, with frequent, worrying cough, I have known inhalation of the fumes of tobacco (from a cigar or pipe) to have a very soothing and relieving effect. A patient unaccustomed to smoking, for example, may puff away to the extent of a quarter or half a cigar at a time, leaving it off as soon as the slightest nausea is produced.—H.]

General tonic measures are called for here, and iron, especially its astringent preparations, as the tincture of the sesquichloride, is of much value. Other astringents must be given also, such as tannic or gallic acid, acetate of lead, and the mineral acids; also the various resins and balsams, especially galbanum, myrrh, ammoniacum, and balsam copaibæ; the last-mentioned is often very useful. Muriate of ammonia has been recommended. This treatment applies generally to cases of bronchorrhœa.

The fluids may not only be produced in excess, but there is also a deficient power of expectoration, owing to the state of the tubes, the adhesive character of the secretion, or other causes. Under these circumstances stimulant expectorants are required, and may be combined with the former class of remedies. The chief of these are sesquicarbonate of ammonia, muriate of ammonia, squill, senega, serpentary, camphor, and tincture of benzoin, in addition to the resins. Alkalies, such as the carbonate of potash or soda, or liquor potassæ with balsam copaibæ, may be tried along with ammonia, if the expectoration is very adhesive and viscid. If there is any tendency to great accumulation, an emetic of sulphate of zinc occasionally will do no harm and may give much relief. Narcotics and sedatives, but particularly opium, must be either avoided or used only with great caution in these cases, particularly in old persons; and the patient should be encouraged to cough frequently, in order to prevent accumulation.

In other instances, the mucous membrane is in an extremely irritable state, but scarcely any secretion is produced; hence there is constant cough, with scanty or no expectoration. Should there be any sign of irritative fever under these circumstances, small doses of tartar emetic or ipecacuanha wine may be given. The most important drugs in these cases, however, are the narcotics and sedatives, which should be administered in full doses. Opium is of essential value here, and may be combined with ipecacuanha, in the form of Dover's powder, or it may be given as the tincture, Battley's solution, or compound tincture of camphor. Solution of morphia is also extremely useful. Hydrocyanic acid, tincture of lobelia, hyoscyamus, conium, stramonium, belladonna, are other beneficial agents, and may be variously combined with other medicines. Gout is frequently present, and hence alkalies and colchicum may prove efficacious. Inhalations are to be recommended here also, but of the sedative class, viz., conium, hyoscyamus, stramonium or ether, with steam.

When there is evidently much spasm, as shown by the breathing and cough, the narcotics and sedatives are likewise employed with advantage, associated with different ethers, especially sulphuric ethers. Tincture of cannabis Indica appears to act well in some cases. Ipecacuanha and tartar emetic, in doses sufficient to nauseate, but not to cause vomiting, are also recommended. A few drops of chloroform may be inhaled occasionally if the tendency to spasm is great, and the sedative inhalations previously mentioned may be employed. In these cases there is always more or less emphysema. The symptomatic indications just considered are generally associated to a greater or less degree in practice, and hence the remedies have to be given in various combinations.

Local Treatment. — Free dry-cupping over the chest is often very serviceable, especially in case of irritable mucous membrane. Different forms of counter-irritation should be employed according to circumstances, viz., sinapisms, blisters over different parts, croton oil liniment, turpentine, acetic acid, or tartar emetic ointment. The croton oil liniment is certainly very often beneficial. Some recommend an issue or seton. When these are not being used, a large warm plaster, such as a pitch-plaster covered by a thick layer of cotton-wool, should be worn over the chest in front.

Under no circumstances does it appear necessary or desirable to remove blood, either generally or locally, in cases of

Chronic Bronchitis; and if an acute attack supervened, the less this mode of treatment were followed, the better would be the patient's chance of recovery. Stimulants, such as sesquicarbonate of ammonia, with chloric ether and squills, as well as wine or brandy, should decidedly be employed in preference when this happens.

General Management.—This requires careful attention. It is necessary that the patient should breathe an atmosphere of good, uniform temperature, without excessive moisture, and should avoid sudden changes. Most patients cannot leave this country during the winter season, and then they should remain indoors when the weather is at all severe, their room being maintained night and day at a regular temperature of 62° to 65° Fahr., and should always wear a respirator when out. Especially must night air and cold winds be avoided. If possible, they should reside in a part of the country possessing suitable atmospheric conditions, which vary in different cases. An entire change of climate to some more temperate region is of the greatest importance, if it can be obtained, or a long voyage may be taken. Different forms of Bronchitis require different climates; but they all require tolerably warm temperature, without sudden changes, a moderately high altitude, and protection from cold winds. For "dry catarrh" a soft and relaxing atmosphere with moderately high temperature, is recommended. One more or less stimulating, dry and hot, is advised where there is much expectoration. In this country the principal places which receive this class of invalids are, Torquay, Penzance, Bournemouth, Grange, Clifton, and Tunbridge Wells. Among foreign parts the chief are Mentone, San Remo, Pisa, Rome, Cannes, Algiers, and Corfu. [To these, for the winter season at least, upper Egypt may be added; and, in America, Florida, and Southern California.—H.] Some go to Harrogate and other places, on account of the mineral waters, which are useful in certain cases.

Sufficient warm clothing should always be worn, with flannel next the skin. The functions of the skin must be maintained in an active state, and a warm or hot-air bath, or even a Turkish bath, may be employed from time to time. When the weather permits, moderate exercise is advisable. The diet should be at all times nutritious, and especially if there is much emaciation. As to stimulants, no definite statement can be made; but in most cases a moderate amount of some alcoholic stimulant will be of service. The digestive organs must be attended to, and aperients administered if required.

When a severe attack of bronchorrhœa comes on, stimulants and sedatives are called for, with a hot-air or vapor bath, and sinapisms over the chest and to the extremities, or free dry-cupping. Emetics may be also employed if the fluids appear to accumulate, and cannot be expelled.

Plastic or Croupous Bronchitis.

This is a very rare form of disease, and will only require a brief notice. Pathologically it differs from ordinary Bronchitis, in that a plastic material is thrown out into the tubes of which it forms casts. These are either solid or hollow, this depending much upon the size of the tubes in which they are formed, and they usually present a series of concentric layers; but this appearance is sometimes wanting. Their size necessarily varies according to the size of the containing tube. Usually they are confined to a limited number of the bronchial divisions, but may extend from the smallest even to the largest, though they never pass into the trachea; whereas the exudation of croup or diphtheria may even reach to the most minute bronchi. Their color is whitish, like decolorized fibrine, but spots of blood may be attached to them. Some have regarded them as the remnants of blood poured out into the tubes, which has coagulated and lost its color. Possibly such casts may be met with occasionally, but those now under consideration are usually, and with greater reason, regarded as the result of a true exudation on the surface of the membrane. Microscopically they consist of an amorphous or fibrillar material, in which there are exudation-corpuscles and fusiform or ovoid cells, most of which are non-nucleated, but some contain nuclei, abundant granular matter, and some oil-globules are also present.

The causes of this affection are very obscure. It is supposed to be due to some diathetic state, and is said to be sometimes associated with tuberculosis. It is most frequent in young adults, but may be met with at any age. Females suffer rather more frequently than males, and those of feeble and delicate constitution are more subject to this form of Bronchitis than those who are strong and healthy.

Symptoms.—In the great majority of instances Plastic Bronchitis is a markedly chronic affection, but it has been known to occur in an acute form, particularly in infants. Though chronic in its general course, there are, however, acute exacerbations on the occasion of the discharge of the casts. The severity of the symptoms will depend upon the size of these and the degree of facility with which they are expectorated. In most cases an irrepressible hacking cough sets in, painful and spasmodic, either dry or attended with slight

expectoration of ordinary characters. This is followed by dyspnœa, which may gradually increase, or come on rather suddenly. It often becomes very intense, with a sense of great tightness and oppression across the chest; and there may be an appearance of threatened asphyxia if some of the larger tubes are obstructed. Walshe has found the pulse-respiration ratio to vary from 2·2 : 1 to 3·5 : 1. The cough becomes more and more severe, and the distress greater, until particles of fibrinous material are expectorated, mixed more or less with ordinary mucus; and finally one or more masses varying in size will be expelled, which, on being disentangled under water, will be found to present complete casts of the tubes, in the form of tree-like branchings, and having the characters already described. The cough and dyspnœa are then either entirely or partially relieved. Streaks or spots of blood are frequently seen on the outside of the casts, and occasionally on their inner surface; and there may be streaks or drops of bright blood in the mucus which is expectorated for a short time after the casts have been discharged. Copious hæmoptysis may occur before the attack comes on, but Walshe believes that the concretions are then merely altered coagula. The length of a paroxysm varies within wide limits, and it may be followed by complete or temporary recovery, or the attacks may recur at longer or shorter intervals for weeks or months. There may be an entire absence of fever, but in many cases febrile reaction sets in, preceded or not by a rigor, and it may be considerable in degree. Frequently abundant mucopurulent expectoration takes place, and extensive Acute Bronchitis or pneumonia is sometimes set up, giving rise to the usual symptoms of each affection. The general health does not suffer much as a rule between the acute paroxysms, and there may be no chest symptoms. Often, however, there is a certain amount of habitual dyspnœa, and signs of imperfect respiration.

Physical Signs.—Sometimes pulmonary resonance is in excess over a part of the chest, owing to partial closure of a tube, and the portion of lung to which it leads being over-distended with air. More commonly localized dulness is met with, owing to complete obstruction and consequent collapse. The respiratory sounds are either weak or totally absent, according to the amount of obstruction. When this is removed, the above signs disappear. Dry rhonchi, especially sibilant and a few of the moist kind, are heard in different parts. Should pneumonia or Acute Bronchitis be produced, the physical signs characteristic of either complication will be present.

Diagnosis.—This form of disease may be mistaken for ordinary Acute Bronchitis, pneumonia, or pleurisy. The history of the case, the characters of the paroxysms, expectoration of membranous fragments or casts, and physical signs serve to distinguish them. The degree of fever will also be important, and the absence of the symptoms usually met with in the above diseases.

Prognosis.—It is not attended with much danger in itself as regards life, but it may lead to pneumonia, phthisis, &c., and thus cause death. Complete recovery sometimes occurs, but usually this is only temporary, the disease being one which has a great tendency to recur.

Treatment.—Various remedies have been recommended, but apparently their use has not been attended with much success. During the paroxysms venesection has been practised, sinapisms and blisters applied to the chest, and various drugs administered, viz., the different sedatives, tartar emetic, ipecacuanha, calomel and opium, alkalies, and salines. Inhalations might be of use, and the patient should be kept in a warm room, having the air well saturated with moisture. In the intervals, Fuller has occasionally seen benefit result from the use of tartar emetic, in moderate doses, for several weeks. Iodide of potassium and inhalations of iodine have been employed with success. The alkalies and their carbonates have also been recommended. The health must be maintained, and tonics given if necessary, more especially if there is any sign of tuberculosis. Quinine, iron, and cod-liver oil are often called for. A change to a warm climate, or a long sea-voyage, might be tried; while every precaution should be taken against cold and wet. If an attack threatens, inhalation of steam should at once be had recourse to, and persevered in.

PLEURODYNIA.

By Francis E. Anstie, M.D., F.R.C.P.

Definition.—Sharp unilateral pain, greatly aggravated by respiration and other movements, in the extra-thoracic or in the intercostal muscles; unattended, except accidentally, with fever.

History.—The attack is sudden, and is usually brought on by either exposure to wet and cold, or by some rather energetic movement of the trunk or of the arm; often it is the sequel of a prolonged effort involving continuous contraction of the muscles of one side of the chest. Very commonly the patient will remember that for some days past movement of the affected part has always been irksome, and followed by aching pain. The affection tends to subside spontaneously under the influence of rest, in a few days. Often the patient has been previously subject to muscular pains in other parts.

Symptoms.—The patient, after experiencing more or less preliminary soreness or aching of the part, is suddenly attacked with stitch-like pains, most commonly in the infra-axillary or infra-mammary region, and more usually in the left side than in the right. The natural play of respiration is interfered with by the severe pain which the movement causes, the expansion is therefore jerking and irregular, and the respiratory sound corresponds with this in character. No percussion-dulness, friction-sound, or other of the physical signs of pleurisy, can be detected; there is no fever, unless by accident the patient is suffering from some coincident febrile affection. *Superficial* tenderness is *not* a characteristic of Pleurodynia, but there may be dysphagia, and pain on movement of the arms.

Etiology and Pathology.—It has been customary to class Pleurodynia as a variety of rheumatism, affecting the thoracic muscles and their tendinous insertions; but I can discover no satisfactory grounds for this proceeding. It appears to me that Pleurodynia is merely an intense variety of the myalgia which, in less striking forms, is very much too common, and besets far too large and miscellaneous a class of patients, to allow us for a moment to assume that the rheumatic taint is a necessary factor, or indeed a factor at all. I have several times seen very severe Pleurodynia in patients whose history showed no trace of rheumatic tendencies; and on the whole there seems to be far better evidence for the connection of this malady with the neurotic than with the rheumatic constitution. In the absence of any sufficiently accurate and extensive statistics, I must provisionally believe that the exciting cause of Pleurodynia, like that of myalgia generally, is over-long or over-severe exertion of a muscle in proportion to the state of its nutrition, and that the predisposing cause is the neurotic constitution.

As regards the intimate pathology of Pleurodynia we know little. There is nothing to point out any special anatomical condition of the affected muscles as a constant attendant of the malady except this, that Pleurodynia occurs, for the most part if not always, in persons with slight and thin muscles, suggesting under-nutrition of those structures. I can see no shadow of reason to suppose that a local *inflammation* has anything to do with Pleurodynia; and the results of treatment are directly opposed to such an idea.

Diagnosis.—This is the really important aspect of Pleurodynia. It is extremely likely to be mistaken for pleurisy, thus causing alarm, and, in the hands of some practitioners, a disastrously heroic treatment. The total absence of alteration in the pulse-respiration ratio, and of the physical signs of pleurisy, must soon undeceive any one who is even moderately careful; but during the first few hours the ablest practitioner may be at fault. This is especially the case in two situations: first, when the malady accidentally coincides with a catarrh, or some other affection causing feverishness; secondly, where the patient is a highly nervous person, whose circulation is habitually much accelerated by pain or any other cause of distress. Such being the fact, it is the more fortunate that the modern treatment of pleurisy no longer includes those heroic measures by which it was once the fashion to attempt to cut short the disease at the outset.

Prognosis is scarcely worth mentioning. The affection is trivial, and certain to yield in a few days at most.

Treatment.—Two remedies only are necessary. The side should be covered with a sheet of spongio-piline or with flannel and oilskin; or a simpler and readier method is to surround the side with a piece of thin mackintosh, which may be put on over the flannel shirt, jersey, or spun-silk vest. One quarter of a grain of morphia should be subcutaneously injected, and repeated if necessary, in two hours' time. This plan never fails to give complete relief, but the patient should be sedulously warned against all movements not absolutely necessary, for a few days after the pain has ceased.

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PLEURISY.

By Francis E. Anstie, M.D., F.R.C.P.

Definition.—Inflammation, partial or general, of one or both pleuræ, attended with the effusion of lymph, lymph and serum, or pus.

History.—The circumstances under which Pleurisy may arise are very various; but a practical line of distinction separates two main varieties of the disease. Pleurisies may be divided into *Primary* and *Secondary*.

By Primary Pleurisies we mean those in which the cause of the affection operates directly or mainly upon the pleura itself; and the inflammatory affection of that serous membrane arises, so to speak, in a time of health, and only secondarily implicates the rest of the body, by means of the constitutional fever which it excites, or by some other results, mechanical or physiological, of the local disease.

By Secondary Pleurisies we mean those cases in which the pleural inflammation is a complication, or a secondary production, of some other visceral disease, or of some constitutional malady which has gained a hold upon the organism. Even this classification may require to be remodelled at a future day; we may possibly find it to be too absolute: but it appears to correspond well with the facts as we know them, and it marks out in a convenient manner some broad features by which two kinds of Pleurisy are distinguished in the important matters of vital significance and appropriate treatment.

Far less practical is the attempt to divide pleurisies into *acute* and *chronic:* at least it is only in discussing the strictly clinical aspect of the disease that we can say anything useful under this heading. Two facts which are the eminently characteristic results of modern investigation have mainly tended to supersede the division into acute and chronic; first, the increasing certainty that primary acute pleurisy is but rarely fatal; and secondly, the discovery that those chronic cases which are merely the prolongation of the acute primary variety, both may and ought to be treated with a boldness and energy which tend greatly to abridge the course they would formerly have been allowed to run. With modern means and maxims of treatment, it is not too much to say that primary chronic pleurisy has lost its most important features and its peculiar terrors: and the only reason for regarding Pleurisy of chronic type in any special way is the fear that underneath the apparently merely local affection there may lurk the taint of, or the tendency to, a constitutional disease like tuberculosis.

Of the acute primary disease, in robust subjects, the history is essentially this. It attacks suddenly, lasts from ten days to three weeks, and then, in the majority of cases, departs, leaving behind it no other than trifling local changes, which are of no injury to the patient's subsequent health and activity. In a smaller number of cases, however, it produces an amount of effusion which is with difficulty got rid of, and unless evacuated by surgical means may remain, and protract the state of ill health, for many weeks longer. In such cases, also, the amount of permanent mechanical danger to the organs may entail disastrous after-consequences; or it may even happen that constitutional disease of a fatal kind (especially tuberculosis) may be secondarily set up.

Of Pleurisies that are "chronic" throughout—*i. e.*, that commence in an insidious manner,—by far the greater number are not primary, but secondary to some constitutional malady or some disease of another viscus. Nevertheless, it unquestionably happens, in a few cases, that apparently healthy persons are attacked with pleuritic mischief of so insidi-

ous a kind, that almost before the patient knows that he is ill (although he may have been slightly ailing for some days or weeks), it is discovered that one pleura is half or three-quarters full of fluid. Such cases are commonly very tedious in their course, and, if they do not compel the performance of paracentesis at first, take the form of an empyema or collection of pus.

When we turn to the consideration of secondary pleurisies, we find a far greater variety of type, and a much more serious prognosis, attaching to these maladies. To speak first of the Pleurisies which come in as a secondary complication of acute fevers. The whole type, and the vital significance of this class, depends on two factors—the virulence of the original disease, and the power of resistance which the organism has so far presented to it. That a patient with typhoid fever, or acute rheumatism, for instance, is suddenly attacked with Pleurisy, may be of the greater or less consequence, according to the amount of vital power of resistance which the tissues generally, and the organs of vegetative life (especially the heart and kidneys) retain. The main points, however, which the history of the pleurisies secondary to acute fever presents, are the protracted course, the tendency to purulent character of the effusion, and the frequent termination, either in death or in disastrous results in the way of constitutional disease, especially tuberculosis.

The pleurisies produced by *pyæmic infection* are mere incidents of an almost necessarily fatal blood-poisoning.

Far different is the history of the pleurisies which are secondary to the common form of *pulmonary phthisis*. The great majority of these take the shape of acute and strictly limited fibrinogenic inflammation, and, unless very injudiciously treated, tend to rapid termination with no worse result than a local adhesion of the pleural surfaces. More must be said on this point hereafter; at present it will be enough to state that until the later stages of pulmonary phthisis, it is decidedly uncommon, in my experience, to see pleuritic attacks causing considerable serous or sero-purulent effusions, unless they are "actively" treated, in the sense of a free use of depressing remedies. But when once a patient, with an already considerable development of chronic destructive lung disease, has acquired, in addition, a large serous or sero-purulent effusion in his pleura, the chances are heavily in favor of a disastrous termination of the disease, and it may even happen that a swift development of true tuberculosis may carry the patient off in a very few weeks, though the pleuritic effusion were, in itself, quite incompetent to endanger life.

The pleurisies which are consequent on acute or subacute diseases of other viscera are of various types. Pneumonia, for instance, is in numberless, perhaps almost in all cases, attended with a certain amount of fibrinogenic pleurisy; but fortunately this is, in the majority of cases, limited to the production of a circumscribed effusion of lymph, which leads to no serious results. It is far otherwise with the more infrequent cases of pneumonia, which become complicated with effusion of a considerable quantity of pleuritic fluid: this form of secondary pleurisy usually presents acute and highly dangerous symptoms at first, and if not rapidly fatal (as it often is) is usually intractable in its after-course.

The form of Pleurisy which is secondary to Bright's disease, is always a grave and intractable affection; but its history differs greatly according to circumstances. Where it is the immediate consequence of the acute albuminuria of scarlet fever, the tendency is towards a rapid change of the effused fluid into pus; and the mildest result probable is a chronic empyema, with too often fatal secondary results. A different type of Pleurisy may be seen occurring as a complication, often a late one, of the cirrhotic or contracting form, or (much more rarely) of the amyloid form, of renal disease. In neither of the two latter forms is there the same tendency towards the rapid production of pus, but rather a tendency towards the effusion of a large quantity of fibro-serous (chiefly serous) effusion.

As for the pleurisies said to be secondary to acute cardiac disorders, it may well be doubted whether these are not always to be considered as results of some blood-poisoning, or constitutional vice, to which the heart affection is also due. Their course depends upon the degree of vital power which the organism has retained in its struggle with the constitutional malady. They can only be considered as incidents in a more formidable disease. It is, however, an open question whether pericarditis may not excite Pleurisy by direct extension of the inflammatory process.

ETIOLOGY.—Upon the etiology of Primary Pleurisy we possess no sure information at all. There is, indeed, a limited class of cases in which the inflammation is the direct result of a blow or some other injury; but we know of no other causes, properly so called. Among exciting causes, *cold* has often been confidently stated to be a frequent one; but some of the best authorities of late years entirely deny this; and Ziemssen, out of 54 cases of Pleurisy of which he minutely examined the history, could not trace the disease to exposure to cold even in a sin-

gle instance. I have myself had some reason to think that extreme muscular over-exertion and exertion in continuous public speaking produces Pleurisy, sometimes, in otherwise healthy persons.

Of predisposing causes, age has been reckoned an important one. It was supposed by some that it never occurred in very young children; *e. g.*, Barrier formally denied its occurrence at all in children under six years of age. It is difficult to ascertain the exact degree of frequency of Pleurisy in young children, because in them the disease is particularly likely to occur without being detected. But all the best authorities now agree that Pleurisy is quite common among children—at any rate, after the first year of life; and Guinier, of Montpellier, actually tapped an empyema in a child twelve months old. Steiner and Neuretuer,[1] in a noteworthy series of papers, express the opinion that in young children Pleurisy with liquid effusion is the rarer, Pleurisy with proliferation of connective tissue the more common. Ziemssen[2] tabulates the ages of 54 children whom he treated for Primary Pleurisy: First year of life, 3; second, 1; third, 7; fourth, 4; fifth, 2; sixth, 4; seventh, 4; eighth, 5; ninth, 9; tenth, 7; eleventh, 2; twelfth, 1, thirteenth, 1; fourteenth, 2; fifteenth, 1; sixteenth, 1. This very interesting record sufficiently disposes of the idea that there is any immunity in infancy.

A similar investigation of Ziemssen seems to show that there is no well-marked influence of *seasons of the year* as a predisposing cause.

Of Secondary Pleurisies, the exciting causes are numerous. Among the fevers, scarlatina and typhoid are especially notable in respect of frequency of occurrence, variola in regard of danger; acute rheumatism is a frequent cause; alcoholism and pyæmic poisoning often produce Pleurisy, *inter alia*. Of tuberculosis proper and catarrhal pneumonic phthisis, it may be said that they frequently act as predisposing, and frequently as exciting causes of Pleurisy. Of diseases of other viscera pneumonia is the most common cause of Pleurisy; after this comes kidney disease, which is, at any rate, a very powerfully predisposing cause; finally, any organic disease which necessitates mechanical pressure on, or irritation of the pleura: and it is possible that inflammation now and then passes over to the pleura, by mere *contiguity*, from neighboring parts, *e. g.* the pericardium.

[1] Prag. Vierteljahresch. 1864–65; Schmidt's Jahrbuch, 129, p. 189. The papers are part of a series of "Clinical Records of Children's Diseases."

[2] Ziemssen, Pneumonie und Pleuritis im Kindesalter.

Clinical History.—The symptoms of Primary Pleurisy of acute type are as follows: The patient, after suffering for a variable number of hours, or hardly suffering at all, from general malaise and loss of appetite, is attacked almost simultaneously with sharp stitch-like pain in some portion of the thoracic wall (by far the most frequently in the anterior or the lateral portion, a little below the level of the nipple), and with more or less shivering. The face is generally pale and contracted with the lines of pain; the patient bends over towards the affected side, and draws his breath with visible difficulty, in a hurried, uneven, and shallow manner (*respiration entre-coupée*). After this has lasted a short time flushing of the face appears,[1] the pulse rises in frequency, and the general phenomena of pyrexia are evident; in some cases the pain now greatly diminishes, in others it maintains its intensity.

The frequency of the pulse in the early stages of Pleurisy varies considerably. There are plenty of slight cases of localized fibrinous inflammation in which hardly anything like pyrexial rapidity of pulse is present; the frequency may not be more than 86 or 90, and I have even seen a case in which it never rose above 80. In cases of primary fibrino-serous Pleurisy the pulse-frequency may be said to vary between 90 and 120 in the stage of febrile reaction after the initial rigor; on examining the notes of twelve such cases, I find the average rate at this period was 99. It must be said that all these patients were adults, and that a considerably higher pulse-rate may be found in young children, though this is by no means always the case.

The *quality* of the pulse is a point which I have particularly investigated in a considerable number of cases, and it seems to me quite certain that this follows a uniform course on the whole, regard being had to the general vital status of the patient. In the first stage of acute pain, with more or less tendency to shivering, the pulse, as tested with the sphygmograph, presents the "algide" form, *i. e.* the pulse-waves are very small and nearly devoid of secondary markings. As soon, however, as flushing of the face occurs, and a general sense of burning heat of the skin, the pulse passes to the true pyrexial type; the waves become large and dicrotic. One reads constantly, in standard works, of pleuritic patients with (sensibly) hot skin, flushed face, and a *hard* bounding pulse: but the sphygmograph, in my belief, destroys this clinical picture, for it uniformly shows that the large and some-

[1] The flushing is never so fixed and deep a color, and especially never so markedly one-sided, in pure Pleurisy as in pneumonia.

what bounding pulse is always decidedly *less resistant* than that of health.

The *temperature* follows no regular course in Pleurisy; in the primary disease we rarely derive any useful indications from it. On this point I agree, in the main, with the conclusions of Wunderlich,[1] and I shall say more about it when treating of Prognosis. It is enough here to say that temperature-changes keep no sort of parallel with the pulse or the respirations.

The respirations in acute Pleurisy are both absolutely rapid, and especially so in comparison with the pulse; the rapidity being mainly due to the impossibility (from pain and soreness) of taking deep breath.

Cough is a very usual, though not universal, accompaniment of the acute stages of Pleurisy. It is short and hacking; and is either perfectly dry or attended with only a moderate amount of thin mucous expectoration; except, indeed, when the Pleurisy is complicated with pneumonia.

The *decubitus* has been made a strong clinical feature by some writers on Pleurisy; but there are contradictory statements, and from my own observation I should say that there is no attitude characteristic of the disease except that which very generally prevails in the first acute agony, viz., half lying, half crouching, *on the affected side.* The decubitus is frequently changed two or three times in the course of the illness, and, except as attracting our attention to the physical examination of the chest, is seldom of any moment.

Along with these phenomena there is a variable amount of nausea, white-coating of the tongue, thirst, and anorexia; the last usually complete.

It must be observed, however, that the above is only the picture of the early stages of a *typical* acute case in an adult. Even in adults there may be, in cases that run a pretty severe course, scarcely one noticeable symptom to arrest attention in the early days of the malady.[2] And in children the febrile symptoms, particularly the initial rigor, are often inconsiderable, and the cough scarcely attracts notice, especially in slight cases.[3]

Physical Signs.—It is to these that we specially direct attention when suspicion of Pleurisy exists, and the information they afford is more valuable than any other.

Inspection.—When the chest is laid bare, it will be seen, in the very early stages, that the pleuritic side of the chest is somewhat retracted, and its intercostal muscles nearly or quite motionless, while increased play and movement of the sound side is observed. At a later period of effusion a positive dilatation of the pleuritic side and a bulging of the intercostal spaces may often be noted, but it is possible, even in cases of very extensive effusion, for the chest wall to remain apparently unaffected, the force of displacement being spent mainly on the neighboring organs. But in all cases where there is the least suspicion of Pleurisy, accurate *repeated* measurements of both sides must be adopted:[1] it will not do to trust the eye, for enlargement of the side may be obscured by the general configuration of the thorax, or it may happen that the expansion of the *sound* side (in its compensatory efforts of breathing) may assist in concealing the fact. One of the most striking pieces of evidence offered to the eye is the visible displacement of the heart which usually presents itself when the effusion is large: in the case of left pleurisy, the apex will be seen beating under or to the right of the sternum, or in the epigastrium; in right pleurisy it may be found beating to the left of the left mammary line, and, in extreme instances, even in the left axilla.

Mensuration.—In the early stages there is commonly no enlargement of the affected side; the sound side, indeed, appears, and is, the most expanded. But as effusion comes on the balance is restored, and when the fluid becomes copious the intercostal spaces yield, the ribs become more separated, and in proportion to the yieldingness of the thoracic wall a real increase in the size of the affected side is observed. It is only in the slight-made chests of young children that this is early perceptible; in adults, the displacement is rather on the side of the viscera until the effusion becomes very large. Strict daily comparative mensuration of the two sides ought nevertheless to be practised from an early stage.

Percussion yields no information in the first stage, nor can it be well tolerated. Supposing the case to be one of merely fibrinous exudation, we may get, from first to last, scarcely any abnormality in the sound elicited; but the chest wall gives a strange sense of deadness and inelasticity to the percussing finger. Very solid and thick fibrinous deposits may cause a really dull percussion sound. When serum is poured out in any quantity, however, the evidence from percussion becomes striking: over the whole

[1] Das Verhalten der Eigenwärme in Krankheiten. 2te Auflage. Leipzig, 1870, pp. 374–6.

[2] See, among other authors, Trousseau (Clin. Médicale, tome i.) for some striking examples.

[3] Ziemssen, Pleuritis u. Pneumonie im Kind alter.

[1] Cf. Verliac (Épanchements pleuritiques, Paris, 1865), particularly case at pp. 19, 20.

space occupied by the fluid there is found a dulness, more pronounced in some cases than in others, but always with a character of its own, which must be heard to be recognized, but is much more marked than that produced by lung-consolidation. In ordinary cases, where the fluid is not bound and localized by adhesion, the dulness reaches upwards from the base of the chest to a variable height, according to the amount of the effusion: its character is very perceptible in comparison with the sound side, but the line of its termination above is by no means always an evenly horizontal one. As the case proceeds, and an increasing quantity of serum is effused, the dulness may extend quite up to the clavicle in front and to the supra-spinal fossa behind; after this, any further extension of the effusion necessitates expansion of the pleural cavity in some fresh direction. So far, space for the fluid has been obtained chiefly by the compression of the lung into the spinal fossa, but already, in most cases, there has been also displacement of the surrounding organs. This displacement may affect chiefly the ribs. But the diaphragm even more certainly yields, and its displacement downwards pushes the liver down (in right pleurisy), and percussion can recognize the depression of this organ; similarly, the stomach and colon may be recognized, by their tympanitic percussion-sound, at an unusually low level, and the spleen-dulness may sometimes be traced at a point below the margin of the right false ribs (in left pleurisy). The most striking phenomena of displacement are, however, connected with the heart. In left pleurisy a large effusion often pushes the heart so much to the right that the cardiac percussion-dulness is found to occupy a space beneath the lower end of the sternum, and even extending considerably beyond its right border. The displacing effect of right pleuritic effusion is less immediately obvious to percussion; though readily identified by inspection and palpation, or at any rate by the stethoscope.

Palpation often gives us very important information. In early stages, and in those cases where the effusion remains merely fibrinous, the grating of the lymph-covered pleural surfaces may communicate a thrill to the chest-wall which is appreciable by the hand; these phenomena, however, are not very frequently observable. More constantly useful is the absence of vocal fremitus when liquid has been poured out in any quantity; this is usually striking when we compare the pleuritic with the sound side. The fremitus of lymph-covered pleural surfaces may sometimes be felt in the later stages after the fluid effusion has become absorbed.

Auscultation rarely reveals much in our earlier examinations. Fluid has not yet been poured out. We may happen to catch the moment when the pleura is rubbing its two fibrine-covered surfaces together at some point or points, in which case the "friction sound" is heard accompanying inspiration and expiration. There is no use in attempting to describe this sound minutely; it does, in fact, considerably vary in pitch and in character; the student must himself repeatedly hear it, and compare it with other sounds (especially various *clicking* bronchial sounds) before he can identify it with confidence. The rarity with which, as I have said, it is heard in the early stages, holds good in the ordinary type of Pleurisy that goes on to liquid effusion; and in the wards of hospitals this is the prevailing type of the disease that is seen; but I have been for some years past surprised at the frequency with which I have detected slight and limited Pleurisy, by means of the friction-sound, in the out-patient room. Most of these cases were tuberculous; but a considerable number afforded no room for any suspicion of the kind. The friction-sound is far more commonly heard in the stage of resolution, where liquid is getting absorbed, and the roughened pleural surfaces come together again.

In the stage previous to fluid effusion, the ear detects only the fact that the lung of the healthy side is expanding more vigorously and noisily than the other. When fluid becomes effused, however, the tendency is at first often towards a bronchial character of the breath-sound, accompanied by bronchophony on the affected side, while the effusion is small. In adults, however, the progress of the effusion rapidly replaces this by weakening, and finally absence, both of breath and voice sounds; meantime the breathing on the healthy side is more and more noisy and puerile. On the pleuritic side, the lung getting pushed back into the spinal fossa there are bronchial breathing and bronchophony to be heard in the upper and inner scapular region and between the scapula and spine, and comparative or complete silence elsewhere.

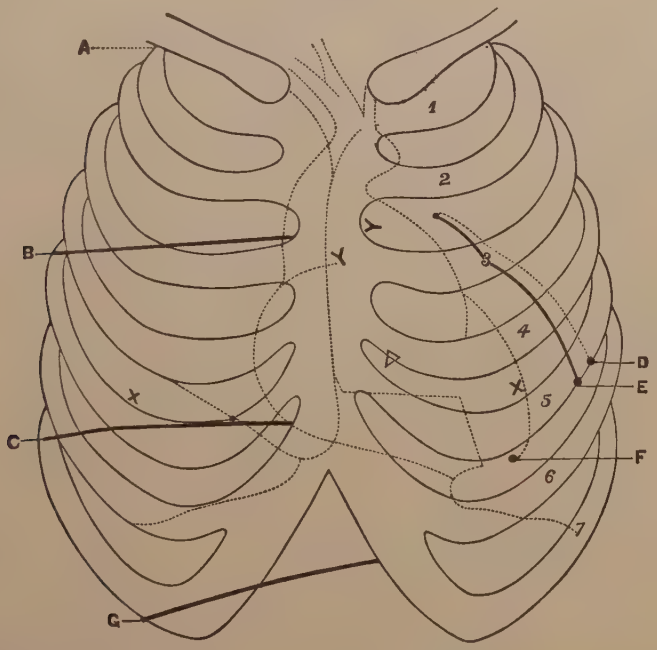
Of *ægophony*, the curious bleating sound of the voice which is sometimes heard at the upper level of the fluid, I feel inclined to say very little. It is in truth one of the *fancy* signs of Pleurisy—interesting rather than useful; it is so inconstant, and there are so many fallacies attending its recognition, that I believe it to be, for ordinary auscultators, rather a snare than a help. In a similar way one must speak of the *succussion sound*, the splashing noise supposed to be heard on shaking the patient; this also is very inconstant. And we may here notice that the changes, both of voice and breath-sounds, and also

of percussion sounds, which are commonly supposed to be induced by *changing the patient's posture*, are very uncertain and unreliable in true Pleurisy.

Auscultation is of great value in indicating the altered position of the heart, which occurs in cases of large effusion.

Physical Signs in Pleurisy of Children.—There are several most important variations from the above general picture of the physical signs of Pleurisy, to be observed in the pleurisies of infancy and childhood. These variations are due to two circumstances: the small size of the chest, and the greater yieldingness of the chest-walls. As regards the auscultation, it is all-important to note that bronchial breathing and voice persist, in nearly every case, even when the effusion occupies the whole chest, and when vocal fremitus is entirely absent. Rilliet and Barthez were the first to notice the remarkable fact that even a pneumonic bronchophony and bronchial breathing, so far from being diminished, are usually much intensified by a supervening pleuritic effusion. Yet there are many text-books that take no notice of this peculiarity of children, and ignorance of it has certainly been the cause of many disastrous mistakes in practice, the practitioner believing firmly that he had merely to do with a consolidated lung, till surprised by the appearance of fluctuation and evident signs of pointing in one or more of the intercostal spaces. Another very important distinction of Pleurisy in young life is the comparative *absence of signs of displacement of viscera*. The fact is that the chest-wall yields more easily, and the force of pressure is not expended, to anything like the same extent as in adults, in dislocating the heart and in driving downwards the diaphragm and the abdominal viscera. This, also, is a peculiarity too little noted in text-books written by those whose experience of Pleurisy is not large; and, joined with the persistence of bronchial breathing and voice, has doubtless caused numbers of mistakes.

Fig. 43.



A. Line of original upper level of fluid; B. Line of fluid on twentieth day; C. Conjectural upper level of liver; G. Termination of dulness below (tympanitis begins). D. Maximum heart-impulse at time of greatest effusion; E. Ditto on twentieth day; F. Ditto in natural state.

Such errors probably cost the lives of many patients who might have been saved by prompt tapping. It is, however, a mistake to say, as some have done, that displacement of viscera never takes place in children. Ziemssen[1] quotes a conclusive series of cases, observed by himself and others, to the contrary effect.

We must now complete the clinical history of Pleurisy by describing what may be called its critical symptoms. If the case takes the turn towards recovery by simple absorption, which is the natural destiny of primary Pleurisy, then, after

[1] Op. cit. pp. 67, 68.

the subsidence of pyrexia, there occurs, usually, a pause of a day or two, after which the work of absorption begins to show itself by physical signs, and by a small but increasing degree of relief to respiration. Among the signs most carefully to be looked for as indicating the commencement of this process is the return of the percussion dulness of the liver, or the tympanic sound of the stomach, to a higher level. The return of the heart to its proper position, even when absorption has made considerable progress, is not always rapid or at first very evident. In the accompanying sketch is represented, with rough but sufficient accuracy, the state of things in the chest of a young but intemperate man, C. J., who, between the sixteenth and twentieth days of right Pleurisy, experienced an amount of absorption of the effused fluid indicated by the distance between the lines A and B. The liver was believed to be somewhat enlarged and fatty in this man.

Instead of a speedy commencement of absorption, the fluid may remain in a passive condition, and the patient may continue in a state marked by no discomfort except some mechanical embarrassment of respiration and heart movements, impeding him in any but the gentlest movements. It is fortunate, but comparatively rare, when a protracted period of this kind is terminated by the occurrence of absorption. More commonly a slight but steady increase of ill-health is experienced, till at last there arises decided febrile disturbance, settling more and more into a hectic type, with copious sweat, morning remissions and evening exacerbations, and, in short, a more or less complete series of indications of extensive suppuration. It is here, at last, that the thermometer, so little to be depended on in other stages of Pleurisy, often gives us precious information of the changed aspect of affairs.

There is no need to carry the clinical description any further, since under the heads of Prognosis and of Treatment sufficient information will be found concerning the favorable and unfavorable termination of suppurative Pleurisy.

Complications and Sequelæ.—Of primary Pleurisy the most frequent complication is pneumonia, and this may either exist from the first or supervene at any tolerably early stage. It does not appear to occur with any frequency after the lung has been compressed into a small space by fluid exudation. Laennec believed that the compression by the fluid always tended to prevent the occurrence of *severe* pneumonia in connection with Pleurisy; but it will be seen, under "Prognosis," that he was at least wrong in this, so far as relates to *children*. But when the lung is compressed to carnification, it is doubtless very incapable of inflammation. The most formidable way in which pneumonia may complicate Pleurisy is where, a considerable effusion existing in one pleura, inflammation attacks the *opposite lung*. It may be doubted, however, whether this ever occurs in truly primary Pleurisy: personally I have never seen a case where inquiry did not show the existence of kidney disease, fever, pyæmia, or some of the many causes of secondary Pleurisy.

This is the place to speak of double Pleurisy, which may fairly be looked on as a complication; and in regard to it I can only repeat the same observation. Primary Pleurisy, as we call it, does seem, at any rate, peculiar in this—that it is an essentially unilateral disease: and I have never been able to see a double case in which there were not ample reasons for thinking the Pleurisy a secondary affection to some condition of general blood-poisoning. It is in the same point of view that we must regard the supervention of other serous inflammations, *e. g.*, peritonitis; but there is a possibility, perhaps, that *pericarditis* may sometimes arise by simple extension of the inflammatory process from the contiguous pleura.

In cases of empyema of some standing a not very uncommon complication is discharge of the pus through a pulmonary fistula into the bronchi; this is associated with phthisical lung disease in a large majority of cases, but a considerable number are recorded in which the accident has occurred in primary Pleurisy without tubercular disease.[1] The cases are rare in which the channel of evacuation has proved sufficient: usually the bronchial discharge is only a preliminary to subsequent perforation outwards, and as regards treatment this is the view that should be taken. The accident of pulmonary perforation must be looked on as the probable commencement of a period of *fetid suppuration* and pyo-pneumothorax.

Of the sequelæ of Pleurisy one outweighs all others in interest, viz., *tuberculosis*. It is now well established, not merely that Pleurisy often occurs in phthisical lung-disease, but that Pleurisy itself is capable of setting up true tubercle, even in previously healthy persons. This is specially apt to occur where a purulent effusion has been allowed to remain too long in the pleura, or where paracentesis has been performed repeatedly for empyema, the wound being closed in the interval. But the latter practice

[1] For a good discussion of the subject of bronchial fistula, see Aristide Attimont, "Résultats de la Paracentèse dans la Pleurisie purulente," Paris, 1869.

is one which, it may be hoped, will no longer be followed.

The other sequelæ of Pleurisy, though they may be very troublesome, are less important. Retraction of the chest-wall and consequent deformity of the spine and shoulder is the ordinary result of the absorption of a large effusion, where the lung has been too much bound by adhesions to re-expand at once, or perhaps at all. The same thing occurs where a collection of pus has been allowed to burst externally; here the lung is firmly bound down, and the orifice of the rupture being valvular no air enters the chest, and so the ribs sink in under atmospheric pressure. It may be at once said, however, that these deformities are merely temporary, and that with proper attention they will always be found to right themselves, in the course of a year or two, almost entirely; this is especially the case in children. As regards the fistulous opening left after the natural bursting of an empyema, the course of events depends on the amount of local mischief which was done during the passage of the pus to the surface; when this has been very tedious, more or less extensive destruction of periosteum and necrosis of ribs may occur, and may give much trouble. A single fistulous opening is merely to be looked at as an unpleasant fact which will disappear in a certain number of months or years.

Pathological Anatomy.—The first stage of change in every case of Pleurisy appears to consist of ordinary injection of the vessels beneath the pleural membrane; in primary cases by far the most frequently this change begins in the costal pleura. Slight ecchymoses are more or less plentifully scattered over the hypervascular and bright-red part. The clear serous membrane also begins to be clouded and swollen, and if the inflammatory process goes on for a very few hours, there occurs a visible deposit of fibrinous lymph, of a reddish-yellowish tinge, and at first very tender and soft, and small in quantity. If the inflammation goes on to be an affair of more than a day or two, not only does the amount of fibrinous deposit increase by successive layers, but a variable proportion of serosity is mixed with the lymph; and often serum is poured out in large quantity from an early period, so as to fill a large portion of the pleural cavity within a few days, more rarely within a few hours. There is great variability in the relative amount of the serous and fibrinous elements of effusion, but in general the contents of the inflamed pleura may be described as consisting of yellowish serum in which float a quantity of concrete masses of the same fibrinous matter which lines the inflamed portion of the membrane; and, as the case advances, bands of fibrinous matter, at first tender and yielding, afterwards firm and tough, form adhesions between the costal and the opposite portions of the pulmonary pleura. In some instances it happens that the fibrinous adhesions are so many and so dense over a limited area, that they inclose and limit the serous exudation, confining it to a comparatively small portion of the pleural cavity. In ordinary cases the pleural cavity becomes progressively and more or less evenly filled to a higher and higher level, the lung receding before the fluid, and being pushed upwards, backwards, and inwards, till it is compressed into the mediastinal or spinal fossa. On the other hand, it may happen that a comparatively small amount of fluid spreads itself rapidly over a large portion of the lung, and, though reaching a high level in the chest, does not greatly compress or alter the position of the lung, at any rate at first.

In those pleurisies where the inflammation is limited to a small area, the effusion often consists almost entirely of plastic fibrinous matter, and then the regular course of the affection is short, ending in an adhesion of a limited portion of the opposed pleural surfaces. Undoubtedly the most frequent examples of this kind are found to occur in the course of chronic pneumonia and of phthisis; but it is certain that they also occur, sufficiently often, in individuals who are otherwise apparently healthy.

[Fig. 44.

Inflammation of the Diaphragmatic Pleura.—Showing the adherent fibrinous layer. *a*. Muscular coat of diaphragm. *b*. Subserous tissue. *c*. Serous membrane. *e*. Fibrinous layer. X 400. (Rindfleisch.)]

Another outcome of inflammation is the effusion of pus, which may either exist from an early period or may slowly develop in the course of an ordinary fibro-serous pleuritic effusion, the pus-cells more and more invading the serosity, until at last the whole mass of fluid assumes a truly purulent character. Pus in the pleura is known under the name of empyema. *Acute* empyema is rare as a

primary disease in adults (more common in children), and is usually the direct result of *injury*, but is common enough as a complication or sequela of certain acute fevers, especially scarlatina, and also pyæmic poisoning; it occurs also in a certain small number of cases of pulmonary phthisis; and one special variety—pyopneumo-thorax form of perforation of the pulmonary pleura—is in such a large proportion of instances due to chronic lung-phthisis, that it may for practical purposes be almost entirely left for consideration along with that malady.

The fibrinous element is, I believe, never really absent from a genuinely pleuritic effusion: many times as I have looked for such a thing in the post-mortem room, I have never seen a purely serous nor a purely purulent Pleurisy. [The cases of miscalled purely serous Pleurisy are always merely hydrothorax, occurring either as a mechanical result of embarrassed circulation, or else as a consequence of poisoned or depraved blood.] The extent to which the fibrinous element exists varies from a slight coating of soft lymph upon limited portions of the pleura (both costal and pulmonary), with some light floating flakes in pretty clear serous fluid, to a dense cortex enveloping the whole of the lung and coating the whole of the pleura, and from one to several lines in thickness; the latter condition is only produced in old-standing cases, and the lymph is deposited in concentric layers of which the external are yellow and rather soft, the deeper ones dense and tough, reddish in color, and exhibiting traces of vessels. The longer the pleural cavity remains distended with fluid, the more firmly the lung is bound down to the neighborhood of the vertebral column; and if the conditions of mechanical pressure last long enough, the fibrous adhesions grow too dense ever to be removed so as to allow the lung to expand again. The final result, in cases of recovery, is the conversion of the layer of fibrinous lymph into a rudely organized cellular tissue, bands of which also stretch between the lung and the chest-wall, and either bind them firmly together, or (according to their length) allow more or less free play. In exceptional cases the whole surface of the lung is left firmly adherent to the chest-wall by a layer of fibrinous matter, which may vary in texture from that of loose cellular tissue to a tough semi-cartilaginous material. The latter condition has been occasionally seen in cases where, apparently, there has been little or no serous effusion, and where the lung, though thus universally coated, has been found (after death from some other disease) fairly expanded and permeable to air.

If we now inquire into the minute anatomy of these changes, we find that the earliest stage, beyond that of mere congestion of the subpleural vessels—that, namely, of cloudy swelling of the membrane—is microscopically distinguished by the appearances of proliferation of the epithelial cells, which tend more and more to multiply and also to be shed from the surface.[1] Very soon there appear, also, masses of fibrinous materials which have nothing to do with epithelial changes, but apparently exude directly from the bloodvessels, and belong to the same process by which the serous effusion is poured out. The proliferated epithelial cells, together with exuded blood-cells, form the cellular element of the fluid; and upon their numbers and the stamp of their vitality depends the question whether that fluid shall turn to pus or not; they are present, along with fibrinous matter, in the flocculi which float in the fluid.[2]

As to the retrograde changes which take place in the solid matters when absorption takes place, the most important matter is this. If the cellular elements are in large quantity, the retrograde process is slow, and passes through a stage of cheesy formation, which may be very lingering: and there is much probability that this state, though not so frequently as the long-continued persistence of a purulent effusion, may give rise to tubercle. Where a very abundant and thick fibrinous deposit, with scattered cells, is the only thing left after the liquid has disappeared, there is still some danger: part of the material must pass through the stage of caseous formation: and it will be well if the patient escapes with a thick, almost cartilaginous, coating of his lung, scattered with calcareous deposits. The most favorable result is when the only trace of the effused matters is a few adhesions, composed of cellular tissue, between the lung and the chest-wall. Such appearances are, as is well known, among the commonest things found after death in the pleura even of persons who may never have been conscious of the pleuritic attacks at the time of their occurrence.

The condition to which the lung is reduced by the pressure of the effusion and the strangulation induced by the compressing fibrous adhesions, is of the highest importance. In simple Pleurisy, without pneumonic complication, the lung, pressed back by the side of the spine, is reduced to a state of so-called *carnification:* the tissue is hard and unyielding, and does not crepitate under pressure. This is the effect of extreme compression; when the effusion has been

[1] Rindfleisch, Handbuch der pathologischen Gewebelehre: Leipzig, 1869.

[2] See the striking engraving in Rindfleisch, op. cit. p. 211.

small, the lung-tissue may still be partially crepitant. Even the extreme degree of carnification does not seem to exclude the possibility of re-expansion, if once the pressure were fairly taken off. The danger is rather—especially when the fluid has been purulent—that cheesy masses, and even true miliary tubercle, may develop within the compressed lung. It is in these conditions that the re-expansion of the lung becomes almost beyond hope.

On the other hand, when there has been antecedent pneumonic inflammation, the lung may never become greatly compressed from first to last; it will be found heavy and solid, much less resisting to the finger than true carnified tissue, perhaps still crepitant, but presenting the characters of hepatization.

DIAGNOSIS.—The most frequent source of fallacy is confusion between Pleurisy and pneumonia.

In both diseases there are fever, dyspnœa, and cough. But in primary Pleurisy the temperature rarely attains a high grade, especially in early stages; while in pneumonia it is not unusual for the thermometer to reach 103° or 104° within the first twenty-four hours. The skin is much more dry and burning to the touch in pneumonia than in Pleurisy: the flush on the face more fixed, and often remarkably unilateral. The feeling of dyspnœa is often much more remarkable in Pleurisy than in pneumonia, but the relative frequency of respiration and pulse is more altered in the latter. The cough in Pleurisy is short and hacking, but attended with no expectoration or with only the discharge of a little thin mucus; whereas in pneumonia expectoration is present in an immense majority of cases, and soon becomes "rusty" in color, and very tenacious. Sharp stitch-like pain in the side is a very frequent characteristic of Pleurisy, whereas in pneumonia there is commonly no pain, or else a much duller and more diffused pain. As regards physical signs, the dulness on percussion is more absolute in Pleurisy than in Pneumonia, and as the case proceeds the breath-sounds and voice become weakened and finally abolished in the former, while they become more and more "bronchial" in the latter. [This is true of adults, but in children bronchial breath and voice persist in Pleurisy.] The vocal fremitus becomes weakened and finally abolished in Pleurisy; it increases in pneumonia as the consolidation proceeds. Displacement of the neighboring viscera is never seen in pneumonia; it is common in Pleurisy, especially in adults. Increase in the volume of the affected side, with widening and bulging of the intercostal spaces, and in extreme cases fluctuation there, are characteristic of Pleurisy, but not of pneumonia.

Several of the above remarks are chiefly applicable to primary Pleurisy, which, as already stated, is nearly, if not quite, always a unilateral disease; whereas pneumonia is more frequently than not bilateral. More absolutely distinctive of Pleurisy, however, is the absence of that fine hair crepitation which in pneumonia precedes consolidation and establishment of bronchial breathing and voice. Where the chest affection is only secondary, Pleurisy is frequently double, and much of the value of comparison of the two sides is lost. Here the percussion and auscultation sounds require to be more finely appreciated; and the presence or absence of special sounds, like the fine pneumonic crepitation, is of the greatest importance. The possibility of the coexistence of pneumonia and Pleurisy must always be kept in mind; and when to fine crepitation, mixed increasingly with patches of bronchial respiration, there succeeds a weakening and then absence of breath and voice sounds, we have good *primâ facie* reason for thinking that the latter disease has supervened upon the former.

Undoubtedly the most generally serviceable physical characteristics of Pleurisy are the combination of very pronounced percussion dulness, absence of vocal fremitus, loud bronchial breathing limited to the superior internal and intrascapular space, and (where the pleura is not yet full) tympanic percussion and almost cavernous respiration just below the clavicle, with more or less complete absence of breathing elsewhere.

The diagnosis of Pleurisy from simple *hydrothorax*—passive exudation resulting from mechanical obstruction of circulation, or in chronic blood-poisoning—rests chiefly on two facts: the absence of fresh febrile disturbance, and the more generally double effusion in the latter affection. Often there is corroboration of these indications, in hydrothorax, from the simultaneous occurrence of dropsical effusions in other parts.

Pleurisy is to be distinguished from *pulmonary phthisis* by the history of the attack, the absence of characteristic expectoration and emaciation, the physical signs of fluid effusion, the family history, &c. But as regards limited attacks of merely fibrinous pleurisy, it may be very difficult to say whether or not there is phthisis also, the form of pleuritic attack being a very common complication of phthisis at all stages.

The presence of a solid tumor, occupying a considerable portion of the pleura, or bulging into it from the mediastinum, may closely simulate most of the physical signs of Pleurisy. We must here depend mainly upon a very accurate inquiry into the history and the "rational" symptoms:

the absence of all febrile disturbance at the commencement will assist our judgment.

But although I might draw out to a much greater length this catalogue of the possible snares which lie in wait for us in the diagnosis of Pleurisy from affections more or less resembling it, I think this unnecessary, because the means of discrimination are now augmented by a most potent test, the modern practice of exploratory puncture. We may fairly say that, with the assistance of the small trocar, fitted with the glass vacuum syringe, it is possible for us to make a puncture into a chest without the least apprehension of damage, whether the trocar shall enter a pleuritic effusion, a hepatized lung, a cancer, or even an aneurism; and with the great advantage of discovering whether there is fluid at all, and if so, what the fluid is. It is unnecessary to say, that except under the stress of urgent symptoms, this should not be done while high febrile excitement is present, unless there is strong probability that fluid effusion is the sole cause of the maintenance of the fever.

Prognosis.—The prognosis of primary Pleurisy is very favorable, though there is by no means that complete immunity from fatal consequences which was asserted by Laennec and Louis. The danger of sudden death from orthopnœa was shown by Trousseau to be a serious one in a small percentage of cases—more especially the *latent* type, with insidious commencement—when the effusion completely fills one pleural cavity; and at the present day it is generally acknowledged that this is a real peril. For prognostic purposes it is now pretty well understood that it is not the mere quantity of the effusion that should most alarm us: the rule is that, large effusion being present, the occurrence of one or more attacks of *severe dyspnœa—orthopnœa*—indicates a dangerous want of tolerance by the organism, and calls for direct interference.

The other danger which must be reckoned with is where a primary pleuritic effusion has remained stationary in the chest without any tendency towards absorption for a considerable period, and signs of its conversion to pus, with attendant severe hectic fever, increasing emaciation, and general prostration of vital power, show themselves. Here the least of dangers is that involved in protracted suppuration: far more formidable is the risk, now well established, of an infective absorption leading to tuberculosis.

It must be said, however, that both the chance of suffocation from mechanical pressure, and the risk of secondary tubercular processes, are indefinitely diminished by the modern practice of prompt paracentesis. It may be questioned whether the experience of the next twenty years will not enable us to ensure an absolute immunity from fatal results of either of these complications.

Very different is the prognosis in secondary pleurisies: though even here we may reckon on a considerable percentage of recoveries. Most fatal of all is the Pleurisy which occurs in the course of pyæmic (*e. g.*, puerperal) infection; here, death is the rule, recovery a rare exception. A considerably smaller mortality, but still a very high one, attends the cases which supervene on scarlatinal albuminuria; and a somewhat similar ratio of deaths, though scarcely so high, attends all pleurisies secondary to the acute general fevers. But the fact is that every case of Pleurisy supervening on a constitutional fever has its individual prognosis depending on the time of its occurrence, the amount of vital resistance which the original disease has spared, &c.; and we are driven here to a minute observation of particular symptoms. It is here that thermometry plays an invaluable part. The following conclusions of Wunderlich, respecting temperature in serous inflammations generally, apply, according to my experience, with admirable correctness to pleurisies secondary to fevers:[1] "Subnormal temperatures are always highly suspicious; death occurs either shortly after their first appearance, or when they have persisted for some time, or have alternated with normal and excessive temperatures. Temperatures of considerable, especially of increasing height, though not necessarily in themselves of bad omen, yet add something to the dangerous momenta. If the temperature falls again, the danger is not past, yet it is less threatening than if the heat had been maintained. Besides the height of the febrile temperature, its constancy, and the absence of remissions, especially heighten the peril; more particularly the long continuance of a high temperature, even if it alternate with considerable morning remissions. In the first case the disease is dangerous, in the latter complete recovery is at least doubtful. . . . . Very considerable and irregular fluctuations between the highest and the lowest temperatures (resembling those of pyæmia) occur, especially in endocarditis; occasionally also in pericarditis, pleurisy, and peritonitis; they are always extremely dangerous, and a fatal result is very probable."

To this element of prognosis let me add examination of the pulse with the sphygmograph, of the value of which I can hardly speak too strongly. The subject would occupy too much space in discussing here; but I would refer the reader to

[1] Wunderlich, op. cit. p. 375.

what I have written elsewhere[1] respecting the favorable and unfavorable pyrexial pulse-forms, and shall merely say that subsequent experience has strengthened my convictions already expressed. I believe that in the dangerous secondary pleurisies the combined use of thermometer and sphygmograph is more valuable for prognostic purposes than all other modes of observation put together.

As regards the prognosis of Pleurisy secondary to pneumonia, it may be said, in general terms, that the amount of danger depends entirely on the moment at which the Pleurisy supervenes. If the system has been severely tried, the chances are bad: thus, Rilliet and Barthez reckoned eight deaths out of ten such cases.

Of Pleurisy secondary to phthisical lung-disease, as already said, the prognosis is usually very favorable, for the moment at any rate; but there is always the danger that any fresh pleuritic process may be the starting-point for a true tubercular infection. And, on the other hand, the subjects of tubercle who (not a very common case) develop extensive Pleurisy with liquid effusion, nearly always die.

Be it said, however, that the increasing tendency to paracentesis, even in secondary pleurisies, will not improbably result in a greatly decreased mortality, even from the most formidable varieties of the disease. It is almost impossible to rate too highly the significance of such a case as that of Kussmaul, hereafter to be cited (*vide* Treatment).

There is one variety of secondary Pleurisy of which I must say a few words here, because it is scarcely discussed in the text-books, viz., Alcoholic Pleurisy. Except in the advanced stages of chronic alcoholism, supervening Pleurisy is rarely of bad prognosis: nearly always it leads only to a certain amount of fibrinous exudation and proliferation of connective tissue. It is only in the last stage of drink-degeneration that a fatal form of empyema is apt to develop itself: I have seen only one such case purely traceable to the results of drink alone, but there are a considerable number of cases in which the fatal result is, perhaps, equally due to this influence and to blood-poisoning from renal disease.

TREATMENT.—The treatment of Pleurisy is naturally divided into that of the primary and that of the secondary forms.

Primary Pleurisy, of a well-marked type, is perhaps as little the fit subject of treatment by drugs or other artificial means, in its acute stages, as any disease that could be named, or rather, the drugs needed are very few, and all of the stimulant-narcotic class. For the vast majority of patients, indeed, the only drug which is of considerable value is opium in one or other form, until the febrile period has passed over, when preparations of iron sometimes become very useful. I do not make this statement without having carefully watched and considered the effects of a number of internal remedies which are still used as a matter of course, and indeed considered essential, by various physicians of good repute.

To take, first, the case of primary simple fibrinogenic pleurisy, one may at once decide against all heroic remedies, since evidence abounds on all sides to show that the disease is a perfectly harmless one, unless the patient has strong tendencies to constitutional disease, and that it tends always to recovery. In fact, one has no need to adopt any treatment whatever beyond keeping the patient in one room, free from draughts, and in the posture which he finds easiest to him; feeding him steadily with nutritious food of the kind best adapted to the degree of fever and digestive derangement that may happen to be present: forbidding unnecessary movements and talking; applying hot poultices to the side, and administering an occasional hypodermic injection of $\frac{1}{6}$ or $\frac{1}{4}$ grain morphia to keep the pain in check. Acetate of ammonia, in doses just short of those which produce decided sweating will sometimes greatly relieve the pain and distress even without the aid of opium, and is at all times a harmless, even if an unnecessary medicament. Recently, the acetate of methylamine (a base which exists in roasted coffee, owing to the transformation by heat of a part of the coffeine) has been proposed, and apparently used with good effect, by Professor Béhier of Paris.[1] There is usually no necessity for alcohol, and it had better be avoided. After from six to seven days in bed, the patient will probably be well able to sit up, and the only thing necessary to forbid to him is *movement*. He should sit perfectly still. If any anæmia remains, the tincture of muriate of iron, in twenty-minim doses thrice daily, is advisable as a tonic; and, on the whole, a very few days ought to see the patient completely fit to resume his ordinary work.

In Pleurisy evidently of considerable extent, and with a notable amount of *serous effusion*, the ideal of treatment should be still, as much as may be, that given above. It is now very decidedly proved

[1] Lectures on Acute Diseases, delivered before the Royal College of Physicians. (Lancet, 1867, vol. ii.)

[1] See a paper in the Practitioner, October, 1868, "On Tonic Medication and on Acetate of Methylamine: a new tonic remedy." By MM. Béhier and Personne.

that the old heroic methods of attacking severe Pleurisy ought to be abandoned. In the first place, as to general blood-letting, I have witnessed enough of this treatment to be sure of two things: first, that the older physicians were perfectly right in the statement that it usually relieves *pain* with great promptitude; and secondly, that the relief thus given is not in the least degree superior to that afforded by hypodermic injection of morphia, except that it operates more quickly, perhaps by some five minutes, than the latter. As to bleeding checking the tendency to effusion, *that* is to me quite incredible. No such effect has been witnessed in either of the five cases of phlebotomy for acute Pleurisy that I have watched at various times; and I observe that Dr. Aitken,[1] while still adhering to the use of this remedy, recommends us not to be discouraged by the fact that the effusion may go on increasing after the bleeding, and the patient also may feel very depressed. It is true, he says, that after a certain time absorption will set in, and that it will then go on more rapidly and well than if the patient had not been bled. I cannot at all imagine on what evidence this last opinion is based; certainly it utterly conflicts with the fact of my own experience; and though I have personally seen little of the actual treatment of Pleurisy by bleeding, I have examined a pretty large number of persons whose past history included one or more pleuritic attacks which had been so treated. The accounts given by such persons show a melancholy uniformity: long weeks and months of suffering from the presence of effusion in the chest, occasionally leading (through empyema) directly into active and rapidly fatal tuberculosis, nearly always slow and imperfect recovery, with diminished vital energy, and especially weakness in the chest, and only in the rarest cases a tolerably prompt and complete recovery. The homœopathists have made their fortunes in no small degree by their "treatment" of Pleurisy, which has had the one sole merit of being purely negative, and avoiding all destructive agencies. [In view of Dr. Anstie's statement (above) that he had seen little of the treatment of Pleurisy by venesection, it may be pardonable to refer to his expression on a later page, in regard to the opposition to paracentesis: not "by men who have fairly tried the practice, but only by theorists who are afraid of its imaginary results." It is hardly too much to say, that those who decry bleeding now maintain their position almost entirely upon theoretical grounds.[2] It is true, however, of Pleurisy as it is of pneumonia, that not nearly all cases require, or will properly bear, venesection; and that almost never will it be appropriate later than the third or fourth day of an acute attack.—H.]

A much better case, no doubt, might be made out on behalf of local blood-letting. Cupping ought never to be mentioned, being actually barbarous in the suffering it inflicts on a pleuritic patient. But leeches unquestionably do relieve pain very often in a speedy and effectual manner, and I only know of one objection to their use, viz., that morphia will relieve the pain with even greater certainty. During five years of dispensary practice I determinedly abstained from the use of leeches in Pleurisy and found morphia, even given by the mouth, a perfectly satisfactory substitute. But since the use of the hypodermic syringe has become more common, the advantages of morphia are far more manifest; and I have no doubt, personally, that leeches are now unnecessary. The first act of the physician in treating a pleuritic patient in the agony of the early acute stage, should be to inject $\frac{1}{6}$ or $\frac{1}{4}$ grain of acetate of morphia (for an adult) under the skin,[1] and to envelop the painful side in a hot poultice. For a child under 2 years, $\frac{1}{40}$ or $\frac{1}{30}$ grain is enough. Such doses as these may be repeated every four hours, if necessary; but in fact it is seldom that more than two or three doses are needed in the first twenty-four hours, and afterwards one dose in each twenty-four hours is generally enough.

I would insist strongly on the advantages, indirect as well as direct, of subcutaneous over gastric administration of opiates; in a direct way, the former is superior as acting much more rapidly; in an indirect way, because it so much less disturbs the functions of the alimentary canal.

Of the treatment by mercury, I can express only the most unqualified disapproval. I have watched many cases of Pleurisy in which, according to the rule formerly acknowledged, mercury was given, either to complete or partial salivation, as soon as the signs of effusion became unequivocal, and I can truly say that these cases, even when they were not further complicated by the depressing influence of blood-letting, contrasted very unfavorably with the results of a treatment which entirely abjures mercury for

[1] Science and Art of Medicine, 3d edition, vol. ii., article "Pleuritis."

[2] Professor N. Chapman, of the University of Penna., used to say to his class, that, in a long experience in practice, he had never had occasion to regret having employed the lancet; while he had often seen reason, too late, to regret that he had not used it.—H.]

[1] I believe, with Mr. Hunter, that there is no need to inject locally: the arm does quite well for the purpose.

any purpose except that of an occasional purgative. I am glad to cite, on this point, the late Dr. Hillier, who says (in his Monograph on Children's Diseases) that from experience he had been led to abandon mercurial treatment for Pleurisy ;[1] and I believe that, whatever some of the class-books may still say, mercury is practically given up by the best physicians in this country, not only in children's pleurisy, but in that of adults. It seems the general opinion among those with whom I have conversed, that the absorptive action with which mercury used to be universally credited is more than doubtful in the case of pleuritic effusions, whether fibrinous or serous. And certainly, if it fails to do good, mercury may do very sensible harm. I have seen cases in which it apparently produced the most decided anæmia—at least there was scarcely any other possible cause for the latter condition—which set in rapidly after the first occurrence of ptyalism.[2]

The treatment by so-called "counter-irritants," as pursued by many physicians, is no less repugnant to me than is that by mercury or bleeding. Let me make two admissions. In the first place, the mere application of a mild mustard plaster, or, still better, of a hot poultice, or epithem, undoubtedly may give some ease ; perhaps even arrest incipient inflammation ; and the use of *small flying* blisters, in the limited attacks of Pleurisy which are so common in phthisis, undoubtedly appears to give relief in many cases. But the use of large blisters, especially if kept open, appears to me both useless and often prejudicial. I shall not repeat here what I have said at length elsewhere ;[1] suffice it to say that I adhere to my opinion already stated, which is the same as that previously announced by many of the greatest masters of practical medicine in the present century.

The practice of painting the chest-wall with iodine, though not open to the same positive objections as apply to blistering, has never, in my experience, yielded any very positive results. It is, I believe, very inferior in utility to the application of the simple adhesive or the Burgundy pitch-plaster, to afford mechanical support ; this really does sometimes appear to favor absorption of the fluid, and it usually gives much comfort.

The employment of diuretics to promote absorption is another point on which I find myself at issue with the opinions of many. The only drug which has appeared to me, in some cases, directly to promote absorption by means of increased diuresis, is iodide of potassium, in quantities amounting to from 6 to 18 grains daily, according to the age of the patient. I think it is worth trial for two or three days (along with the external use of iodine) when effusion comes to a standstill. [Dr. Da Costa, in a case of chronic pleuritic effusion, recently reported, gave a drachm of jaborandi, four times daily ; with the effect of profuse diaphoresis, followed by disappearance of the fluid, and recovery.—H.]

The medicine, however, which stands quite alone in its power to promote the process of absorption is *iron*—best given in the form of the *muriated tincture;* and in all cases where there is marked anæmia it should be exclusively employed from the moment when the necessity for administering opium ceases.

As regards purgative medicines, the utmost that I can recommend is that, if necessary, such mild medicines may be used as may suffice to prevent actual loading of the bowels, which, especially in the case of children,[2] might seriously increase the mechanical distress in the chest. Actual purgation is always mischievous in Pleurisy, although it is sometimes very useful in hydrothorax.

The use of alcohol is a matter requiring much care and judgment. In primary acute pleurisy it is usually best dispensed with, unless the patient is unable to take other nourishment ; in this respect Pleurisy differs much from pneumonia. But in secondary pleurisies stimulants will often be needed, and here the amount of the dose must be ruled, not by any rou-

[1] See also Meigs and Pepper on the Diseases of Children.

[2] I cannot help making a digression here on the subject of the supposed absorptive action of mercury on inflammatory lymph. So repeatedly have I seen attempts made, without one particle of success, to induce the absorption of pleuritic, peritonitic, and pericarditic lymph by means of this drug, that I have seriously reflected on what could possibly have given rise to the old unreasoning confidence in its power to act in this way. After the best inquiry possible to me, it seems pretty certain that the only groundwork was the assumption of a necessary analogy between *lymph effused in the iris* and that effused elsewhere. Now, to say nothing of the special relations (unintelligible, no doubt) of mercury to *syphilitic* products, it is certain that mercury possesses a strong physiological predilection for the whole territory innervated by the trigeminal nerve ; and I believe that there is something quite peculiar in its action on the nutrition of the eye, the mouth, the nose, and the face, and on the pathological products of inflammation in these parts.

[1] "On the Popular Idea of Counter-irritation," Lancet, Feb. 26, 1869 ; "The Theory of Counter-irritation," Practitioner, April, 1870.

[2] Ziemssen (op. cit.) particularly points this out.

tine, but according to those indications of the pulse, the temperature, and the urine, which I have fully described in my lectures on Acute Diseases, at the Royal College of Physicians,[1] and elsewhere.[2]

As regards all other matters in the treatment of secondary pleurisies, it is absolutely necessary that I should leave them to be dealt with by the authors who describe in this "System of Medicine" the various diseases of which Pleurisy is apt to be a complication.

One word must be said about a mode of treatment for Pleurisy which I confess that I have never attempted: I mean the employment, so common on the Continent, of *cold* to the chest, and the use of cool baths. I desire to pronounce no judgment whatever on the matter; but those who wish to know more of the system should study the remarkable statements of Niemeyer,[3] a very trustworthy witness, as to the effects of ice-cold applications to the chest.

Paracentesis Thoracis.—A new era has been inaugurated in the treatment of Pleurisy by the development which the operation of tapping the chest has received within the last few years. There is practically no use in going back further into the history of the operation than about thirty years; previously to this there was no real certainty or agreement as to its use except as a *last resort*. It was Trousseau who first had the acuteness and courage to lay down the proposition that in extensive effusions, whether of serum or pus, we ought not to wait till death is imminent, but operate with the view of warding off the dangerous attacks of orthopnœa which, as he proves by a series of remarkable cases, may unexpectedly seize the patient, and carry him off with great rapidity. Trousseau, however, encountered great opposition, both in his own country and elsewhere, and although some of his brilliant results undoubtedly startled the medical world, it may be doubted whether the operation would not have been relegated, after his death, to its former limited sphere, had it not been for the interposition of a very able and clear-sighted American physician, Dr. Bowditch.[4] This gentleman had long felt the futility and the culpable inefficiency of treatment which allowed patients to suffer the misery and danger involved in the retention for months together of fluid in the pleura, but it was not until the invention by Dr. Morill Wyman of his excellent suction instrument, that Dr. Bowditch saw his way to the safe and effective performance of paracentesis on the large scale. From that date (1850) till the present time Dr. Bowditch has performed the operation 250 *times, in* 154 *persons, without once seeing any evil, or even any very distressing symptoms resulting from it;* while, on the other hand, it has saved a large number of lives that must otherwise have been sacrificed. "Surely," as the author remarks, "this amount of experience by any one deserves the attention of the profession." To this I warmly assent, and must add that there appears to me to be no opposition to Dr. Bowditch's views by men who have fairly tried his practice, but only by theorists who are afraid of its imaginary results.

Formerly paracentesis was supposed to have two functions only in Pleurisy: that of averting suffocation which was *actually* impending, and that of letting out collections which were pretty certainly conjectured to consist of pus. But against these advantages were to be set, it was thought, the fact that the fluid would inevitably reform, and re-form *ad infinitum*, and after very few tappings would become purulent (even if air could be excluded from the pleura, which was held almost impossible), thus surely undermining the patient's constitution. But the great and dreadful danger was that of admitting air into the pleural cavity; that, it was said, inevitably led, not merely to a continuance or aggravation of the purulent formation, but also to the putrescence of the pus, and the rapid depression of the vital powers under the combined influences of profuse suppuration, the absorption of noxious gases, and often the absorption of matter capable of inducing pyæmia. Tapping was therefore held to be inapplicable to the treatment of a merely serous effusion which did not immediately threaten life from mechanical pressure. This feeling prevailed the more strongly because some of the greatest masters of medicine of the present century had declared that primary Pleurisy, with proper medicinal treatment, should never be fatal; while in secondary pleurisies it was felt that an element of uncertainty underlies the whole prognosis, which disinclines the physician for doubtful, and possibly dangerous, modes of treatment.

It can hardly be doubted that the whole feeling about the dangerousness of paracentesis rested upon the use of clumsy and imperfect means of operation, and on exaggerated ideas of the evil effects of admitting a small quantity of air into the pleural sac. With regard to the first

[1] Lancet, vol. ii., 1867.

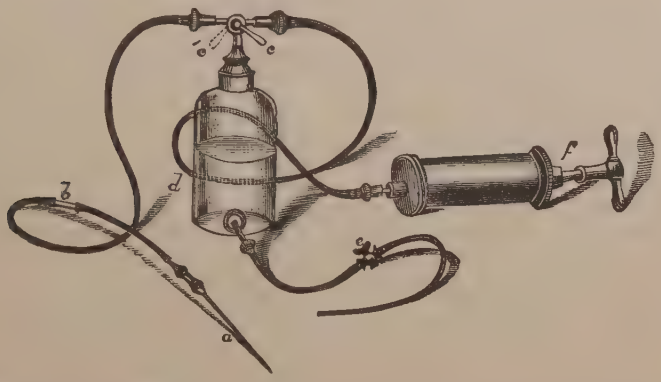
[2] Practitioner, "Wines in Acute Diseases," August, 1870.

[3] Handbuch der Speciellen Pathologie u. Therapie, vol. i.

[4] Dr. Bowditch's original papers are in American Journ. of Med. Science, April, 1852; American Med. Monthly, January, 1853; Boston Med. and Surg. Journ., May, 1857. See also his final paper before New York Academy, 1870.

point, we are entitled to say that it is quite possible so to operate as to ensure that no damage will be done to viscera, and that no more than a trifling quantity of air will be admitted to the pleura. And upon the second point we may certainly now assure ourselves that there is no reason to fear serious mischief from the admission of a limited quantity of air if the opening made in the operation be afterwards properly closed. It is even unnecessary, as Dr. Bowditch's large experience has shown, to make the opening valvular. But the most important advance that has been made is the invention of apparatus which allows of the operation being made either simply exploratory, or carried on at once to evacuation of the fluid. With the instrument either of Bowditch or of Dieulafoy[1] we introduce a very small trocar and canula guarded with a tap, and by attaching a suction syringe and opening the tap, we withdraw a small amount of fluid, the exact nature of which we can identify: if we elect to continue the evacuation, we can do so with the aid of the syringe; if, on the other hand, no fluid can be obtained, the guard-tap has prevented the entrance of air, and we can withdraw the canula and close the wound without having done the least mischief. By the use of the small canula we are able to operate without risk, because, in the case of an

[Fig. 45.



The aspirator. *a*. The perforated needle or sharp-pointed canula, which is introduced into the collection of fluid. It communicates with the bottle, *d*, by means of an india-rubber tube, which is interrupted at *b* by a portion of glass tubing, so that the nature of the fluid evacuated can be judged of at once, and the canula either plunged deeper or withdrawn. When the handle, *c*, is in the position shown, the communication between the canula and the bottle is closed. The bottle is then exhausted of air by means of the pump, *f*. When *c* is moved to *c'*, the canula-tube is opened. *e* is the waste-tube of the bottle, and is closed by a button at *e*. In using this aspirator the vacuum is formed, and the handle, *c*, is kept in the position shown till the canula has been introduced into the fluid, then it is turned to *c'*, and the fluid fills the bottle. If there be still more fluid, the handle is turned back to *c*, the waste-pipe opened, and the fluid emptied out of the bottle, which is then again exhausted, and the handle turned back to *c'*. This is one of the simplest of the many forms of the aspirator. (Holmes.)]

entirely mistaken diagnosis, we should have done no damage, even though we had perforated a consolidated lung, a solid tumor, or an intercostal artery. The suction power of the vacuum-syringe will enable even thick fluid, such as somewhat concentrated pus, to be withdrawn through the smaller-sized canulæ; but the puncture is such a trifle that, in case of our desiderating a larger tube, the smaller one can be withdrawn, the finger being pressed on the spot as it emerges, and the more capacious canula introduced at the same place.

The site of puncture should be selected in ordinary cases according to Bowditch's rules: Find the inferior limit of the sound lung behind, and tap two inches higher than this on the pleuritic side; at a point in a line let fall perpendicularly from the angle of the scapula. Push in the intercostal space here with the point of the finger, and plunge the trocar quickly in at the depressed part; be sure to puncture rapidly and to a sufficient depth, or you may be balked by the false membranes occluding the canula.

It will sometimes happen that with the greatest care and trouble we are unable to get a flow of fluid at the point where we first puncture; it is then our duty to try elsewhere, for our failure may be owing to unusual thickness of the false membranes in the lowest inch or two of the pleural cavity. We thereupon repeat the puncture a little higher up, and further

[1] It is right to say that Dr. Protheroe Smith claims, with apparent reason, to have been the actual inventor of the instrument now made by a French instrument maker, and employed by Dr. Dieulafoy.

towards the axillary line;[1] and here we perhaps find fluid: at any rate, no harm has been done by the two punctures.

The circumstances under which paracentesis ought to be performed for Pleurisy are the following:—

1. In all cases of Pleurisy, at whatever date, where the fluid is so copious as to fill one pleura, and begins to compress the lung of the other side; for in all such cases there is the possibility of sudden and fatal orthopnœa.

2. In all cases of double Pleurisy, when the total fluid may be said to occupy a space equal to half the united dimensions of the two pleural cavities.

3. In all cases where, the effusion being large, there have been one or more *fits* of orthopnœa.

4. In all cases where the contained fluid can be suspected to be pus, an exploratory puncture must be made; if purulent, the fluid must be let out.

5. In all cases where a pleuritic effusion occupying as much as half of one pleural cavity has existed as long as one month, and shows no sign of progressive absorption.

The *limits* of the operation form an important question. Formerly one great error seems to have been, that operators were often too anxious to extract the whole of the fluid; in this way they often protracted the operation to a mischievous extent, and gave abundant opportunity for that very entrance of air to the pleura which was theoretically so much to be dreaded. Among the latest writers, Bowditch and Murchison[2] have most authoritatively shown that it is neither necessary nor useful to extract the whole of the fluid, and that the removal of just so much as may be necessary to relieve substantially the mechanical distress will in most cases give the necessary spur to the natural process of absorption, by means of which the rest of the fluid will be taken up. One rule seems absolute: the withdrawal of fluid must be arrested the moment that the patient begins to complain of constricting pain in the chest or epigastrium. Even in the case of purulent effusion there can be little doubt that absorption often takes place, though unquestionably there is here a danger that concrete cheesy matter may be left unabsorbed, and under unfavorable circumstances may become the starting-point of tubercular infection.

The case of Pleurisy in *children*, as regards paracentesis, requires special consideration. There can be no doubt that in young subjects there are physical and vital reasons which might lead one to hope more strongly for complete recovery by natural means than we could do in the case of adults. The softness of the lymph exuded is proportionably greater than in later life, and it is comparatively rare to find adhesions so strong as to bind the lung down with a firmness which renders subsequent expansion impossible; and, unquestionably, the vital activity of the processes of absorption is greatest in early life. But, on the other hand, there is a much greater tendency of effused serum to take on a purulent character in children than in adults; and the dangers of a long-retained purulent effusion are now seen to be much more formidable in presence of recent investigations as to the artificial generation of tuberculosis than they formerly appeared. This latter view of the case has been painfully impressed on my mind by a succession of cases, three in number, in all of which empyema has preceded and apparently caused tuberculosis in children who were, individually, remarkably well formed and robust. Two of the patients belonged to families in which there was a taint of phthisis, the other to a family perfectly free, for at least two generations, from any such disease. And seeing that there is in children a greater possibility of rapid re-expansion of the lung (both on account of the vigor of their respiratory efforts and the relative weakness of the fibrinous adhesions), we may the more reasonably hope that the removal of the whole or a portion of the liquid will be followed by a favorable turn in the progress of the disease. I regard as a typical instance of judicious and successful treatment the case recorded by Dr. Murchison (Lancet, loc. cit.), in which a boy of seven years was tapped on the twelfth day from the initial shivering and attack of pain, and twenty-four ounces of clear serum were withdrawn. Only two days were spent in therapeutic experiments after his admission into the hospital; and as these were without effect, and the effusion was large, displacing the heart and causing some (though not great) dyspnœa and weakness of pulse, the operation was done. Only part of the fluid was withdrawn, and, notwithstanding precautions, some air entered; but the case did perfectly well, and in one month more recovery was substantially complete. The only thing lacking in this case, according to my thinking, is, that the vacuum instrument of Bowditch or of Dieulafoy (Protheroe Smith) should have been employed; the discharge-pipe terminating in an india-rubber tube which dipped under water (*ex abundante cautelâ*).

The following statistics of the operation on children, as gathered from the hospital service of M. Barthez, are reported by Verliac.[1] Thoracentesis was performed on nineteen patients:—1. Simple acute

[1] Bowditch, last pamphlet, 1870.
[2] Lancet, 1870, vol. i. p. 221.

[1] Op. cit. p. 107.

Pleurisy, two cases; simple puncture, cure of both without reproduction of the liquid. 2. Serous tubercular Pleurisy, two cases; simple puncture, one cure without reproduction of the liquid, one death from convulsions six days after the operation. (It is not likely that the latter caused the fatal attack.) 3. Pleurisy symptomatic of heart-disease and vascular compression, one case; cure of Pleurisy after six punctures. 4. Pleurisy with purulent effusion, twelve cases; five cures, seven deaths.

Let me add to this the statistics collected by M. Guinier[1] of Montpellier, of 31 cases of children tapped by himself and others. The patients were of all ages up to 14; as many as 16 were in their seventh, eighth, or ninth year. In one case the operation cured a large sero-purulent effusion in a sucking child *twelve months old*. There were six times as many successes as failures, and the mortality was not in the ratio of the age. The operation itself *never* seemed to do any harm; in every case much immediate relief was obtained, and in the few fatal cases the operation never seemed to accelerate, but rather to retard, the advent of death.

I must cite, also, the valuable authority of Hillier for operation in Pleurisy in children; if done early, he says, it is not dangerous.[2]

Among other highly respected names, may be quoted Dr. Gairdner, of Glasgow,[3] Dr. J. W. Begbie,[4] and Dr. Fraser,[5] who have had the courage to follow out the more extended application of thoracentesis which Dr. Bowditch has inaugurated. Personally I have been so unfortunate that I have scarcely had any opportunities for employing the improved vacuum instruments since I became acquainted with them: although I have witnessed their results in the hands of others. But I was a believer in the need for more extended use of the operation long before I chanced to hear of Bowditch's discovery: and in two cases, as far back as 1862 and 1863, I tapped with the distinct intention of withdrawing a part, only, of a serous effusion (of four and six weeks' date respectively) and employed no other precaution than that of making the opening valvular. I did not conduct the liquid under water, but merely guarded the orifice of the canula with the thumb the moment the stream showed signs of interruption, took much pains in withdrawing the canula without unnecessary admission of air, and immediately well closed the external wound. No doubt some air entered, but no harm was done; both patients steadily recovered without reproduction of the fluid. One was a girl aged 17, otherwise healthy; the other a lad of 12, singularly bright and precocious, but with a dangerously suggestive family history.

I have thought it pardonable, and even necessary, to devote a somewhat large proportion of this article to the question of paracentesis, because I believe it is the duty of the writer on Pleurisy, in a "System of Medicine" published at the present day, to speak with no uncertain sound on this question; and in order to command the confidence of readers, it has been necessary to show the manifest tendency of a large number of the best practical men of the day. Fortified by the evidence above cited, and by the remembrance of a great deal more that could be produced, I venture to say, decidedly, that practitioners must throw aside the timid and vacillating rules of conduct which the majority of the text-books still prescribe. Tapping is not to be looked at as a dangerous last resort, appropriate only to a few cases. It must become an every-day remedy for cases where an effusion, purulent or not lingers for more than a very limited period: for the operation may be so conducted as to be perfectly harmless, while no one who knows the facts of recent pathology dare say that even a serous effusion will remain harmless, still less a purulent one. It remains to say a few words on the treatment of those least fortunate cases where from one cause or another, a purulent fluid forms and re-forms with great rapidity after each tapping, and perhaps becomes putrid and stinking. Where it is only a question of excessive purulent secretion, simple washing out of the pleura with warm water after tapping may possibly change the action of the membrane, but in most cases it will be necessary to keep the canula in, cork it up, and daily allow the exit of pus, and then wash out the cavity. But in my opinion, if it comes to this, the better plan by far is the drainage tube. A needle-eyed probe, being introduced through the original opening, is carried through to the opposite chest-walls, and is there made to protrude the muscle and skin of an intercostal space, the finger outside carefully feeling for it. The probe is cut down upon, forced out through the chest-wall, and threaded with a strong thread; this is then drawn back through the chest till it comes out at the original opening. The thread is fastened to an india-rubber drainage tube (pierced with openings in the manner devised by Chassaignac), and the latter is then drawn through the chest till it issues through both orifices. Nothing more then remains but to tie the ends of the tubes lightly together.

The use of iodine injections need not, I

[1] Bull. de l'Acad. de Médecine, tome xxx. p. 645.
[2] Brit. Med. Journal, Aug. 3, 1867.
[3] Clinical Medicine, 1862.
[4] Edin. Med. Journal, 1866.
[5] London Hospital Reports, June, 1865.

think, be recommended, save in cases of fetid purulent secretion; in this I agree with the opinions of Guinier, Fraser, and other high authorities. The solution should be one part tincture of iodine to four of tepid water. There is abundant evidence that even a long course of such injections does no harm, and it often appears to do good. Possibly the iodine injections may alternately be altogether superseded by the use of weak carbolic acid solutions. The combined use of disinfectant injections and the drainage tube has proved successful in a good many cases apparently of the worst augury; even, for example, in putrid empyema, secondary to puerperal fever.[1]

I shall sum up the treatment of Pleurisy in a few words. The pain must be met by opium or morphia (preferably injected), by hot poultices, and abstinence from movement (at a later stage the side may be supported by stout adhesive plaster for the same purpose). Acetate of ammonia or acetate of methylamine may be given—not in doses to produce *sweating*—but in moderate *stimulant* doses. The diet should be highly nourishing, but carefully adapted to the state of digestion. The bowels should be kept from actual loading, but no *purgation* should be attempted. The only diuretic worth trying in the stage of fixed effusion is iodide of potassium in small doses; and if this fails, it is best at once to have recourse to muriate of iron. But if at the end of fourteen to twenty days for a child, or three weeks to a month for an adult, from the initial symptoms, the fluid does not show real signs of diminution, paracentesis should be performed: and this rule is absolute, both for primary and secondary pleurisies, except where the case is hopeless on other grounds.

HYDROTHORAX.

By Francis E. Anstie, M.D., F.R.C.P.

Definition.—Passive non-inflammatory effusion of serum, due either to mechanical obstruction of circulation, or to blood-poisoning.

History.—The history of Hydrothorax really constitutes a part of the history of the various organic and constitutional diseases of which it is a mere episode. It was once the custom to speak of this malady as if it were a variety of pleurisy; in reality there is a broad distinction between the two affections. We shall discuss the points in which they approximate under the heading of Pathology; meantime, we may say that their history is essentially different. Hydrothorax, properly so-called, lacks several of the most important "notes" of inflammation. It arises, without febrile disturbance, in the later stages of disorders which either mechanically embarrass the circulation through the chest, or alter the specific gravity and the chemical relations of the blood-serum, or do both these things so as to promote a purely physical exosmosis. It is thus often due to diseases of the heart, particularly those of the right side, and it is not a very infrequent result of renal disease; but in perhaps the majority of cases a combination of renal and cardiac mischief is the cause. The course of Hydrothorax is eminently chronic, and the disease is often entirely intractable; in fact, Hydrothorax occurs in many cases only as a part of the closing scene of chronic organic disease.

Symptoms.—The invasion of Hydrothorax is usually stealthy and unnoticed, there is no febrile movement, and the only noticeable matter is the steady increase of dyspnœa. At last, and sometimes after a day or two only, the patient is in a state of gasping orthopnœa, with livid lips and the greatest appearance of distress; he is quite unable to lie down. Then, on examination, we find the physical signs of fluid in both pleuræ; it may be also in the pericardium. The effusion being bilateral, we find no displacement of the heart; but the diaphragm is nearly always pushed downwards, sometimes very greatly. When the effusion is large, the embarrassment of the heart is shown by the small and feeble pulse.

Pathology.—The nature of the effusion in Hydrothorax may vary within

[1] See Kussmaul (Deutsches Archiv für Klin. Med. iv. 1868) for some interesting recoveries after paracentesis and disinfection of stinking fluids.

rather wide limits; but it usually contains far less albuminous and fibrinous material, and far fewer cells (whether of epithelium, or white blood-corpuscles) than are found in pleuritic effusions. It may even be doubted whether the fluid of a passive effusion contains any blood-corpuscles at all; but from the readiness with which a clear Hydrothorax serum sometimes converts itself, if air be admitted to the chest, into pus, the presence of blood-corpuscles would appear probable.[1] When death has taken place without any puncture having been made, the pleura is found free from all lamellar fibrinous deposit, and the lung is simply compressed, not bound down by adhesions.

Diagnosis.—The distinction of this affection from real pleurisy is not always easy; but in most cases the history points strongly to the true nature of the effusion. The simultaneous occurrence of other dropsies, together with the absence of initial fever, enable us, usually, to say that Hydrothorax and not pleurisy is present; but on the one hand there may be no distinct dropsy anywhere but in the chest; and, on the other hand, true pleurisy may sometimes (*e. g.* after scarlatina) coincide with anasarca. Acute rheumatism supervening on old cardiac, or cardiac and renal disease, sometimes presents signs of a double pleural effusion, the nature of which it is difficult to decide; especially as the greatest pallor and depression in such cases may coincide equally with a pleurisy or a Hydrothorax. Notwithstanding these occasional difficulties, however, it is usually possible to give a tolerably decided diagnosis from a comparison of the history and the clinical features of the disease.

Prognosis.—How bad this is will be evident from the circumstances of great bodily depression in which Hydrothorax always arises, and from the necessarily more or less constant operation of the cause of dropsy, tending to a continual reproduction of the fluid even if we have been fortunate enough to witness its reduction or removal. Nevertheless there is great room for bold and intelligent treatment in a certain percentage of cases of Hydrothorax; and recoveries sometimes take place in a surprising manner. Many patients have had weeks, months, or even a few years, added to their lives in this way.

Treatment.—The tendency of the best modern practice in regard to Hydrothorax may be said to be nearly the reverse of that with regard to pleurisy. The operation of paracentesis is rarely applicable: it should be reserved almost exclusively for the prevention of threatened asphyxia when both pleuræ fill rapidly to a great height. On the other hand, the effect of diuretics, and still more of hydragogue purgatives, is often most striking. Of the former, infusion of digitalis in half-ounce doses, with thirty grains of bitartrate of potash twice or three times daily, has yielded me better results than any other. Of purgatives I only recommend one, viz. elaterium, which is incomparably superior, in my opinion, to all others. Great care ought to be taken to select a first-rate specimen of the drug, and then (diuretics having been fairly tried first) we need not scruple to use the elaterium boldly. One-fourth of a grain may be given (combined with a little hyoscyamus), and repeated in four hours; very usually two, or at most three doses will suffice to produce a very copious watery catharsis. It might be thought that this would kill such feeble creatures as Hydrothorax patients generally are, but if care be taken to give a little stimulant at the time that the bowels act, the effect is very far from exhaustive; the rapidity with which the fluid diminishes in the chest, and the consequent relief to all the patient's sensations, in favorable cases, must be seen to be believed. The moment that a decided impression has been produced, either by diuretics or by purgation, we must begin the use of muriate of iron, in twenty-drop doses of the tincture four or five times in the twenty-four hours: in this way we secure the best chance open to us of preventing the re-accumulation of the fluid.

Do what we will, however, it is of course inevitable that a majority of our patients will succumb: and those whom we for the moment cure of Hydrothorax are only temporarily relieved from danger.

[1] I tried to convince myself on this point, some time since, by microscopic examination of a typical hydrothorax fluid; but could not make up my mind upon the matter. Dr. Walshe speaks of "pus-cells" as being present.

PNEUMOTHORAX.

By Francis E. Anstie, M.D., F.R.C.P.

Definition.—Accumulation of atmospheric air, or other gas, in the pleura.

Varieties.—I. Non-perforative. Collection of gas (*a*) from decomposition in gangrene of the pleura; (*b*) from decomposition of an ordinary pleuritic fluid; (*c*) air replacing sero-purulent fluid, suddenly absorbed; (*d*) secretion of air by pleura.

II. Perforative. (*a*) Surgical, from penetrating wounds of thorax, or fractured ribs lacerating the lung, or violent contusion tearing the lung. (*b*) Perforation of lung and pulmonary pleura, from disease in the lung: (1) Tubercular, (2) gangrenous, (3) diffuse pulmonary apoplexy, (4) hydatids, (5) cancer, (6) emphysema, (7) abscess, (8) rupture in hooping-cough. (*c*) Perforation of lung from without: (1) by disease of bronchial glands, opening into pleura and bronchi, (2) by emphysema, (3) by parietal abscess. (*d*) Rupture of œsophagus opening into pleura.

This formidable-looking list of possible varieties of Pneumothorax simplifies itself greatly when looked at from the practical physician's point of view. We may usefully abstain from special consideration of the non-perforative kinds altogether, from their great rarity. Of the perforative kinds, we put aside the surgical varieties, as not coming within the scope of this work. Of the remaining varieties of perforative Pneumothorax, all, save one, are individually so rare as to deserve little more than the bare record of their occasional occurrence. More than 90 per cent. of perforative cases from disease of the lung itself are, according to Walshe, "tuberculous" (*i. e.* produced by some form of phthisical lung-disease), and, in fact, the subject of Pneumothorax, from the physician's standpoint, falls almost entirely under the domain of phthisis.

Clinical History.—The typical access of Pneumothorax is distinguished by the sudden occurrence of sharp pain in the side, and intense dyspnœa of the most distressing kind; occasionally, besides these, there is the distinct sensation, at the moment, of tearing inside the chest followed by a feeling as if fluid trickled or poured down the side. Collapse, with coldness of surface and cold sweat, is present in the majority of cases.

But the symptoms by no means always take this striking form; there are cases in which neither pain nor dyspnœa is present at first in at all a high degree; and there are many more in which, after the first moments of severe suffering, the patient enjoys comparative repose until the secondary symptoms, viz. those of pleural inflammation, set in; and this sometimes represents a considerable period of comparative pause. But the inflammatory process invariably, and for the most part very speedily and severely, sets in: and often there is again very rapid breathing before the recurrence of great conscious distress. In fact, *rapidity* of breathing is almost a physical necessity from the moment of the rupture, and it is great, not merely absolutely, but relatively to the pulse frequency, though the latter is also very much augmented. In the worst cases there is never one minute's cessation, from the moment of the catastrophe till death, of the most acute pain and the most distressing orthopnœa; this was exemplified in a little boy who was under my care at the Belgrave Hospital for children about four years ago.

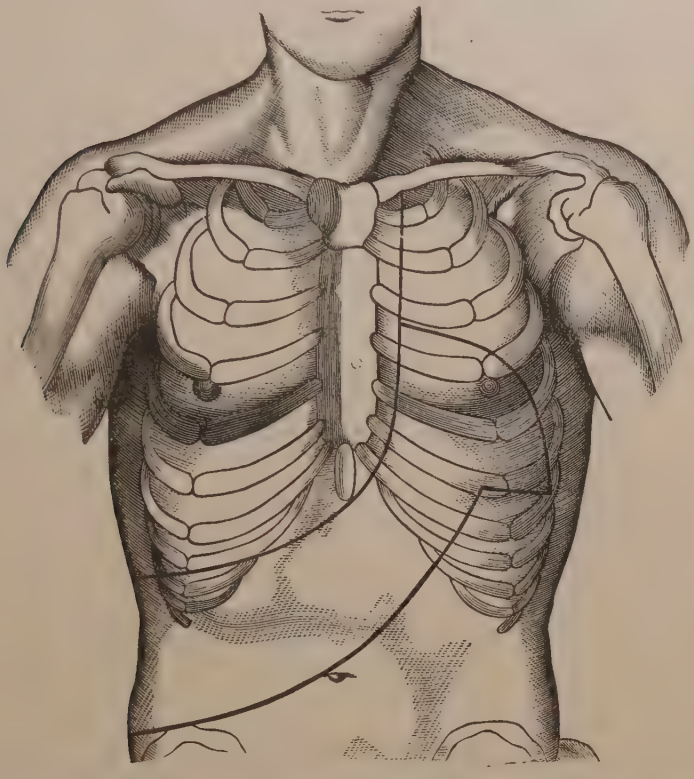
The physical signs of Pneumothorax give a very decided answer to our suspicions as to the nature of the case. The chest is very much, the affected side almost altogether (especially at the lower part), debarred from movement, the breathing is carried on mainly by the abdominal muscles; if the affected side moves at all evidently, the intercostal spaces are seen to be greatly depressed. Percussion gives out at first a merely much louder sound, with a graver pitch than in health; as the distension increases it becomes quite drum-like, and, if distension reaches the very highest grade, it becomes dull and muffled again—a well-known phenomenon of extreme air-tension. Occasionally percussion gives out an amphoric note. Palpation discovers weakening or abolition of vocal fremitus. Auscultation detects either great enfeeblement or complete suppression of the breath-cough, and voice-sounds, according to the amount of air in the pleura; the heart-sounds are either greatly weakened, or, occasionally, they are transmitted with a metallic ring.

As the distension proceeds the case becomes the more unmistakable; the medi-

astinum, heart, and diaphragm are notably displaced, and the tympanitic percussion-sound is heard to extend continuously even beyond the further sternal border.

When fluid becomes effused to a notable extent there are of course the signs described under pleurisy, of a liquid effusion in the lower part of the chest, together with the signs above mentioned, of air in the chest. There is also, when the fluid reaches any considerable amount, easily detectable fluctuation when the patient is shaken; and more occasionally and variably we can thus produce the true *splash*, with *metallic ring*. Moreover, with rare exceptions, the fluid demonstrably changes

[Fig. 46.



Displacement of mediastinum, heart, and liver from pneumothorax of the right side. (Weil.)]

its position with changes of the patient's posture: in this respect Pneumothorax assimilates to hydrothorax rather than to pleurisy. At the boundary line between fluid and air there may be amphoric percussion note, and a vibratile sensation communicated to the fingers. The displacement of viscera reaches, in bad cases of hydropneumothorax, the extreme degree which is ever observed.

DIAGNOSIS.—The only affection with which Pneumothorax can possibly be confounded is extreme emphysema; but there cannot be more than a momentary difficulty even here. Emphysema must be most unusually pronounced before the percussion note reaches anything like the tone of that heard in Pneumothorax; but then such emphysema is always symmetrical, while Pneumothorax affects only one side. But, indeed, the whole aspect of the two affections is quite different.

PROGNOSIS.—The prognosis of Pneumothorax is, on the whole, very bad, especially during the first day or two; if the patient survives for two or three days, his chances have materially improved. The great majority of fatal cases die within a week, and of these the largest part within the first two days. By common consent of authorities, however, there is a great uncertainty in the matter, cases which appear comparatively slight at first sometimes terminating fatally in a few days, while others, which at the outset seemed desperate, go on steadily improving, and regain comparative health; usually, however, they retain the signs of air and fluid in the pleura. In a few cases an absolute cure takes place; these

are mostly instances either of traumatic Pneumothorax, or else of empyema discharging itself through the bronchi. A few cases, however, even of phthisical Pneumothorax do recover; the opening becoming closed by lymph, and the air and fluid getting partly or wholly reabsorbed. A variety of Pneumothorax from which striking recoveries have taken place is that in which the rupture has been more the consequence of great muscular exertion than of any severely diseased condition of the lung. Such are some of the cases where the rupture has taken place during the paroxysms of hooping-cough; and a remarkable instance of an analogous kind has been reported by A. Vogel.[1] An unmarried woman, aged twenty-nine, who had borne ten children, had acted as a wet-nurse for a long time after each confinement, and had been perfectly well except that recently she had suffered from catarrh and an obstinate cough, in the midst of a sudden muscular exertion felt a sharp pain in the right side, and was seized with the most intense dyspnœa. When seen some hours later there were all the signs of the most complete Pneumothorax of the right side, with great displacement of the heart, lung, liver, &c., and severe collapse. Opium gave temporary ease, but on the next morning the anguish returned with waking, together with vomiting and choking sensations; yet no sign of pleuritic exudation could be detected. Morphia again gave relief to the pain and dyspnœa, and from this time all the symptoms speedily declined. In four days from the attack the patient had entirely recovered, and when seen a year later not a single trace of any mischief could be detected.

Of late years, indeed, a great deal of evidence has been collected to show that the mere influence of air upon a healthy pleura is extremely slight, and scarcely predisposes to inflammation at all: this comes out remarkably in the experiments and observations of Demarquay.[2] The same observation has also shown that a gradually decomposing collection of gas in the pleura is likewise harmless, except where sulphuretted hydrogen or sulphide of ammonium is developed. On the whole, it can scarcely be doubted that the larger part of the influences which determine the fate of patients with Pneumothorax depends upon unknown vital differences which there is little probability of our ever being able to estimate beforehand.

[1] Deutsch. Arch. f. klin. Med. ii. p. 244, 1866.
[2] Gaz. Médicale, 32, 1865.

TREATMENT.—The treatment of Pneumothorax is, necessarily, entirely palliative, and directed to the object of enabling the patient to survive the intensely depressing influence of the first shock, and that of the subsequent inflammation. The first step to be taken is the hypodermic injection of a full dose (half-grain) of morphia; and this medication may be administered twice, or in exceptional cases three times a day during the first two or three days, the hope being that it may possibly avert the threatened pleuritic inflammation. Dry cupping to the chest, frequently repeated, has been said to give very great relief in many cases. I cannot approve either of blood-letting, in any form, or of mercury; for the phthisical cases they are directly injurious, and in any case hypodermic morphia is likely to effect all the good which either of these remedies could be supposed capable of producing. Hot poultices to the chest undoubtedly give ease; they should be continually renewed. The great depression which is felt can, I think, generally be more suitably met by the internal administration of ʒss doses of ether, every three or four hours, than by alcoholic stimulants, though the latter are sometimes absolutely necessary.[1] If the patient survives the first few days, it will be proper to administer mineral acid and bark, cod-liver oil, or muriate of iron. And throughout the illness the greatest pains must constantly be taken to maintain the strength by easily digestible nutriment; and if the stomach be too irritable to bear this well, nutritive enemata must be unhesitatingly resorted to.

The question of paracentesis may be suggested by the extreme distress of respiration. In the phthisical cases, which form the large majority of those which the physician has to treat, this step could only be regarded as a very temporary palliative, and accordingly should only be employed as a last resort to procure a temporary respite when some very important object is to be secured by keeping the patient alive a little longer. It might be far more justifiable in cases where we had strong reason to suppose that the rupture was mainly accidental, and that the lung was free from serious internal disease. But I am not aware that any considerable statistics exist which might guide us to a conclusion on such a very doubtful point.

[1] I have seen cases in which alcoholic stimulants apparently much increased the acute pain. On the other hand, they occasionally do striking good.

DISEASES OF THE ORGANS OF CIRCULATION.

A. The Heart.

Weight and Size of the Heart.
Position and form of the Heart and great Vessels.
Malpositions of the Heart.
Lateral or Partial Aneurism of the Heart.
Adventitious Products in the Heart.
Pneumo-Pericardium.
Pericarditis.
Adherent Pericardium.
Endocarditis.
Carditis.
Hydropericardium.
Angina Pectoris and Allied States; including certain kinds of sudden Death.
Diseases of the Valves of the Heart.
Atrophy of the Heart.
Hypertrophy of the Heart.
Dilatation of the Heart.
Fatty Diseases of the Heart.
Fibroid Disease of the Heart.

WEIGHT AND SIZE OF THE HEART.

By Thomas B. Peacock, M.D., F.R.C.P.

1. *Of the Healthy Heart.* — From an early period pathologists have felt the necessity of some standard by which the size of the heart might be estimated, and its healthy and diseased conditions compared. Corvisart was unable to suggest any such, and Laennec compared the size of the healthy heart to the fist of the subject—a comparison too indefinite to afford any satisfactory estimate. Meckel and Kerkring, as quoted by Senac, were apparently the earliest writers who gave any estimate of the normal weight of the heart; and Lobstein and Bouillaud were the first to suggest the employment of the balance as a means of comparison between the healthy and diseased organs. The latter writer, in the first edition of his work, published in 1835, gave some observations of the weight both of healthy and diseased hearts, but they were too few in number to form the basis of satisfactory conclusions. Bizot conceived that the dimensions of the organ would furnish a better standard; and in 1837, in the Mémoires of the "Société Médicale d'Observation," published a large series of very careful measurements. Dr. Glendinning, in 1838, contributed numerous observations of the weight of the heart in a paper in the "Medico-Chirurgical Transactions;" and Dr. Ranking, in 1849, published in the *Medical Gazette* a series of measurements, both of healthy and diseased organs. In 1843 the late Professor Reid appended to his paper on the weights of the different organs of the human body, tables of the weight and dimensions of the heart; and in 1854 I published a considerable number of observations of the weight and size of the organ, under different circumstances of health and disease; together with various tables compiled from them. Both these sets of observations were published in the *Edinburgh Monthly Journal*. More recently, Dr. Boyd has recorded in the "Philosophical Transactions" a larger and more complete series of observations than had been published by any previous writer.

It is useless to refer to the estimates of the weight of the healthy heart given by any of the earlier writers, for we have no means of knowing the number of observations upon which they are based; the age and sex of the subjects; the condition of the organs weighed, or the precise weight employed. Of the more recent observers, M. Bouillaud estimated the weight of the

healthy heart in adults, not distinguishing the two sexes, as ranging from 8 oz. 10 drachms to 9 oz. 11 drachms imperial. Dr. Glendinning inferred that the mean weight of the healthy organ was in adult males 8¾ oz., and in females 7¾ oz.; and Dr. Reid deduced the average in males as 11 oz. 12 drachms, and in females as 9 oz. These estimates are sufficient to show how wide the differences may be according to the mode in which the calculations are made. It is evident that the weight of the heart must vary considerably according to the cause producing death; the organ being heavier when the patient dies suddenly or after only a short attack of illness, and lighter when death has taken place from lingering diseases, provided the diseases are not such as to interfere with the functions of the organ, and so give rise to over-nutrition. Thus, while Dr. Reid, as just stated, estimated the average weight of the male heart at about 11 oz., he found that in twelve men who were killed the weight attained an average of 12 oz.; and, on the other hand, I have examined the hearts of persons who have died from cirrhosis of the liver and cancer of the pylorus, &c., which were only 5 or 6 oz. in weight. To form, therefore, an accurate estimate, not only must the age and sex be taken into consideration, but the weight of the organ must be given in acute and chronic diseases separately; and the cases in which the nutrition of the heart may have been materially modified by the disease causing death must be excluded from the calculation. The size of the heart will also be similarly influenced, and especially the dimensions must vary with the degree of distension of the cavities at the time of death. To form a thoroughly satisfactory estimate, the weight and dimensions of the heart must therefore both be given, and the previous circumstances must be taken into consideration.

In the following tables I have endeavored to carry out these views. In the first table the weight of the heart in the adult is given separately, for males and females, and for acute and chronic diseases. In the second, the dimensions of the organ, also in the adult and in males and females, are stated, in Paris lines, millimetres, and parts of English inches. The third table gives the average weight of the heart in males and females at different ages.

From the first table it will be seen that

TABLE I.

Average Weight of Healthy Heart in Males and Females and in Acute and Chronic Diseases from Twenty to Fifty-five Years of Age.

MALES—	
Mean weight	9 oz. 8 drs.
Ordinary range in acute cases	9 oz. to 11 oz.
" " in chronic cases	8 oz. to 10 oz.
FEMALES—	
Mean weight	8 oz. 13 drs.
Ordinary range in acute cases	8 oz. to 10 oz.
" " in chronic cases	7 oz. to 9 oz.

in adult males who have died from acute diseases, or from the effects of accident, the ordinary weight of the heart is from 9 to 11 oz.; and in those who have died from chronic diseases, 8 to 10 oz. In females, the ordinary weight of the heart in acute cases may be estimated at from 8 to 10 oz., and in chronic diseases from 7 to 9 oz. Occasionally, however, in persons of small and delicate frame, who have died from emaciating diseases, such as cancer of the stomach, bowels, or mesentery, or chronic affections of the liver, the heart will be found to weigh only 5 or 6 oz.; and in large and powerful men who have been killed or have died after short illnesses, the organ may weigh 12 oz. or even more, without exceeding the limit of health. Some writers have given calculations of the relation of the weight of the heart to that of the whole body, but the bulk of the body, and also, as before stated, the size of the heart, vary so greatly from the duration of illness and the mode in which death occurs, that such calculations do not possess much value. The height of the subject and the weight and size of the heart probably bear a more just relation.

From the second table it will be seen that the girth of the right ventricle, measured externally, exceeds that of the left, in males by about one-sixth, and in females by one-fifth. The length of the cavity of the right ventricle is greater than that of the left, in males by one-seventh, and in females by one-sixth. In both

TABLE II.

Dimensions[1] *of the Healthy Heart (in French Lines, Millimetres, and English inches) in Males and Females.*

	Males.			Females.		
	Lines.	Millimetres.	Inches.	Lines.	Millimetres.	Inches.
Circumference of heart	103·7	233·32	9·209	104	234	9·236
Girth of right ventricle	55·4	123·85	4·919	58·4	131·4	5·184
" left "	48·3	108·67	4·289	45·6	102·6	4·049
Length of cavity of right ventricle	43·3	96·42	3·821	44·3	99·67	3·925
" " left "	37·6	84·6	3·333	37·1	83·47	3·197
Thickness of walls of right ventricle, base	1·85	4·16	·164	1·85	4·16	·164
" " " " " midpoint	1·98	4·35	·176	2·0	4·5	·177
" " " " " apex	1·42	3·19	·125	1·3	2·92	·118
" " " left " base	5·15	11·58	·425	4·9	11·02	·432
" " " " " midpoint	6	13·5	·532	5·6	12·6	·497
" " " " " apex	2·4	5·4	·214	2·5	5·62	·222
Thickness of septum.	5·73	12·89	·51	4·7	10·57	·421
Circumference of right auriculo-ventricular aperture	53·4	120·15	4·74	51·4	115·65	4·562
Circumference of left auriculo-ventricular aperture	45·2	101·7	4	45	101·25	3·996
Circumference of pulmonic aperture	40	90	3·552	39·3	88·42	3·493
" of aortic "	35·6	80·1	3·146	34	76·5	3·019

sexes the thickness of the walls of the right ventricle is about one-third that of those of the left. The thickness of the septum is intermediate between that of the external walls of the right and left ventricles. In males the pulmonic orifice is about one-eighth more in circumference than the aortic. The left auriculo-ventricular aperture is one-fourth more than that of the aorta, and the right auriculo-ventricular aperture one-half larger. In females the differences between the aortic and other orifices are somewhat greater.

It has been generally supposed that the heart increases in weight with the progress of life; and this opinion is supported by the facts recorded relating to males, in the third table. It may, however, be doubted whether the result thus indicated is applicable to the heart in its strictly healthy state. It is well known that in advanced age there is a decided diminution in the weight of the brain, and there seems no reason why a similar decrease of weight should not occur in the heart, provided that organ be not the seat of disease interfering with its normal nutrition. As we well know, but few elderly persons, especially men, are entirely free from any form of disease which, by occasioning obstruction, might lead to over action, and so to some degree of hypertrophy. And even if the heart be not itself diseased, there are few old persons who do not display some affection of the lungs, kidneys, or other parts of the system, which might more or less interfere with the functions of the heart, and so lead to its enlargement. That this is the more correct view is supported by the diminution in the weight of the organ in elderly females, as also shown in the table.

TABLE III.

Weight[2] *of the Healthy Heart at Different Ages in Males and Females.*

	Males.	Females.
Ages 10 to 14 inclusive—Mean weight	6 oz. 1·5 drs.	5 oz. 0 drs.
" 15 " 20 " " "	8 " 2·66 "	8 " 1·66 "
From 20 " 30 " " "	8 " 0·14 "	8 " 10·42 "
" 30 " 40 " " "	9 " 7·95 "	8 " 13·94 "
" 40 " 50 " " "	9 " 11·11 "	9 " 3 "
" 50 " 60 " " "	9 " 12 "	9 " 7·33 "
" 60 " 70 " " "	10 " 13·33 "	7 " 0 "
Mean weight between 20 and 55 years of age—in 76 males	9 oz.	8·74 drs.
" " " " " " " in 49 females	8 "	13·16 "
Difference		11·58 "

[1] The dimensions of the orifices are taken by balls, the first of which is 12 lines in circumference, which increase in circumference three Paris lines, and are numbered from 1 to 20.

[2] The weight employed is Avoirdupois or Imperial.

2. The alterations in the weight of the

heart in disease are illustrated by Tables IV. and V.

It was supposed by Dr. Glendinning, that the heart in cases of *phthisis*, contrary to what would *à priori* have been expected, acquires an increase of weight, while the rest of the body becomes emaciated. This idea appears to have arisen from a misapprehension of the facts which he collected. The effect of the pulmonary affection upon the nutrition of the heart appears to vary with the form of the disease. In cases of uncomplicated constitutional or tubercular phthisis, the progress of which is generally rapid and which is usually attended with great emaciation, the heart is found to weigh considerably below the healthy average, and the organ, on examination, often displays the appearance of atrophy. In cases of chronic phthisis, whether tubercular or inflammatory, on the other hand, and especially when one or both lungs are considerably contracted, or when there have been marked bronchitic symptoms, so that the blood has for a long time been transmitted with difficulty through the lungs, the heart is generally found to be enlarged, or, at least not to have undergone any marked diminution in size; its weight equalling or exceeding the healthy standard. So also when, in cases of phthisis, there is any great impediment to the transmission of the blood from the heart from valvular or aortic disease, notwithstanding the general tendency to emaciation, the organ may exceed even very greatly the natural size.

TABLE IV.

Range of Weight of Heart, in Different Forms of Disease and when Diseased.

	Mean.		Extremes.			
	oz.	drs.	oz.	drs.	oz.	drs.
Phthisis. Males	9	3·4	6	4·5 to	11	0
" Females	8	6·06	5	9 "	11	0
Chronic Bronchitis. Males	14	8	11	8 "	21	0
" " Females	12	2·0	9	0 "	12	8
Morbus Renum.—Males	9	12	7	4 "	14	8
" " Females	10	5·4	7	4 "	15	8
Simple Hypertrophy.—Males	...	...	12	0 "	40	12
Aortic Disease.—Males	...	...	10	0 "	24	0
" " Females	...	...	8	8 "	20	0
Aortic valvular obstruction.—Males	...	...	14	0 "	21	0
" " " Females	...	...	13	0 "	18	8
Aortic valvular regurgitation.—Males	...	...	14	0 "	34	0
" " " Females	...	...	16	0 "	23	0
Mitral valvular obstruction or regurgitation, or both—Males	...	...	14	0 "	17	0
" " " " " " Females	...	...	13	0 "	18	8
Combined aortic and mitral valvular disease.—Males	...	...	14	8 "	21	0
" " " " " Females	...	...	7	8 "	23	0

Chronic Bronchitis.—When there is long-continued obstruction to the pulmonary circulation from chronic bronchitis, with or without deformity of the spine, the right side of the heart becomes hypertrophied; but, even a great increase in the thickness of the walls of the right ventricle does not very much augment the weight of the heart, and it is only after the left side has become implicated that

TABLE V.

Extreme Dimensions of the Heart with the Different Forms of Disease in which they occur.

	Lines.	Millimetres.	Inches.	
Circumference.—Males	182	409·5	16·16	In simple hypertrophy.
" Females	127	285·75	11·27	Mitral disease.
Thickness of walls of right ventricle.—Males	5·75	12·93	·51	Mitral disease.
" " " " Females	7	15·73	·62	Congenital obstruction at pulmonic orifice.
" " left " Males	14	31·5	1·24	Aortic valvular disease.
" " " " Females	11	24·75	·97	Combined aortic and mitral disease.
Circumf. right auriculo-ventricular apert.—Males	63	141·76	5·59	Simple hypertrophy.
" " " " " Females	60	135	5·32	Aortic valvular obstruction.
" left " " " Males	60	135	5·32	Simple hypertrophy.
" " " " " Females	45	101·25	3·99	Aortic valvular obstruction.
" pulmonic aperture.—Males	54	121·5	4·79	Simple hypertrophy.
" " " Females	39	87·78	3·46	Mitral valvular disease, chronic bronchitis, and deformed spine.
" aortic " Males	45	101·25	3·99	Aortic valvular disease.
" " " Females	35	78·85	3·1	Aortic valvular disease and combined aortic and mitral disease.

the weight is found much to exceed the healthy standard. In some such cases, however, the organ may attain a weight considerably greater than the natural, amounting, in hearts I have weighed, to 15 or 16 oz.

Morbus Renum. — Dr. Bright noticed that the heart in cases of chronic disease of the kidneys was frequently found increased in size without there being any valvular disease to explain the enlargement; and the occurrence of simple hypertrophy in such cases has been noticed by other pathologists. Of eighteen cases in which the organ was weighed by myself, in seven the weight was below the average of chronic diseases, while in eleven it exceeded it, attaining in some cases in males the weight of upwards of 14 or 15 oz. In these cases the hypertrophy is doubtless due to the over-action of the heart from its efforts to overcome the obstruction to the transmission of the blood through the capillaries.

3.—Weight and Dimensions of the Diseased Heart.

Simple Hypertrophy. — The most remarkable case of increase in the weight and size of the heart which I have myself met with was in a case of hypertrophy, without any material valvular disease or any obvious source of obstruction in the aorta, to explain the condition. In this instance, which, however, is a very extreme one, the organ weighed 40 oz. 12 dr.; but in various other cases I have found the weight considerably to exceed the average or extreme limit of health. In a heart which I examined with Mr. Hutchinson, the weight attained was 26 oz.; and Dr. Bristowe exhibited one at the Pathological Society which weighed 27 oz. The ages of these patients were sixty-five, thirty-five, and forty-one respectively. It is difficult to explain the great enlargement which exists in some cases of this description. It may depend on some disease of the smaller arteries which may have escaped observation, or on obstruction in the capillaries; but in other instances, it is probably due to habitual over-action from athletic pursuits, and possibly in some cases to palpitation, at first originating in emotional causes. Enlargement unconnected with valvular disease is, however, rarely seen except in men, and in no instance have I found the heart much hypertrophied in females without there being some obvious source of obstruction to which the change was referable.

Aortic Obstruction and Aortic Valvular Disease.—The occurrence of obstruction in the aorta, and especially in the upper portion of that vessel, is generally attended by considerable increase of weight in the heart. In various cases of this description I have found the heart to range from near the natural standard to 24 oz. in males, 17 oz. in females. In the Transactions of the Medico-Chirurgical Society, a case is recorded by Dr. Risdon Bennett, in which the heart weighed 22½ oz. in a man fifty-three years of age, who died from rupture of the aorta, giving rise to dissecting aneurism and hemiplegia. The increase of size in the heart was apparently due to atheromatous disease of the aortic coats.

In aortic valvular disease still greater increase of weight is often met with. In cases of obstructive disease, I have weighed hearts ranging from 14 oz. to 21 oz. in males, and from 13 oz. to 18 oz. 8 drs. in females. In cases of aortic valvular incompetency, I have found the heart to weigh from 14 to 34 oz. in males, and from 16 oz. to 23 oz. in females. Dr. Vanderbyl, in a paper in the "Pathological Transactions," relates instances in which the heart weighed 36 oz. in a case of aortic valvular incompetency, in a man of twenty-eight; 30 oz. in a case of aortic disease with aneurism in the aorta, in a man of thirty-three; and 30 oz. in a case of aortic and mitral disease, in a man of sixty-two.

In the cases of incompetency of the aortic valves, it is often impossible to say how much of the great enlargement of the heart is due to the obstruction, and how much to the incompetency of the valves; for the latter condition is generally only the final stage of the former. In cases of rupture of the aortic valves during violent effort, we have, however, the opportunity of seeing the remarkable changes which may occur in the heart even during short periods of time, when, in organs previously healthy, the valves are rendered incompetent and the left ventricle rapidly becomes hypertrophied and dilated. Thus, in a case of this description which occurred to myself, the patient, a man of thirty-three years, survived the accident only twenty-seven months, yet the heart was found to weigh 17¾ oz. In a case related by Dr. Quain, the patient lived two years, and the heart weighed 22½ oz.; and in another case which I had the opportunity of examining after death, though the patient, a man thirty-six years of age, only survived three and a half months, the weight was 23 oz. If, in this instance, the heart was sound at the time of the occurrence of the injury, the process of enlargement must have been most rapid; but it may be doubted whether the organ was not more or less hypertrophied before the accident, though the patient stated that he was previously quite well.

In some cases of disease, also, the enlargement must take place very rapidly.

In a boy of eighteen, who died of aortic valvular disease originating in malformation, the duration of active illness was only three and a half years, yet the heart weighed 28 oz. In a case of aortic valvular incompetency, with probably regurgitation through the left auriculo-ventricular aperture from maladjustment of the valves, described by Dr. Bristowe in the "Pathological Transactions," the heart weighed 46½ oz. avoirdupois, though the subject of the disease was only twenty-two years of age. In an instance of very great obstruction at the aortic valves, doubtless from malformation, which I have recently exhibited at the Pathological Society, the heart weighed 24 oz., the patient being only twenty-three years of age. These examples show that the heart may attain a very great increase of size even in comparatively young subjects; but, usually, those in whom the heart is very large are advanced in age. Probably, also, in most cases, the disease must be of long duration for the organ to become very greatly hypertrophied, and this great prolongation of life is only compatible with comparatively slight disease, or with disease which has been very slowly progressive, though at the time of death it may have become extreme.

In *mitral valvular disease*, whether consisting in obstruction and regurgitation, from contraction of the orifice and rigidity of the valves, or in free regurgitation from expansion of the aperture or maladjustment of the valves, the heart does not ordinarily attain by any means so great an increase of weight as in cases of aortic disease. In the former class of cases, the hypertrophy is chiefly limited to the right ventricle, and only affects the left ventricle secondarily, though in the latter the left ventricle also partakes of the change from the first. In cases of mitral disease, the weights have ranged from 14 oz. 8 drachms to 17 oz. 8 drachms in males, and from 12 oz. to 18 oz. in females.

As might be expected, in *combined aortic and mitral valvular disease*, the weight of the heart is intermediate between that which obtains in the two separate forms of disease, the organ being lighter than in aortic, heavier than in mitral disease. In males, in cases of this kind the heart was found to weigh 14 oz. 8 dr. to 21 oz. 8 dr., and in females from 17 oz. to 23 oz.

In cases of obstruction at the right side, consisting in *congenital contraction of the pulmonic orifice*, the effect produced on the nutrition of the heart is very similar to that which results from chronic bronchitis. In the first instance the right ventricle is chiefly hypertrophied, but subsequently the left also becomes involved; and similar changes ensue in the cases in which the aorta communicates with both ventricles, provided the life of the patient be sufficiently prolonged. In a male of twenty, in whom the pulmonary orifice was contracted from adhesion of the valves, the heart weighed 12 oz.; and in a female of nineteen, in whom there was similar disease of the pulmonic valves, and the aorta arose from both ventricles and the ductus arteriosus was open, the organ weighed 17½ oz.

The effect produced by *adhesions of the pericardium* on the functions and nutrition of the heart has been the subject of much discussion. On the one hand, adhesions have been supposed to interfere with the free movement of the heart, and so to give rise to hypertrophy; on the other, it has been thought that by the compression exercised upon the organ they might cause atrophy. The question is one which it is very difficult to decide, for there are few cases in which the pericardium is entirely adherent, in which the valves are not also more or less involved, and in which therefore the effects produced by the one condition may not be modified by the other. I find, however, that in three men in whom the pericardium was entirely adherent, while the valves were free from disease, the hearts weighed 16 oz., 17 oz. 4 dr., and 18 oz.; but I have examined other organs under similar circumstances in which the weight did not exceed the healthy standard. The general rule is, however, that in cases of adhesion the heart becomes hypertrophied.

General Remarks on the Weight of the Heart.—M. Bouillaud has collected some cases in which the heart otherwise healthy weighed considerably less than natural; and others in which in various states of disease it exceeded that point. The former are all cases in which the organ was reduced in weight with the progressive emaciation of cancer, consumption, &c. The lightest heart, that of a female of forty-five, weighed 4 oz. 5 dr. in a case of cancer; the heaviest organ weighed 24 oz. 4 dr. in a case of obstructive and probably also regurgitant disease of the aortic valves, in a female of fifty-three. From these observations, and his estimate of the average weight of the healthy organ as ranging from 8 oz. 10 dr. to 9 oz. 11 dr., he infers that the heart may attain when diseased three times the weight of the average healthy organ, and five times that of the most atrophied organ. These estimates are, however, considerably less than the variations of weight which actually obtain. I have found the heart to weigh only 5 oz. in a man fifty-three years of age who died of cirrhosis of the liver, and 6 oz. in a man of thirty-nine who had cancer of the pylorus. The average weights I have estimated in males at 9 oz. 8 dr., and the heaviest heart weighed was 40 oz. 12 dr. It follows therefore that in men the heart may attain a weight

which is four times that of the healthy and eight times that of the atrophied organ. In females, the variations in the weight of the heart are sufficiently remarkable, though considerably less than in men. The average weight has been shown to be 8 oz. 13 dr.: the lightest hearts weighed 5 oz. 8 dr. in cases of phthisis in twenty-five and thirty years of age; the heaviest organ was 23 oz. The most enlarged heart was therefore three times the weight of the average, and four times that of the atrophied organ. It has also been mentioned that Dr. Bristowe has placed on record a case in which the heart of a man twenty-two years of age weighed 46½ oz.; and Dr. Church has recently exhibited at the Pathological Society the heart of a female forty-seven years of age, who died of cancer of the pylorus, which weighed only 3 oz. 1 dr.

The heart described by Dr. Bristowe is, as far as I know, the heaviest on record. Dr. Hope says that he examined at St. George's a heart which weighed 2½ lbs., which, if the weight employed were avoirdupois, would nearly equal the size of the largest heart which I have myself weighed—40 oz. 12 dr. M. Lobstein refers to a heart which weighed 34 oz., and Dr. Vanderbyl to one of 36 oz,

The dimensions of the heart in different forms of disease bear a general relation to the weights of the organ in similar conditions. Of the observations which I have myself made, the greatest weight was attained in cases of simple hypertrophy, obstruction in the course of the aorta, and obstructive, or obstructive and regurgitant disease of the aortic valves. It is equally in these forms of disease that the dimensions of the organ are most considerably enlarged, the cavities and orifices, especially those of the left side, being the most expanded, and the walls the most remarkably increased in thickness. There are, however, differences in the condition of the organ in these several forms of disease. In cases of obstruction, on whatever cause dependent, the heart is not generally so large as in cases of incompetency, and the form of the organ is also somewhat different. In the former class of cases the heart is peculiarly long and pointed at the apex, and the walls attain the greatest width near the base. In the latter the ventricle is usually of larger size and rounded at the apex, and the thickening is more equally diffused over the walls. In both forms of disease, the enlargement, though most marked on the left side of the heart, affects the right also very considerably.

In cases of mitral valvular disease, the size of the organ is considerably less than in the former class of cases, and the shape is very different, but the precise condition of the organ varies with the form of disease. In cases of great contraction of the left auriculo-ventricular aperture, the stress falls chiefly on the left auricle and the right cavities, and they are all found expanded and the walls increased in thickness, and much firmer than natural; the orifices also being dilated; while the left ventricle is not much if at all enlarged, and its walls are not materially hypertrophied. It has, indeed, been supposed that the left ventricle becomes atrophied. In cases, on the other hand in which the defect consists chiefly or to a marked degree in incompetency of the valves, on whatever cause dependent, the left ventricle is found to be considerably dilated and hypertrophied, and the changes on the right side are less marked. In both these forms of disease, however, the alteration in the shape of the heart is very marked, the organ being wide and blunted at the apex—in the one case chiefly in consequence of the expansion of the right side, in the other of the dilatation of the left ventricle, and especially the widening of its apex. In cases of combined aortic and mitral valvular disease, the enlargement is intermediate, both in shape and extent, between the other two forms. In cases of chronic bronchitis, chronic phthisis, deformity of the chest, and pulmonic valvular obstruction, the hypertrophy and dilatation are at first limited to the right side, but subsequently, if life be much prolonged, involve the left also. Table V. shows some of the extreme dimensions which I have recorded in different forms of disease.

It will be observed that not only do the size of the cavities and the width of the walls vary greatly in different forms of disease, but the capacity of the orifices also undergoes remarkable change; and this not only in cases of old disease, but even during comparatively short periods of illness. Thus it will generally be found in cases of acute bronchitis and very acute phthisis, that the pulmonic aperture, which ordinarily exceeds the aortic somewhat in capacity, is disproportionately larger than the aortic, and the right auriculo-ventricular aperture equally out of proportion with the left. I have also reason to believe that the apertures may not only expand in a short time, but may have their dimensions reduced without being otherwise diseased, and thus it is possible that in some forms of valvular defect the size of the orifice may be reduced and the incompetency diminished.

POSITION AND FORM OF THE HEART AND GREAT VESSELS.

By Francis Sibson, M.D., F.R.S.

FRONT VIEW; AFTER DEATH.

The following observations on the position and anatomical relations of the healthy heart and great vessels after death are founded on the examination of a number of diagrams showing the position of the internal organs after death. This examination was restricted to those instances in which the heart was healthy and was not enlarged. The diagrams were made by drawing the outlines of the organs on a piece of lace or net, stretched upon a frame and placed over the body.

The heart and great vessels present great variety in form and position both after death and during life.

During the illness or injury that ends in death, at the time of death and after death, the heart and great vessels undergo a series of changes in position and form. According to the nature and direction of these changes, the heart after death may, in different instances, be (I.) higher or lower in position; or (II.) it may deviate more to the right or more to the left.

(I.) The Higher or Lower Position of the Heart and Great Vessels.

Three main conditions may influence the higher or lower position of the heart after death: (1) The contraction or expansion of the lungs; (2) The distension or flaccidity of the abdomen; and (3) The state of the heart itself.

(1) When death is associated with bronchitis, or pneumonia, or affections of a like nature, in which the lungs are large, and are expanded after death, the chest is broad and deep, the diaphragm is low, and the heart, which is charged with blood, especially in its right cavities, is large, and occupies a low position. As a rule, however, the lungs, when they are not thus affected, lessen in size and contract during the final expirations. The cage of the chest then becomes more flat and narrow; it lengthens downwards, and the sternum and costal cartilages and ribs in front are all lowered in position. The diaphragm at the same time is elevated. While the front of the chest is thus lowered, the heart, resting on the diaphragm, is raised, and the whole organ, and the great vessels occupy a higher position. We thus have a double and contrary movement in the descent of the bony framework of the front of the chest, and the ascent of the heart immediately behind that framework. As the heart within, and the sternum and cartilages without are both thus elevated by the distension of the abdomen, the actual elevation of the heart and great vessels is much greater than their apparent elevation, estimated as that usually is by the relation of those parts to the walls of the chest immediately in front of them.

(2) When the abdomen is distended, whether by fluid or air in the cavity itself, by an accumulation of gas in the stomach and intestines, or by other causes, the whole diaphragm is forcibly elevated, and the heart, resting as it does on the central tendon of the diaphragm, is lifted upwards. The sternum and costal cartilages in front of the heart are, at the same time, also raised in position, and the lower ribs on either side are pressed outwards. Although the actual elevation of the heart is, in these cases, often very great, its apparent elevation, which is measured by the relation of the heart to the walls of the chest in front of it, may be slight, owing to the simultaneous elevation of the heart and the sternum and cartilages in front of the heart caused by the distension of the abdomen.

When the abdomen is flaccid, owing to the stomach and intestines being empty, the reverse effects take place. The diaphragm descends, the heart drops downwards, the sternum and costal cartilages are lowered in position, and the inferior ribs fall inwards.

(3) During the final illness or injury that precedes death the heart may lessen or enlarge. Fatal hemorrhage or wasting disease reduces the size of the heart and great vessels. On the other hand, the heart is swollen, especially on the right side, under the influence of suffocation or bronchitis; while its left ventricle may be thickened and enlarged in cases of Bright's disease with contracted kidney. Thus the right or the left side of the heart may be enlarged when the obstacle to the flow

of blood is respectively in the lungs or the body.

At the time of death, the left ventricle usually closes firmly upon itself; while then or soon afterwards the right cavities of the heart become permanently swollen with blood.

After death the heart shrinks upwards to a greater or less extent. This is owing partly to the diminution of the organ, but mainly, I believe, to the contraction of the arch of the aorta, for the shortening of that vessel draws the heart upwards, just as its lengthening pushes the organ downwards.

The exact extent to which the heart is thus raised, is measured by the space that is left between the lower boundary of the heart, and the lower boundary of the *front* of the pericardium. During life these two adjoining parts fit each other exactly; but after death they are separated by a space that varies according to the degree to which the heart shrinks upwards. Thus in the body of a youth who died from hemorrhage after fever, and in that of a man who expelled two or three pints of blood from a cavity in the left lung, an inch of space intervened between the lower edge of the heart and that of the lower boundary of the *front* of the pericardium. In another instance that space was only the tenth of an inch. As a rule the space varied from a quarter to seven-tenths of an inch (in 38 of 44 instances) and its average measurement was nearly half an inch (0·46 inch). (Note 1.)

The heart and the great vessels mainly occupy the centre of the chest, being protected in front by the sternum and the adjoining costal cartilages. It is, however, my present object, not so much to describe the relative bearings of those parts after death, as to indicate the *variation* in the anatomical situation of the more important boundaries or landmarks of the healthy heart and great vessels observed by myself in different instances after death.

The lower Boundary of the Heart.—In one instance, a woman who died from starvation, the lower boundary of the heart was situated behind the ensiform cartilage an inch and a half below the lower end of the sternum (that term being restricted here and elsewhere to the manubrium and blade or osseous part of the sternum), while in another it was almost as much (1·4 inch) above that end of the bone. Between these two extreme points this boundary occupied every variety of position. In one-fifth of the instances observed (15 in 71) the lower boundary of the right ventricle was just behind the lower end of the sternum, while in two-fifths of them it was above (30 in 71), and in two-fifths of them it was below (26 in 71) that end of the bone. (Note 2.)

As we have already seen, the lower edge of the heart usually shrinks upwards after death for nearly half an inch, the extent varying from one inch to one-tenth of an inch. The position of the lower border of the *front* of the pericardium, which points out the position of the lower border of the heart at the time of death was indicated in four-fifths of the cases (55 in 71) in which the inferior boundary of the heart was observed after death. In one-fifth of these instances (11 in 55) the lower limit of the pericardium was on a level with the lower end of the sternum; while in two-thirds of them (37 in 55) it was below that point, being situated behind the ensiform cartilage; and in only one-eighth of them (7 in 55) was it above that point. We thus see that at the time of death, in the great majority of instances (40 in 59) the inferior border of the heart was below the lower end of the sternum, being situated behind the ensiform cartilage. (Note 3.)

The seat of the lower boundary of the apex in relation to the left fifth space is a more important landmark for the clinical observer than that of the lower boundary of the heart in relation to the lower end of the sternum.

The lower edge of the heart at the apex was on a level with the lower edge of the left fifth cartilage in one-seventh of the instances observed (9 in 69), it was below that edge in two-fifths of them (26 in 69), and it was above that edge in almost one-half of them (34 in 69). In five instances the lower boundary of the apex was situated one inch above the lower edge of the fifth cartilage, and in four it was fully one inch below that edge. (Note 4.)

The lower border of the pericardium just below the apex, which corresponds with the seat of the lower border of the apex at the time of death, was on a level with the lower edge of the fifth cartilage in one-sixth of the instances observed (9 in 55), was situated below that edge in three-fourths of them (41 in 55), and was above that edge in only one-eleventh of them (5 in 55). (Note 5.)

We thus see that there was a general, but not a constant correspondence between the relation of the inferior boundary of the right ventricle to the lower end of the sternum, and that of the inferior boundary of the apex to the lower edge of the fifth cartilage, both at the time of death, and after death when the examination of the body was made. This correspondence would have been more constant but for variation in (1) the comparative height of the fifth cartilage and the lower end of the sternum, (2) the degree of inclination from above downwards and from right to

left of the lower boundary of the heart, and (3) the extent to which the right ventricle is situated to the right and to the left of the middle line of the sternum.

(1) In the great majority of instances (60 in 71) the inferior edge of the left fifth cartilage was lower in position than the inferior extremity of the sternum, to an extent varying from a quarter of an inch to an inch and a quarter; in five cases those two parts were on the same level; and in six the lower edge of the fifth cartilage was higher by from a quarter to three-quarters of an inch than the lower end of the sternum.

The height of the fifth cartilage in relation to that of the lower end of the sternum is influenced by (1) respiration, (2) abdominal distension, and (3) natural and acquired formation. (1) Inspiration raises and expiration lowers both the sternum and the fifth cartilage attached to the sternum, but as the cartilage has an additional movement of its own, during the double act of breathing, it is more lowered during expiration and more raised during inspiration than the sternum. The artificial distension of the lungs after death elevates the fifth cartilage from the sixth to the third of an inch more than the corresponding part of the sternum. If the chest is broad the left fifth cartilage is higher, and if the chest or the left side of it is narrow, the left fifth cartilage is lower in relation to the lower end of the sternum than it would have been otherwise. (2) Abdominal distension raises, and abdominal collapse lowers both the sternum and the fifth cartilage, but the raising or lowering of the fifth cartilage under these circumstances is greater than the respective raising or lowering of the sternum. (3) In some persons the fifth cartilage is naturally higher or lower than in others. Thus the fifth cartilage is sometimes integrally attached to the sixth cartilage and it is restrained by and shares its movements. When this is so the fifth cartilage tends to be lower in relation to the lower end of the sternum than when that cartilage is free. In robust persons with ample chests the fifth cartilage is higher relatively to the sternum than in thin persons with contracted chests, in whom the cartilage tends to be low in position in relation to the end of the sternum.

(2) In nearly all instances (67 in 70) the lower boundary of the heart inclined downwards from the auricle to the apex, in a direction from right to left. In one instance the lower boundary of the heart was an inch, and in another it was only the tenth of an inch lower at the apex than at the lower end of the sternum. Between these two extremes there was every variety, the average dip of the lower boundary of the heart from that point to the apex being about half an inch. (Note 6.)

The inclination or dip of the lower boundary of the right ventricle ceased at the apex, and thence the lower boundary of the heart curved gently upwards.

(3) The lower boundary of the heart usually extended from two inches to two inches and three-quarters to the left of the middle line of the sternum (in 43 instances in 65), but in one-third of the cases (20 in 65) it only extended from an inch and a quarter to an inch and three-quarters, while in five instances it extended as much as three inches to the left of the middle line of the sternum. (Note 7.)

The Top of the Arch of the Aorta.—The top of the arch of the aorta, which is indicated by the adjacent origin of the innominate and left subclavian arteries, forms the upper limit of the system of the heart and great vessels. The position of the top of the arch, like that of the lower border of the heart, is subject to great variety.

In one instance the top of the arch was an inch and a half below the top of the manubrium, so that it was buried deep down in the chest and the innominate artery did not appear in the neck. In another, the top of the arch was seated in the neck, being half an inch above the top of the sternum, so that before the chest was opened the whole innominate artery was visible in the neck, coursing upwards and from left to right across the front of the trachea. The summit of the aorta occupied in different instances every variety of position between these two extreme limits. In five cases it was above, and in six it was on a level with the top of the manubrium; while in seven, instead of being thus almost or quite visible in the neck, it was situated quite an inch below the top of the manubrium and the whole of the innominate artery was shielded by that bone. In two-thirds of the instances (30 in 48) however, the top of the aorta occupied an intermediate place behind the upper half of the manubrium, its average position being half an inch below the top of that bone. (Note 8.)

In forty-eight instances the position both of the lower boundary of the heart and the upper boundary of the arch of the aorta was observed, and, as might have been looked for, there was a general correspondence in the position of these two boundaries in those cases in which they occupied respectively a very high or a very low position. Thus, of the five cases in which the top of the arch of the aorta rose above the top of the sternum, the lower boundary of the heart was situated above the lower end of the sternum in three, at that point in one, and less than half an inch below it in one. Again, the top of the arch of the aorta

was situated below the upper end of the manubrium in the whole of six cases in which the lower boundary of the heart was from half an inch to an inch and a quarter below the lower end of the sternum. Again, of seven instances in which the top of the arch was deep in the chest, being more than an inch below the top of the manubrium, in three the lower boundary of the heart was below the lower end of the sternum, in one at that point, and in three above it. Here the correspondence of the upper and lower boundaries is rather indicated than kept up, but this correspondence can scarcely be recognized when we compare these boundaries with each other in those cases in which they occupied a less extreme position. (Note 9.)

The Boundary-line between the Upper Border of the Heart and the Lower Limit of the Great Arteries. — The origin of the pulmonary artery and the top of the auricular portion of the right auricle may be regarded as the upper boundary of the heart and the lower boundary of the great arteries. The highest position of the origin of the pulmonary artery was at the top of the second cartilage, while that of the top of the auricle was a little higher or on a level with the first space. The lowest position of the origin of the pulmonary artery was the upper edge of the fourth cartilage, while that of the top of the auricle was a little less low, or on a level with the lower border of the third space. Between these two extreme limits the origin of the pulmonary artery and the top of the right auricle occupied every variety of position, but their favorite seat was at or on a level with the second space and the third costal cartilage, which was the situation of those parts in two-thirds of the instances (36 in 49 for the pulmonary artery; 43 in 63 for the top of the auricle).

In the majority of instances there was but little difference between the height of the origin of the pulmonary artery and the top of the right auricle, the height of the two being identical in one-fourth of the instances (10 in 44), and the difference in their height being respectively less than the third of an inch or the third of the breadth of a space or cartilage in one-half of them (21 and 20 in 44). Of the remaining instances, in twelve the difference of the height of those two parts varied from one-third to two-thirds of an inch or two-thirds of a space or cartilage, and in one the difference of their height amounted almost to an inch. As a rule, the origin of the pulmonary artery tended to be higher in position than the top of the right auricle, the former part being the higher of the two in twenty instances, and the latter part being the higher of the two in fourteen instances. (Note 10.)

The varying position, higher or lower, of (1) the pulmonary artery, (2) the aorta, (3) the right ventricle, and (4) the right auricle, in relation to the costal cartilages and the spaces between them, and to the sternum will next be considered.

The Pulmonary Artery.—A knowledge of the position of the pulmonary artery is important to the clinical worker, because it is near the surface of the chest, and because the signs afforded by it reveal the condition of the cavities and valves of the heart, and the ease or difficulty with which the blood finds its way from and to those cavities, the lungs, and the body. Among those signs are, the character of the first sound, whether loud and sharp, or feeble and almost silent, or presenting a pulmonic murmur; the character of the second sound, whether feeble or intense, blunt or sharp, or presenting a double sound, giving in quick succession the aortic and the pulmonic second sounds or the reverse, the latter sound being the louder of the two.

The trunk of the pulmonary artery varied in length from three-quarters of an inch to two inches and a half. In more than a third of the instances (17 in 46) the artery was from an inch to an inch and a half in length, while in less than a third of them it was below (15 in 46), and in less than a third of them (14 in 46) it was above that length.

The vertical measurement of the right ventricle from the origin of the pulmonary artery to the lower boundary of the heart, varied in these instances from two inches and a half to a little over four inches. The length of the ventricle thus measured was from three inches to three inches and a half in less than one-half of the cases (20 in 46).

The proportion between the length of the pulmonary artery and the length of the right ventricle, measured from above downwards, presented great variety. In one instance the length of the artery was nearly equal to the length of the ventricle, that of the former being two inches and a half, that of the latter scarcely three inches; while in two others the vertical measurement of the ventricle was five times as great as that of the artery, the length of the latter in one instance being three-quarters of an inch, and that of the former being fully four inches. The average length of the ventricle in relation to that of the artery was as three to one. As a rule, the length of the pulmonary artery regulated the proportion in length which that vessel bore to the ventricle; thus in the whole of the fifteen instances in which the length of the pulmonary artery was less than an inch, the length of the right ventricle was more than three times that of the artery; while in the whole of the fourteen in which the artery

was an inch and a half in length and upwards, the length of the right ventricle was less than three times that of the vessel. (Note 11.)

As we have already seen, the origin of the pulmonary artery varied in position from the second to the fourth cartilage, its usual situation being the second space and the third cartilage. The top of the pulmonary artery was in one instance almost as high as the clavicle, and in almost one half of the cases (25 in 63) it was situated behind the manubrium or the first rib; while in one case it was so low as to be almost on a level with the upper edge of the third cartilage. In more than one half of the cases (33 in 63) it was seated behind the first space or the second cartilage. (Note 12.)

The situation of the pulmonary artery during its course is regulated by the length of the vessel and by the position of its starting place and upper end. In one instance it was so high as to be entirely concealed by the manubrium, while in another it was so low as to be entirely covered by the third cartilage and third space. The artery was rarely limited in position to one space or one cartilage: thus in but one instance it only occupied the first space, and in but one it was quite covered by the second cartilage. The artery usually lay behind one space and one costal cartilage (35 times in 60), but in one-third of the instances (21 in 60) it extended to an additional space or cartilage. In two-thirds of the instances it was present behind the second cartilage (43 in 60); in more than half of them it lay behind the first space (35 in 60), and in nearly as many behind the second space (32 in 60); while in one-fourth it was covered by the third cartilage (15 in 60), and in one-sixth by the manubrium (9 in 60). (Note 12.)

When the pulmonary artery was long (it was so in 14 of 46 instances), its origin occupied, as a rule, a low position. Thus in sixteen instances the origin of the artery was entirely above the second space, and in only two of these was it long, while in seven it was short. On the other hand, in thirty instances the pulmonary artery at the first part of its course was at or below the second space, and in twelve of them the artery was long, while in eight it was short. (Note 12.)

The Arch of the Aorta.—The arch of the aorta is not, like the pulmonary artery, visible in its whole course from its root to its summit, being hidden at its root by the right auricle and ventricle. I shall, therefore, not speak here of the whole of the ascending aorta, but of that portion of it which comes into view above the right auricular appendix and between it and the beginning of the pulmonary artery and the arterial cone of the right ventricle.

The arch of the aorta, from the part in its course just spoken of where it first becomes visible, to its highest point at the origin of the innominate and left carotid arteries, varied much in length. In two female subjects, one aged nine, the other a few years older, the arch was an inch and a half in length, but in the adult subject its length ranged from an inch and three-quarters to three inches. The arch, measured from the lower to the higher points just named, was from just over two inches to two inches and a half in length in two-fifths of the instances (19 in 47), that being about the average or standard length; from an inch and three-quarters to two inches in more than one-fifth (11 in 47), and from two inches and a half to three inches in less than two-fifths of them (17 in 47). (Note 13.)

Viewed in proportionate relation to the length of the body, measured approximately from the chin to the pubes, the vertical measurement of the arch varied from one-seventh to one-fourteenth of the vertical measurement of the body thus taken, and in one-half of the instances (23 in 45) the length of the aorta was one-tenth that of the body.

In three instances the vertical measurement of the arch of the aorta was the same as the vertical measurement of the right ventricle taken from the part at which the aorta was visible to the lower boundary of the organ. In two instances the arch was longer than the ventricle in the proportion of ten to nine, but in the remainder the length of the ventricle was greater than that of the aorta, the relative proportion varying from 10 to 10·1 to 10 to 19·17, so that in the last example the ventricle was nearly twice as long as the arch. The average length of the arch in proportion to that of the right ventricle was about 10 to 14 (14 in 47).

The variation in the proportionate length of the arch of the aorta and the right ventricle, although thus considerable, is not nearly so great as the variation in the proportionate length of the pulmonary artery and the right ventricle; since that artery varied in length from more than one-half to less than one-fifth of the vertical measurement of the ventricle, while the arch was about the same length as the vertical measurement of the ventricle at one end of the scale, and was of half that length at the other end.

There was some correspondence between the length of the aorta and that of the pulmonary artery. Thus the pulmonary artery was short, long, or of medium length in two-fifths of the instances in which the aorta was respectively short, long, or of medium length (13 in 33). In the remaining instances (20 in 33) this strict proportion was not maintained, but in only two of them was the difference in

the proportionate length of the vessels great, the aorta being long while the pulmonary artery was short.

The position of the lower boundary of the heart in relation to the lower end of the sternum, whether above, at or below that point is, as a rule, governed to a considerable extent by the length of the arch of the aorta. Thus in nine instances in which the arch was short, measuring two inches or less, the lower boundary of the heart was above the lower end of the sternum in seven instances, and below that point in two. The other circumstances that regulate the position of the lower boundary of the heart in relation to the lower end of the sternum are (1) youth; (2) the distension or collapse of the right ventricle; (3) the length of the sternum; (4) the important influence of the higher or lower position of the sternum, higher when the chest is ample, being of an inspiratory type, and lower when the chest is narrow and flat, being of an expiratory type; (5) the higher or lower position of the top of the arch of the aorta which is often ruled by (4) the lower or higher position of the sternum; (6) the extent to which the heart shrinks upwards after death which is evinced by the space intervening between the lower boundary of the heart and the lower boundary of the *front* of the pericardium; and (7) the elevation or depression of the diaphragm, which is the most important influence in producing respectively the elevation or depression of the heart, and which may be caused by (*a*) the contraction or expansion of the lungs, or (*b*) the distension or collapse of the abdomen. These points are illustrated by the two exceptional cases just cited, in which, although the arch of the aorta was short, the lower boundary of the heart was below the level of the lower end of the sternum. Both of these cases were quite young (1); in both the vertical measurement of the right ventricle was long, while in one of them that cavity was distended and large (2); in both of them the sternum was short, its length being less than four inches in one, while in the other it was four inches and a half (3); again in one of them the sternum was high, the length of the neck being only two inches, that of the sternum four inches and a half, and that of the abdomen fourteen inches, while in the other instance in which the right ventricle was large, the sternum was low in position, the length of the neck being almost four inches, that of the sternum less than four inches, and that of the abdomen only ten inches and a half (4). In neither of these examples was the position of the lower boundary of the heart lowered owing to the low position of the top of the arch, for in one of them that point was above the top of the sternum and in the other it was a little way below it (5). In fact this influence, which tended to elevate the lower boundary of the heart in relation to the lower end of the sternum was more than counter-balanced by the combined influences of which I have just spoken, all working in the opposite direction so as to lower the inferior border of the heart.

In further illustration of this point, the influence, namely, of the shortness or length of the arch in respectively raising or lowering the lower boundary of the heart, we find that of seventeen cases in which the aorta was long, measuring two and a half inches and upwards, in ten the lower boundary of the heart was below the level of the lower end of the sternum, in four it was at that point, while in only three was it above the lower end of the sternum. The three exceptional cases in which the lower boundary of the heart was above the level of the lower end of the sternum were adults of full size (1); the right ventricle was narrow and contracted in two of them (2); and in two the heart deviated to the left so that the lower border of the right ventricle was situated to the left of the lower end of the sternum, instead of being to the right, as is usual. The sternum was long in two of them, measuring in one case over seven inches (3); in all of them the sternum was low in position, the length of the neck being five inches and a half, four inches, and three inches and a half respectively, while that of the abdomen was in each instance less than fourteen inches (4); in one of them the top of the arch was situated above the top of the sternum (5); in one of them the space between the lower limit of the heart and the lower limit of the front of the pericardium was nearly an inch, while in another it was fully half an inch in width, showing that the upward shrinking of the heart after death had been considerable (6); and finally one of them, that in which the space between the heart and the lower rim of the pericardium was small, the stomach was globose and much distended so as to push the heart upwards (7*b*).

In twenty-three cases the arch of the aorta was of intermediate length, or from a little over two inches to two inches and a half, and in these the lower boundary of the heart was in equal relative proportion above, at, and below the level of the lower end of the sternum.

It is evident and is illustrated by what has just been said that if we group the cases as I have just done, according to the actual length of the arch of the aorta without relation to age or the dimensions of the body, we shall include some instances in which the arch of the aorta is relatively short or long with those in which it is respectively actually long or short. I have therefore grouped the

whole cases anew, and according to the proportional length of the aorta in relation to the length of the body. It will suffice here if I say that the results thus obtained are exactly confirmatory of those that I have just related, and show that the higher or lower position of the lower boundary of the heart in relation to the lower end of the sternum is to a considerable extent governed by the proportional shortness or length of the arch of the aorta. They show those results indeed more strikingly, for the conflicting element of (1) youth has been removed.

Two exceptional instances have been brought into the group in which the arch of the aorta was long in proportion to the length of the body, that were not included in the parallel group in which the arch was actually long. In these two examples the lower boundary of the heart was above the level of the lower end of the sternum, although the aorta was proportionally long. The heart was lifted directly upwards to a great extent in both of these instances, in one of them by very great enlargement of the liver, upwards, as well as downwards, owing to the presence of malignant disease in the organ, the sternum being in this case very long (6·8 inches); and in the other by excessive distension of the stomach and intestines owing to peritonitis, the sternum in this instance being short (4·7 inches) and the top of the aorta being situated in the root of the neck, a third of an inch above the level of the top of the sternum (7*b*).

The Right Auricle.—The right auricle is, as a rule, hidden from observation by the couch of lung that is interposed between it and the sternum and cartilages. It comes, however, to the surface in cases of pericarditis when the effusion into the sac accumulates in sufficient quantity to press aside that portion of lung with which the auricle is covered. With the exception of the important point just considered, the right auricle cannot be recognized locally by the clinical observer, the condition of that cavity being in fact best told by the state of the veins in the neck. The right auricle measured from the top of its auricular portion to its lowest point, varied in length from one inch to four inches and a half. Its length was usually from two and a half to three and a half inches (in 41 of 62 instances). In one-fifth of the cases (12 in 62) its length was less than two and a half inches; but one-half of these were youthful subjects (7 in 12). The vertical measurement of the right ventricle was longer than that of the right auricle in more than two-thirds of the cases in which the comparison was made (35 in 49); in one-fifth of them the two cavities were nearly or quite of equal length (10 in 49); and in one-twelfth of them the auricle was longer than the ventricle. (Note 14.)

The auricular portion of the auricle, which during life laps, like a tongue, to and fro, from right to left and back again, was usually nearly on the same level as the top of the right ventricle, the top of the auricle being of the same height as that of the ventricle in ten instances, higher than that of the ventricle in fourteen instances, and lower in twenty. It was at the lower boundary that the right auricle failed. In one case, in which there was fatal hemorrhage, the auricle, which was quite insignificant in size, was only half as long as the ventricle. Usually, however, the auricle was shorter than the ventricle by from one-tenth to one-third of its vertical measurement (in 29 of 35 instances).

The right auricle, from the variable extent to which, on the one hand it receives blood, and on the other retains or parts with it before, during, and after death, and from its passive nature, is more variable in form and size than any other cavity of the heart. This point will be briefly illustrated when the lateral dimensions of the cavities are considered.

The Right Ventricle.—The vertical measurement of the right ventricle in relation to the pulmonary artery and the aorta has already been considered.

The right ventricle, measured from the origin of the pulmonary artery to the lower boundary of the cavity, varied in length from two inches and three-quarters to four inches. In one-fifth of the instances (9 in 46) the length of the ventricle thus measured was less than two inches, the majority of these being youthful subjects (5 in 9); in nearly one-half of them (20 in 46) this measurement was from three inches to nearly three inches and a half; and in the remainder it was three inches and a half and upwards, being fully four inches in six of them. The variable dimensions and form of the ventricle will be briefly noticed when its lateral measurements are considered. (Note 15.)

The extent of the vertical measurement or length of the right ventricle produces a marked influence on the position of the lower boundary of the heart in relation to the lower end of the sternum. Thus, of the nine cases in which the ventricle was short, its lower boundary was above the level of the end of the sternum in five instances, and below that level in only one; while of the sixteen instances in which the ventricle was long, in ten of them its inferior border was below the end of the sternum, while in only six of them was it above that point. It is, indeed, self-evident that the lower border of the ventricle must be lower in position when the cavity increases, and higher when it lessens in size.

The extent to which the upper part of the bony sternum covers the great arteries, and the lower part of it, the heart, is very variable. In one instance the great arteries occupied only the upper fourth of the sternum, while the heart occupied its lower three-fourths. In another instance this proportion was to a considerable extent reversed, for the vessels lay behind the upper five-eighths of the bone, the heart itself being limited to its lower three-eighths. In three-fourths of the instances (39 in 52) the greater share of the sternum lay in front of the heart, but in one-fourth the greater share of the bone was given to the great vessels. On an average, the position of the heart was behind the lower four-sevenths, and that of the great arteries was behind the upper three-sevenths of the sternum. (Note 16.)

(II.) The Position of the Heart and Great Vessels from Side to Side.

Relation of the Breadth of the Heart to the Breadth of the Chest.—The proportionate transverse diameter of the heart, compared with the transverse diameter of the chest, varied considerably. Thus in one instance, in which death was the result of hemorrhage, the width of the heart was less than one-third of the width of the chest, on a level with the lower end of the ensiform cartilage (3·2 to 10 inches); while in another instance the measurement across the heart was nearly two-thirds of that across the chest (5·1 inches to 8·2 inches).

In a large number of the cases observed (39 in 65) the breadth of the heart was somewhat less than one-half of the breadth of the chest, the proportion varying from 10 to 4 to 10 to 5. In one-sixth of the instances (11 in 65) the width of the heart was less than two-fifths (10 to 3 to 10 to 3·9), and in one-third of them (15 in 65) it was more than one-half (10 to 5 to 10 to 6·2) of the width of the chest. The size of the chest from side to side did not appear to exercise any material influence on the proportional breadth of the heart, but the heart was more frequently of the average proportional width in those instances in which the chest was of medium breadth (9 to 9·9 inches) than in those in which it was either wide (10 to 12 inches) or narrow (6 to 8·9 inches). Thus, the heart was of the average proportional breadth in five-sixth of the instances in which the chest was of the medium breadth (10 in 12); in one-half of those in which the chest was wide (12 in 22); and in two-thirds of those in which the chest was narrow (19 in 31). The heart was comparatively wide and comparatively narrow in equal numbers in those instances in which the chest was wide (6 of each kind in 22); while the organ was more frequently comparatively wide than narrow, in those in which the chest was narrow (wide in 8, narrow in 4, of 31). Great distension and great collapse of the abdomen produced a marked effect on the proportionate width of the heart in relation to that of the chest. Thus, in fully two-thirds of the instances in which the heart was proportionally narrow, the abdomen was distended (8 in 11), and in one-half of these the distension was very great (4 in 11); while in one of the three remaining cases the abdomen was large, in one it was of moderate size, and in only one was it small. Then the reverse took place in those cases in which the heart was proportionately wide, since in only one-fifth of them was the abdomen distended (3 in 15), and in but one of these was the distension very great. Distension of the abdomen seemed to produce this effect by acting in two directions, one upon the chest, by widening it, the other upon the heart itself, by lessening it. The chest is widened because the distended abdomen pushes the ribs outwards on either side, and elevates the lower border of the chest in front and at each side; and the heart is lessened because the distended abdomen compresses the heart upwards into the contracting space of the higher part of the cone of the chest, and so lessens the amount of blood in the organ. (Note 17.)

The proportional size of the anterior transverse diameter of the combined right auricle and ventricle, compared with that of the left ventricle, exercises a marked effect on the proportional breadth of the heart in relation to the breadth of the chest. This might indeed be anticipated, for when the proportional width of the combined right auricle and ventricle is great in relation to the width of the left ventricle, the right cavities are distended with blood, and the whole heart is consequently large, measured from side to side. In more than one-half of the cases (7 in 12) in which the proportional breadth of the heart to that of the chest was great, the proportional breadth of the combined right auricle and ventricle to the left ventricle in front was very great, the former being about ten times wider than the latter; and in none of them was the proportional breadth of the right cavities small. Again, in almost one-half of the instances (5 in 11) in which the proportional width of the heart in relation to that of the chest was small, the proportional width of the right auricle and ventricle in relation to that of the left ventricle was also small, the ratio being about 10 to 4. (Note 18.)

Extent to which the Heart occupied the Right and the Left Sides of the Chest.—The extent to which the heart occupied re-

spectively the right and the left sides of the chest varied much in different instances. Thus in one example, the heart extended one inch and a tenth to the right and four inches to the left of a vertical line drawn down the middle of the sternum; and in another the organ extended nearly two inches and a half to the right, and only two inches and a quarter to the left of that line; while in two other instances the heart occupied the right and the left sides of the chest in exactly equal proportions. Thus, taking the two extreme cases, in one of them one-fifth of the heart occupied the right side, and four-fifths of it the left side of the chest; while in the other fully one-half of the heart was lodged in the right side, and less than one-half of it in the left side of the chest.

There was every gradation of difference between these two extreme examples. In fully two-fifths of the instances (27 in 67) one-third of the heart or less was situated in the right side, and two-thirds of the heart or more, in the left side of the chest; while in fully two-fifths of them (28 in 67), three-fifths of the heart or less was seated in the left side, and two-fifths of it or more in the right side (literally 16 to 10).

In twelve intermediate or standard instances, the heart was distributed to the right and to the left of the middle line of the sternum in the proportion respectively of ten and eighteen, and this was the average position of the organ in sixty-seven bodies, so that nearly two-thirds of the organ lay in the left side, and more than one-third of it in the right side of the chest. (Note 19.)

The influences that cause the deviation of the heart towards the right or the left side of the chest, are (1) before all others, the difference in size of the right lung and the left; (2) the encroachment upwards of the liver or the stomach to an unusual extent on the right or the left side of the chest respectively; (3) the position of the patient before death on the right side or on the left, an occurrence that may take place in certain rare cases, such, for instance, as bed-sores and affections of one side of the chest; (4) the shrinking of the heart upwards after death, as evinced by the extent of the space intervening between the lower boundary of the heart and the lower boundary of the *front* of the pericardium; (5) the shortening of the aorta; (6) the relative size of the heart and of its cavities, measured from side to side. There are doubtless other influences at work to produce the effect in question, but I have not discovered them.

(1) Of the small number of instances (6 in 66) in which the heart swerved very far to the left, so as to occupy that side of the chest to a greater extent by from three to four times than the right side of the chest, the two lungs were equal in size in one-third (2 in 6), while the right lung was greater than the left in the remaining two-thirds. On the other hand, of the cases in which the heart was lodged equally in the right and the left sides of the chest (3 in 66), and those in which it bore only a little more to the left than the right side of the chest (12 in 66), the two lungs were of equal size in one-fourth, and the left lung was larger than the right in the remaining three-fourths. Thus in none of the instances in which the heart deviated greatly to the left was the left lung larger than the right; and in none of those in which the heart tended towards the right side of the chest was the right lung greater than the left. In the whole of the remaining instances, with a few exceptions, an analogous condition obtained, the right lung being the larger when the heart was lodged to an unusual extent in the left side of the chest, and the left lung being the larger when the heart was lodged to an unusual extent in the right side of the chest. (Note 20.)

(2) The position of the upper surface of the liver, covered by the diaphragm, was higher in the right side of the chest than that of the stomach in the left side of the chest in all but a fraction of the instances observed (57 in 61). On an average, the liver at this situation was higher than the stomach by more than half an inch (·6 inch). In two-fifths of the cases (25 in 61) the heart occupied the left side of the chest to an unusual extent; of these, in nearly two-thirds the height of the liver in relation to that of the stomach was above the average (14 in 25); in nearly one-third it was below the average (7 in 25); and in a fraction it was at the average (3 in 25). In all but one of the five instances in which the heart was very far to the left, the relative height of the liver was above the average. In one-fourth of the cases (14 in 61), the heart occupied the right side of the chest to an unusual extent, and in nearly three-fifths of these (8 in 14) the height of the liver was below the average, while in fully two-fifths of them (6 in 14) it was above the average. When the top of the liver encroached to an unusual proportional extent on the right side of the chest, it may be said that the unduly-elevated organ tended to displace the heart to the left. There were, however, a few remarkable exceptions to this rule. Thus, in one instance the heart occupied equally the right and the left sides of the chest, and yet the top of the liver rose higher by nearly an inch and a half into the right side of the chest than the stomach did into the left side of the chest. The reason of this was obvious.

There was contraction of the right lung in this case, owing to phthisis, with the effect of drawing both the heart and the liver mainly into the space previously occupied by the right lung.

(3) I have no after-death evidence to show that the position of the patient on the right side or the left during the period preceding death caused the heart to occupy unduly the right or the left side of the chest. We know, however, that during life the heart falls towards the side on which the person lies. At the same time that side of the chest expands less during inspiration than the opposite side, owing to the restraint offered to the movement of the ribs that bear the weight of the chest, while, to compensate for the deficient expansion of the restrained side, the free side of the chest expands to an increased extent. After death, the organs, as a rule, retain pretty nearly the place they occupied during life, and the effect of position during life in displacing the heart more towards the right side or the left, is retained after death.

(4) When the heart shrinks upwards, so as to leave a considerable space between the lower boundary of the organ and the lower boundary of the *front* of the pericardium, the heart, as a rule, bears more towards the right than the left side of the chest. Thus the space below the heart was large in two-thirds of the cases in which that organ bore unusually to the right (8 in 12); and in only two-fifths of those in which it bore unusually to the left (8 in 19).

(5) I am of opinion that in those cases in which the heart shrinks thus upwards, and bears unusually to the right, the contraction and shortening of the aorta is one of the principal agents that draws the apex and the body of the heart to the right as well as upwards.

(6) The relative size of the heart and of its cavities, measured from side to side, exercised much less influence than the relative size of the right and left lung, and the relative height of the liver and stomach, on the extent to which the heart occupied after death the right and left sides of the chest respectively.

When the heart is large, the lungs necessarily make way for it, to the right and left equally if the development of the lungs is equal; but when one lung is expanded and the other is contracted, the heart when large encroaches more upon the contracted than the expanded lung, for that lung offers the least resistance. The stronger influence of the greater size of one lung overrides then the weaker influence of the size of the heart. But it is evident that the size of the heart must produce an influence supplementing and modifying the influence of the greater size of one lung. When the heart is large it enhances the influence of the greater size of one lung, and the heart encroaches more on the side containing the contracted lung; and when the heart is small it lessens the influence of the greater size of one lung, and the heart encroaches less on the side containing the contracted lung. Thus in the large group of cases in which the heart occupied the left side of the chest to an unusual extent (1 to 3·9 to 1 to 2, in 23 in 60), and in the equally large group in which the heart was distributed in the average proportion to the right and left sides of the chest (1 to 1·5 to 1 to 1·9 in 23 in 60), the heart was large in fully one-fourth of the respective instances (6 in 23 and 7 in 23), while in no instance was the heart large in the group in which that organ occupied the right side of the chest.

The heart was small in two of the three instances in which the organ occupied the right and the left sides of the chest to an equal extent. The heart is attached at the centre of the chest, behind, to the roots of the lungs by the pulmonary veins and pulmonary arteries; and above and in front, to the great arteries and the descending vena cava from which it is suspended. The heart, therefore, when it does not bear to the left or to the right owing to the greater or less size of the right or left lung, hangs directly downwards from the points of its suspension at the centre of the chest, and tends to occupy a central position, bearing equally to the right and to the left.

Breadth of the Combined Right Auricle and Ventricle in Relation to that of the Left Ventricle as seen in Front.—The breadth of the combined right auricle and ventricle in relation to the breadth of the left ventricle as seen in front, varied from 10 to 1 to 10 to 4½. Thus the right cavities occupied almost the whole front of the heart in some examples, and little more than two-thirds of it in others. Every shade of variation existed between these two extreme instances; but the average or standard proportion between the breadth of the right cavities and that of the left ventricles in front was as 4 to 1. (Note 21.)

Breadth of the Right Auricle.—The auricular portion of the right auricle varied in breadth from a little over half an inch (·55 inch) to two inches and a third (2·3 inches), its average breadth being one inch and a third (1·3 inch). (Note 22.)

The body of the right auricle[1] varied in

[1] The right auricle is about half an inch wider, and the right ventricle is about half an inch narrower than the measurements given in this article. Those measurements have been necessarily taken from the right auriculo-ventricular furrow, which is the apparent boundary-line between the right

breadth from a quarter of an inch to an inch and a half, its average breadth being four-fifths of an inch (8·1 inch). (Note 23.)

The left edge of the auricular portion of the right auricle extended to the left of the left edge of the sternum in four instances; it was placed nearer to the left than the right edge of the sternum in twenty-four cases; it was situated about midway between the left and the right edge of the sternum in eight instances; and it was nearer to the right than the left edge of that bone in fourteen. (Note 24.)

The right edge of the right auricle extended to the right of the right edge of the sternum to an extent varying from a quarter of an inch to an inch and three-quarters, so that to that extent the auricle lay behind the right costal cartilages. The right auricle extended on an average from half an inch to a little over an inch to the right of the sternum. (Note 25.)

The auricular portion of the right auricle was wider than the body of the auricle in all but two instances, in which instances their breadth was the same. As a rule, the auricular portion was wider than the body of the auricle in the proportion of ten to six and a half (10 to 6·4), but in two instances that portion was nearly three times as wide as the body of the auricle. (Note 26.)

The proportional breadth of the auricular portion of the right auricle varied from two-fifths to one-fifth of the breadth of the heart itself. The width of the heart was, on an average, nearly four times as great as that of the auricular portion of the right auricle. (Note 27.)

The proportional breadth of the body of the right auricle varied from about a fourth (10 to 36) to a ninth (10 to 86) of the breadth of the heart. In one exceptional case in which death took place from hemorrhage, the heart was twelve times as wide as the right auricle, that cavity being quite empty. The width of the heart was, on an average, nearly six times as great as the width of the right auricle. (Note 28.)

Breadth of the Right Ventricle. — The breadth of the right ventricle[1] varied from four-fifths (in 6 of 38 instances) to a little over one-half (in 11 of 38 instances) of the whole breadth of the heart. The average or standard breadth of the right ventricle was two-thirds of the breadth of the heart (10 to 15), and in one-half of the cases observed the proportional width of the right ventricle in relation to that of the heart was above (19 in 38), and in one-half of them it was below that average (19 in 38). (Note 29.)

The breadth of the arterial cone of the right ventricle a little way below the origin of the pulmonary artery varied from four-fifths to two-fifths of the breadth of the right ventricle at its middle, the average width of the arterial cone being nearly three-fifths of that of the body of the right ventricle. As a rule, when the body of the right ventricle was wide or narrow in relation to the heart, the arterial cone was respectively narrow or wide in relation to the body of the right ventricle. (Note 30.)

The vertical diameter or length of the right ventricle,[1] measured from the origin of the pulmonary artery to the lower boundary of that cavity, was somewhat shorter than the transverse diameter or breadth of the ventricle in one-sixth of the cases (5 in 30). In the rest of them the length of the right ventricle was greater than its breadth. In one instance the length of the ventricle was to its breadth as 17·3 to 10, but the average or standard measurement of the length to the breadth of that cavity was as 4 to 3. (Note 31.)

The breadth of the right ventricle in relation to that of the right auricle below its auricular portion varied from 10 to 1·4 to 10 to 5·2, the average proportion being 10 to 3. (Note 32.)

The actual breadth of the right ventricle in adults, without distinction of sex, varied from two to four inches. In three-fifths of them the width of the ventricle was from three to three and a half inches (in 14 in 24); in one-fifth of them it was above three and a half inches; and in two-fifths of them it was less than three inches. (Note 33.)

In one instance the right ventricle extended further to the right than to the left of a vertical line drawn down the middle of the sternum, but in every other instance the ventricle extended more to the left than to the right of that line. In one case, nine-tenths of the right ventricle was situated in the left side of the chest, and only one-tenth of it in the

auricle and ventricle, but is situated half an inch to the right of the real boundary-line between those cavities.

[1] The right ventricle is about half an inch narrower, and the right auricle is about half an inch wider than the measurements of those cavities given in this article, for the reason stated in the preceding foot-note.

[1] As the breadth of the body of the right ventricle is about half an inch narrower than the measurements of that cavity given in this article, for the reason stated in the foot-note at page 379, the actual relation of the transverse diameter or width of the body of the right ventricle here stated to that of the *conus arteriosus*, and to the vertical diameter or length of the ventricle, is half an inch narrower than the proportional measurements here given.

right side; but, on an average, the ventricle extended nearly three times farther to the left than the right of the middle line (27 to 10). (Note 34.)

The limits of the body of the right ventricle and of its arterial cone are indicated, (1) to the left by the position of the longitudinal furrow between the ventricles; and (2) to the right by the position of the transverse furrow between the right ventricle, including the right edge of the origin of the pulmonary artery and the right auricle, including its auricular portion.

(1) As a rule, the inter-ventricular furrow takes an oblique direction outwards, or to the left from above downwards, so that the ventricle occupies a wider space below than above (in 26 of 39 instances). In a small number of cases (6 in 39) the reverse takes place, and the furrow tends inwards, and then outwards with a peculiar double curve as it descends. In these instances the right ventricle was in a state of contraction, and the left ventricle was exposed to a large extent, while in those in which the septum inclined markedly outwards during its descent, the right ventricle was distended so as to cover all but a small portion of the left ventricle. The greatest inclination of the longitudinal furrow to the left was one inch, and its greatest inclination to the right was half an inch (·45 inch). (Note 35.)

In one instance, a case in which the right ventricle was contracted, the longitudinal furrow in its descent curved to the right, and the body of the right ventricle towards its left border was completely shielded by the sternum; but in every other instance that cavity was covered in front to a greater or less extent by the cardiac costal cartilages, to the left of the lower half of the sternum. In a small proportion of the cases (6 in 36) the right ventricle lay behind the costal cartilages from end to end, from the sternum, namely, to the ribs to which they are united; and in half of these (3 in 6) the ventricle extended to the left, beyond the cartilages and behind the ribs. In the majority of the cases (19 in 36) the longitudinal furrow extended either up to the ends of the cartilages, a little beyond them, or half an inch or less to the right of them, so that in all these cases the cardiac cartilages covered the right ventricle almost or quite from end to end. In the remaining instances (17 in 36) a considerable portion of the cartilages, varying from less than an inch to more than an inch and a half (·7 to 1·7 inch) extended beyond the right ventricle. (Note 36.)

The body of the right ventricle, starting from a vertical line drawn down the middle of the sternum, extended to the left in all the cases, from a little over half an inch (·6 inch) to almost four inches (3·8 inch). Between these two extreme instances there was every shade of difference. In the great majority of the cases (35 in 52) the right ventricle extended from one inch and a half to two inches and a half to the left of the middle line of the sternum, and behind the cardiac cartilages. (Note 37.)

(2)[1] The transverse or right auriculo-ventricular furrow was situated to the right of the right edge of the lower portion of the sternum, and behind the right costal cartilages, in fully two-thirds of the cases (36 in 51), at that edge in a fraction of them (3 in 51), and to the left of that edge, and therefore behind the lower portion of the sternum, in one-fourth of them (12 in 51). In one instance the right auriculo-ventricular furrow extended an inch and a third (1·3 inch) to the right of the right edge of the sternum, so as to lie behind the right costal cartilages to that extent, and in five instances its right limit was situated behind the middle line of the sternum. Between these two extreme limits there was every gradation in the position of the right auriculo-ventricular furrow.

The left edge of the auricular portion of the right auricle gives, as a rule, very nearly the position of the right edge of the arterial cone of the right ventricle, where it is about to end in the pulmonary artery. The right edge of the arterial cone, starting from the tricuspid orifice, invariably inclines, as it ascends, from right to left. There was considerable difference in the degree of its inclination, which was measured by the distance between the right limit of the auriculo-ventricular furrow and a line drawn downwards from the right edge of the pulmonary artery. The right edge of the arterial cone swerved as it ascended from right to left in one instance, a man, to the extent of two inches, and in another, also a man, to that of a little over half an inch (·6 inch). There was every variety of inclination between these extreme instances, but in the great majority of cases (34 in 51) the curved line of the right border of the arterial cone bent downwards, with an inclination from left to right of from an inch to an inch and a half, the boundary line starting above from the right border of the origin of the pulmonary artery, and ending below in the auriculo-ventricular furrow. (Note 38.)

Breadth and Position of the Pulmonary Artery.—As the origin of the pulmonary artery is the point of convergence towards

[1] The transverse furrow, which is the apparent boundary-line between the right auricle and the right ventricle, is about half an inch to the right of the real boundary-line between those cavities. See note at page 379.

which the right ventricle propels its blood, this is the natural place for examining the position of that artery. The pulmonary artery forms, indeed, the pointed apex of a triangle, the body of which is constituted by the front of the right ventricle, its base by the lower boundary of that cavity, resting on the central tendon of the diaphragm, its left side by the longitudinal furrow, and its right side by the auriculo-ventricular furrow.[1]

The breadth of the pulmonary artery varied from a little over half an inch (·6 inch) to a little under an inch and a half (1·45 inch). Between these two extreme limits, both of which occurred in men, there was every kind of variation in the breadth of the artery. The width of the artery depended as much on the amount of blood that it happened to contain as on the natural size of the vessel. In one-third of the cases (18 in 45) the breadth of the artery varied from three-quarters of an inch to less than an inch, and of these three were boys and four were young people; and in one-third of them (17 in 45) the breadth varied from an inch to an inch and a quarter, and of these the youngest was a girl of 16, the rest being adults. The pulmonary artery was wider than the aorta in twenty-seven cases, narrower than the aorta in eleven, and of the same width as the aorta in six. (Note 39.)

In one instance the right border of the pulmonary artery at its origin lay two-thirds of an inch to the left of the sternum, and in another it was covered by the sternum to the extent of an inch, so that a mere rim of the artery (·25 inch) appeared in the second left space. Between these two extreme instances there was every degree of difference in the position of the origin of the pulmonary artery to the right or the left.

In two-thirds of the cases (31 in 45) the pulmonary artery was situated partly behind the sternum, and partly behind the upper cartilages and spaces to the left of the sternum; but in one-third of them (14 in 45) the vessel lay entirely to the left of that bone, and behind the upper spaces and cartilages.

Of those instances in which the artery lay completely to the left of the sternum, in three-fourths (11 in 14) the right border of the vessel was on a line with or a little beyond the left border of the bone, and in the remainder (3 in 14) it was placed from one-third to two-thirds of an inch to the left of that bone. Of the instances in which the artery lay partly behind the sternum, partly behind the cartilages and their spaces, in all but one-fifth (6 in 31) the vessel was situated to a greater extent behind the spaces than the sternum. In no single instance was the artery entirely covered by that bone. In the large majority of the cases, therefore, the greater part (in 25 of 45 instances), or the whole (in 14 of 45 instances), of the artery bore to the left of the sternum and presented itself behind the upper costal cartilages and their spaces from the first cartilage to the third space. (Note 40.)

Breadth of the Left Ventricle. — The breadth of the left ventricle as it is seen in front where it extends from the septum between the ventricles to the left border of the heart, varied from almost half an inch (·4 inch) to almost an inch and a half (1·4 inch). The average width of the ventricle was four-fifths of an inch (·8 inch). The proportion that the width of the left ventricle at its anterior aspect bore to the width of the whole heart varied from less than one-tenth (·08 to 10) to more than three-tenths (3·2 to 10). As a rule, when the ventricle was actually narrow, it was also proportionally narrow in relation to the breadth of the heart; and when the ventricle was actually wide, it was also proportionally wide in relation to the breadth of the heart. The exceptions to this rule are so few that I need not give the details here. (Note 41.)

Position of the Apex of the Heart.—The line of junction of the fourth and fifth ribs to their cartilages is a landmark of some clinical importance, for, aided by knowledge, this line may be pretty nearly ascertained during life. A downward bow is made by the descending curves of those ribs and of their cartilages, and their junction usually corresponds to the deepest part of the bow. The left boundary of the heart at the apex was situated in one instance an inch to the left, and in another instance an inch to the right of the junction of the fourth or fifth rib to its cartilage; in five cases out of forty-two this left boundary was at that junction, in eighteen it extended to the left of it, and in six it was seated to the right of it.

The relation of the lower anterior edge of the upper lobe of the left lung to the apex of the heart is a point of clinical value. The septum between the upper and lower lobes is situated a little way to the left of the apex of the heart, and this portion of the upper lobe is detached as it were from the body of the lung and dips downwards and forwards, so that it may devote itself to the protection of the apex around which it is folded, being situated outside, behind and in front of, above and slightly below the apex. A small tongue of lung, the existence of which I pointed out in 1844, frequently interposes itself between the front and under surface of the apex and the walls of the chest. This tongue of lung and the adjoining

[1] Or rather by a line half an inch to the left of the furrow. See note at page 379.

structure of the lower portion of the upper lobe play backwards and forwards with the forward and backward play of the apex of the heart. When the apex comes forward towards the ribs and spaces during the contraction of the ventricle, the tongue of lung retracts; when the apex retracts, the tongue of lung expands; and thus those two structures interchange with and adapt themselves to each other during the movements of the heart and the lungs. This tongue of lung that thus laps round and in front of the apex was present in two-fifths of the series of cases under observation (24 in 61), was absent in one-half of them (31 in 61), and was just indicated in the form of an inward curve in one-tenth of them (6 in 61). This tongue was strongly marked in one-third of the instances in which it existed (8 in 24), was slightly marked in another third (9 in 24), and was of intermediate form in the remaining third (7 in 24). Besides these instances, this tongue was present in eighteen additional examples in my possession: in one-half of these it was large and pronounced (9 in 18), in four of them it was of medium size, and in four it was small.

During and after death the apex contracts in one direction, or upwards and to the right towards the centre of the heart, and the left lung retracts in another direction or to the left. The heart is therefore more exposed after death than during life. This especially applies to the apex of the heart. As a rule, however, in these cases, the apex and the adjoining portion of the heart are still covered to a certain extent by lung (in 34 instances out of 58). In two of these instances the lung covered the heart from the apex towards the sternum to the extent of two inches and a half, but in the rest of them the extent of lung in front of the apex varied in breadth from an inch and a quarter to the tenth of an inch. In one-sixth of the cases (9 in 58) the edge of the lung was on a line with or crossed the apex, and in one-fourth of them (15 in 58) it was situated to the left of the heart, so as to expose the apex. The space thus left between the lung and the apex varied from one inch to the eighth of an inch. (Note 42.)

The Breadth and Position of the Ascending Aorta.—The breadth of the ascending aorta varied from half an inch to an inch and a half (1·45), its average breath being nearly one inch (·96 inch). (Note 43.)

The aorta was usually narrower than the pulmonary artery (in 27 of 44 cases), but it was sometimes wider than that vessel (11 in 44), and in a few instances (6 in 44), the two arteries were of equal breadth. When the aorta was less than an inch in width, it was very seldom wider than the pulmonary artery (in 2 of 36 cases); but when the aorta was an inch or more in breadth, it was more often the wider of the two arteries, in the proportion of nine to eight. (Note 44.)

The ascending aorta was completely covered by the sternum in nearly one-half of the cases (19 in 45), and of these instances, in one-third the artery was central (6), in one-third (6) it inclined to the right, and in one-third (7) it inclined to the left.

In one-third of the cases (15 in 45) the ascending aorta was visible to a greater or less extent to the right of the sternum, and in six of these the exposure of the artery to the right was great, the whole artery being brought into view in one case in which there was excessive distension of the abdomen.

In one-fourth of the cases (11 in 45), the ascending aorta was partially visible to the left of the sternum, but in only one instance did the breadth of the portion of the artery thus exposed amount to more than the third of an inch. (Note 45.)

The Position of the "Root of the Aorta,"[1] *including the Orifice, Valve,*[2] *and Sinuses of the Aorta.*—I possess only seven illustrations of the position of the root of the aorta. They, however, show the aortic valve in a variety of situations, and as the anatomical relations of the "root of the aorta" to the root of the pulmonary artery, and to the visible portion of the ascending aorta are very definite, it is easy to infer the position of the aortic valve, when we know that of the pulmonic valve, and that of the ascending aorta.

[1] I have adopted the term "root of the aorta" at the suggestion of Mr. Marshall and with the approval of Dr. Sharpey.

[2] Haller, writing in Latin, correctly designates the valves of the heart under the term "valvulæ," derived from "valvæ," folding doors, thus—"valvulæ semilunares," "valvulæ mitrales," "valvulæ in quas annulum venosum diviserunt." Senac (Structure du Cœur), speaking of the valves of the heart, uses the terms "valvules tricuspidales, mitrales, et sigmoides;" and Douglas, in his translation of Winslow, describes the "tricuspid valves," the "mitral valves," and the "semilunar valves."

Portal was apparently the first to speak of the auriculo-ventricular valves in the singular number, under the name respectively of "valvule mitrale" and "valvule triglochine," on the ground, long previously recognized by anatomists, that the flaps of each of those valves are attached to a valvular ring.

The English word "valve" has been applied by engineers and in common use to the mechanism, as a whole, for preventing the back-flow of fluid, and not to one or other of the flaps composing that mechanism. I have therefore, here and elsewhere, spoken of the semilunar flaps of the aortic or pulmonic valve, and not of the semilunar valves.

The ascending aorta, as it mounts upwards, curves first to the right and then to the left. The upper and lower ends of the curve bear to the left, and the centre of the curve bears to the right. When, therefore, the visible portion of the ascending aorta is situated far to the left or far to the right, the sinuses and valve of the aorta are also situated far to the left or far to the right, their bearing being always more to the left than that of the ascending aorta. The lower boundary of the pulmonic orifice corresponds with the upper boundary of the aortic orifice at the junction of the anterior and the left posterior flaps of the aortic valve. Nearly one-half of the root of the pulmonary artery is situated just above the left posterior aortic sinus, and more than one-half of it extends to the left of the root of the aorta. The root of the aorta extends obliquely downwards to the extent of about one inch below, and fully half an inch to the left of the pulmonary artery, the extent being greater or less in accordance with the oblique diameter of the root of the aorta.

In one instance the greater part of the anterior aortic sinus was situated behind the second left space from its upper to its lower boundary, while the remainder of the root of the aorta was covered by the left border of the sternum. In this case the ascending aorta occupied the left half or three-fifths of the sternum, the right side of that bone being occupied by the descending cava, and the pulmonic valve was situated entirely to the left of the sternum behind the second cartilage and the upper third of the second space.

In another instance the right border of the right posterior sinus of the aorta was present in the third right space close to the right edge of the sternum, and the whole of the rest of the root of the aorta was covered by the right three-fifths of the sternum, its left two-fifths being occupied by the arterial cone of the right ventricle. In that case the whole heart lay more to the right than to the left of the median line, the ascending aorta extended four-fifths of an inch to the right of the right edge of the sternum, and four-fifths of the origin of the pulmonary artery, which was on a level with the third cartilage, was covered by the sternum.

In the first of these two cases, the situation of the ascending aorta, and that of the origin of the pulmonary artery were high and much to the left, and the situation of the root of the aorta was correspondingly also high and much to the left. In the second of them, the ascending aorta and the origin of the pulmonary artery were low in situation, and were placed very far to the right; and the root of the aorta was also low in situation, and was placed very far to the right.

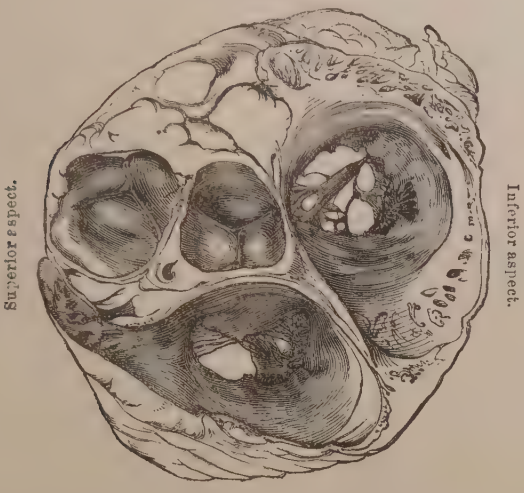
Of the remaining five instances, in two the root of the aorta was situated for one-fifth of its breadth in the second left space, and for four-fifths of its breadth behind the sternum on a level with the second space and the third cartilage. In two other cases, the proportion of the root of the aorta behind the sternum and to the left of that bone was about the same as in the two cases just quoted; but in one of them it was situated behind the third left cartilage and the upper third of the third left space; while in the other instance it was still lower, being on a level with the lower border of the third cartilage, the third space, and the upper border of the fourth cartilage.

The root of the aorta, including, as I have said, in that term the orifice, valve, and sinuses of the artery, was oblique in direction in all instances. Its longest or oblique diameter ranged from one inch to almost an inch and a half (1·4); its vertical diameter varied from ·8 inch to 1·05 inch; and its transverse diameter from ·8 inch to 1·2 inch. In three instances the transverse and vertical measurements were equal; in two the transverse diameter exceeded the vertical; and in two the vertical diameter exceeded the transverse.

Although the observation of the actual position of the root of the aorta in health has been limited to the seven cases just examined, yet we are able to infer its proximate position by the knowledge already obtained of the situation of the right edge of the ascending aorta, and of that of the origin of the pulmonary artery. The origin of the pulmonary artery was in one case as high as the upper border of the second cartilage, and in another it was as low as the upper border of the fourth cartilage. In the former case the root of the aorta must have been on a level with the second cartilage and the upper portion of the second space, while in the latter case it must have been on a level with the fourth cartilage and the upper portion of the fourth space. The usual position of the origin of the pulmonary artery was behind the second space or the third costal cartilage, and the usual position of the root of the aorta, following in the wake of its companion great artery, must have been on a level with the third cartilage and the third space. The average situation of the root of the aorta must therefore have been on a level with the lower portion of the third cartilage and the third space. In the seven cases just examined, the right edge of the ascending aorta was situated on a line to the right of the right edge of the root of the aorta, to an extent varying from the eighth of an inch to more than half an inch. In the same instances the left edge of the ascending aorta was situated on a line to the right of the left edge of the root of the aorta, to an extent varying from one-third

(·3 inch) to three-fifths of an inch. The extent to which the ascending aorta bore to the right in relation to the root of the aorta was governed by two circumstances: (1) the degree to which the ascending aorta was situated to the right or to the left: and (2) the distension or collapse of the artery. (1) The root of the aorta was situated further to the left in relation to the ascending aorta, when the position of the ascending aorta was far to the left than when it was far to the right. (2)

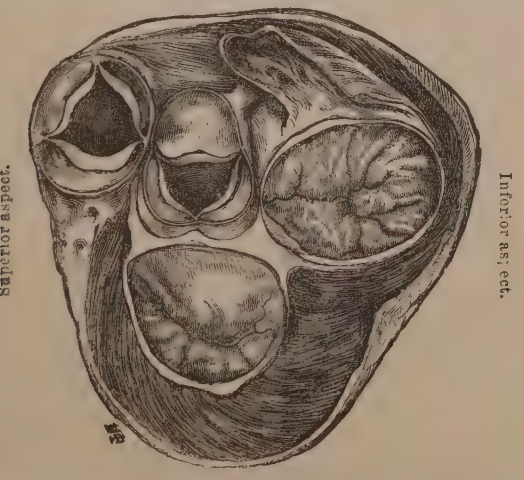
Fig. 47.



Posterior aspect of the heart.

Showing the pulmonic and aortic valves closed; the tricuspid and mitral orifices open. Period of the *diastole* of the ventricles.

Fig. 48.



Showing the pulmonic and aortic orifices open; the tricuspid and mitral orifices shut. Period of the *systole* of the ventricles.

The root of the aorta was further to the left in relation to the ascending aorta when the breadth of the artery was great owing to distension, than when it was small owing to collapse.

In one instance, a case with great intestinal distension, the whole of the ascending aorta was situated to the right of the sternum, and in that instance the greater portion of the root of the aorta must have been also situated to the right of the sternum. In another instance, the ascending aorta was situated to the extent of more than one-half of its breadth to the left of the sternum, and in that instance the greater portion of the root of the aorta must have been also situated to the left of the sternum.

In one-half of the cases (19 in 45), the whole of the aorta was covered by the sternum, and in most of these the greater part of the root of the aorta must have been also covered by the sternum, but its left border must have usually passed a little to the left of that bone, being situated behind one of the cartilages or spaces close to the left edge of the sternum.

Under these circumstances the average or standard position of the root of the aorta must have been behind the left two-thirds or half of the sternum on a level with the third cartilage and the third space, its left border being placed behind and below that cartilage at its articulation to the sternum. (Note 46.)

The Position of the Aortic Sinuses, and the Flaps of the Aortic Valve.[1]—The aortic orifice looks towards the apex of the ventricle in a direction to the left downwards, and slightly forwards. The aspect of the orifice is therefore oblique, its obliquity being usually quite as great from above downwards, as from left to right. When the heart bears unduly to the left, the downward obliquity of the aortic orifice is greater than when it bears unduly to the right.

The root of the aorta, including the aortic orifice, valve, and sinuses, projects forwards, in front of the mitral valve and the cavity of the left ventricle, so as to interpose itself between the orifice of the pulmonary artery above and the tricuspid orifice below. The root of the aorta thus separates those two openings from each other, the *conus arteriosus* being situated in front of it. When a section is made through the auricles across the base of the heart, so as to expose the four great openings of the heart, the pulmonic, the aortic, and the tricuspid orifices, viewed in their *natural position*, are seen to range themselves in a line from above downwards, the mitral orifice being situated behind the lower half of the aortic and the upper two-thirds of the tricuspid orifice. This line is not, however, straight, but is somewhat convex, the convexity looking backwards, so that the pulmonic and tricuspid orifices which are situated at the upper and lower portions of the line are somewhat in advance of the aortic orifice, which occupies the central position. When the line of the three orifices is looked at in front, it is seen to take an oblique direction from above downwards, and from right to left, the pulmonic orifice at the upper end of the line being situated partly behind and chiefly to the left of the left edge of the sternum at the second left cartilage and space, and the tricuspid orifice being situated behind the right half of the sternum at its lower portion.

[1] See Figs. 47, 48, and 49.

The "Aortic Vestibule," or Intervalvular Space of the Left Ventricle.—When the semilunar flaps of the aortic valve meet together so as to shut the aortic aperture, they fall backwards into a short space that I have described in my "Medical Anatomy" under the name of the "intervalvular space in the left ventricle." I have here, however, at the suggestion of Dr. Sharpey, adopted the appropriate name of the "aortic vestibule" for this space, which is well seen in the preparation from which Fig. 49 was taken, in which the semilunar flaps of the aortic valve are seen through an opening cut in the anterior flap of the mitral valve. The aortic vestibule bends forwards and to the right from the upper part of the left ventricle, and forms the channel between the cavity of that ventricle and its outlet at the aortic aperture. The walls of the aortic vestibule are rigid and unyielding, and it therefore retains its size during every stage of the action of the heart. These walls are muscular in front and to the left, where they are lined by rigid fibrous tissue, and where the space is situated immediately behind the *conus arteriosus* of the right ventricle; fibro-cartilaginous on the right, where they are formed by the central fibro-cartilage and "fibrous septum" of the heart; and fibrous behind, where they are formed by the base of the anterior flap of the mitral valve and the adjoining wall of the left auricle, upon which the posterior sinuses of the aortic valve are implanted.

The aortic vestibule occupies the centre of the heart, and is surrounded by all the more important parts of the organ. The *conus arteriosus* and the orifice of the pulmonary artery are in front of it; the tricuspid valve and right auricle are to the right of it; and the mitral valve and left auricle are behind it. During the ventricular diastole, when the left ventricle is of full size, the aortic vestibule is the narrowest portion or bent neck of the ventricle, and it then receives the flaps of the closed aortic valve which fall back into its cavity. During the ventricular systole, on the other hand, when the ventricle has completely contracted upon its contents so as to present an almost solid mass, the aortic vestibule moves downwards and to the left towards the apex, and becomes the widest part of the small remaining cavity, and the presence of this space then allows the mitral valve to remain closed up to the end of the systole by the pressure of the blood on its anterior flap.

The "aortic vestibule," as Mr. Marshall suggests, is a short *conus arteriosus*, since it corresponds in relative position and function, though not in shape or size, or in the structure of its walls, to the *conus arteriosus* of the right ventricle, immediately behind which it is situated. These

two analogous parts take opposite directions in relation to each other, and respectively to the ventricle from which they spring and the great artery to which they proceed. The right *conus arteriosus* ascends with a bearing to the left, and curves backwards to end in the pulmonary artery; while the aortic vestibule or left *conus arteriosus* ascends with a bearing to the right and bends forwards to terminate in the root of the aorta. Those two great arteries, following the direction of the

Fig. 49.

Aortic valve shut, seen in the aortic vestibule of the left ventricle, which parts are exposed by cutting a flap in the anterior cusp of the mitral valve and pinning it backwards.

Fig. 50.

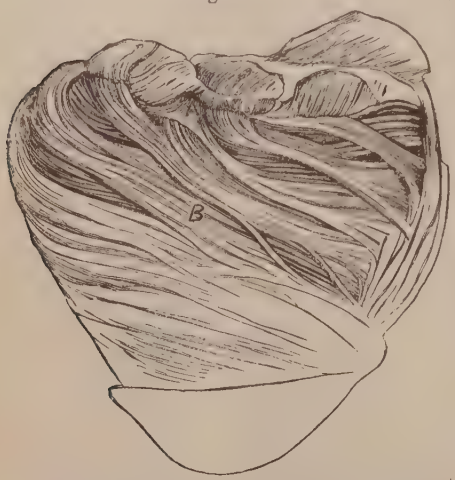
Other half of the heart represented in Fig. 51, showing the mitral and tricuspid valves and the fleshy septum (D) with its continuation in the form of a "fibrous septum," which is also seen in the companion figure.

conus arteriosus from which they respectively spring, cross each other in their onward and upward course, so that the pulmonary artery proceeds backwards to the left and then to the right, while the ascending aorta proceeds forwards to the right and then to the left. If the two cavities be looked at as a combined whole, each with its ventricle, its *conus arteriosus*, and its great artery, they resemble somewhat the curious double oil and vinegar flask that is met with so commonly in the most beautiful parts of South Germany.

The *central fibro-cartilage* and "*tendinous septum*" of the heart form, as I have just said, the right wall of the aortic vestibule. The fleshy septum terminates at its base in a strong tendinous aponeurosis or fibro-cartilage, which forms a part of great importance in the structure of the heart, and which is well seen in the preparation from which Figs. 49 and 50 have been taken. The muscular septum (D) is, in fact, converted at this region into a fibrous septum; but while the muscular septum separates the two ventricles, the fibrous septum separates the left ventricle from the right auricle as well as from the top of the right ventricle. Higher up this fibrous septum is converted into the central fibro-cartilage, which corresponds to the central fibro-cartilage and bone of the heart of the ox (Fig. 52), and which is converted into bone in a human heart in my possession. The central fibro-cartilage, as may be seen in Fig. 48, forms a firm bond of connection between the tendinous rings of the mitral and tricuspid orifices, the central or inner angles of the mitral and tricuspid valves, the right posterior sinus of the aorta, and the aortic vestibule. It also gives insertion to muscular fibres from the left and the right ventricles (Fig. 51 A), which, sweeping round from the left and the right respectively, blend together toward the base of the posterior longitudinal furrow, so as to form short central bands of fibres, which dip forwards at right angles to the circular fibres, deepening as they advance,

enter and go to form the septum and end in the central fibro-cartilage, which gives origin to numerous muscular fibres, to the inter-auricular septum, and the right and left auricles. During the ventricular systole the central fibro-cartilage, and with it the aortic vestibule and all the adjacent parts, are drawn downwards and to the

Fig. 51.



Showing the muscular fibres unravelled of the left and right ventricles. B, Fibres from the left and right ventricles going to the central fibro-cartilage of the heart, and forming a portion of the septum.

left towards the apex by the contraction of the ventricular fibres inserted into the tendinous ring and especially into the central fibro-cartilage, which thus becomes the focus and movable pivot of the heart, which binds together all those important parts and gives to them a common movement.

The setting of the orifice of the aorta is muscular anteriorly and to the left, and fibrous posteriorly and to the right. The muscular setting is made by the anterior half of the base of the left ventricle, and the fibrous setting by the anterior cusp of the mitral valve and its continuation towards the left auricle, and by the central fibro-cartilage. During the diastole the anterior cusp of the mitral valve divides the ventricle into two portions, each with its own aperture, an anterior or aortic portion, out of which the blood pours during the systole through the aortic orifice, and a posterior or mitral portion, into which the blood flows during the diastole through the mitral orifice.

There is one anterior, and there are two posterior and lateral aortic sinuses. The right or anterior coronary artery springs from the anterior sinus, and the left or posterior coronary artery from the left posterior sinus. The right posterior sinus is sometimes called the intercoronary sinus. Owing to the obliquity downwards, forwards, and to the right of the orifice of the aorta, the right posterior flap of the aortic valve is much lower in position than the other flaps. Thus the lower boundary of that flap was in two instances half an inch lower than the lower boundary of either of the other flaps. In another example, in which the aorta was far to the right, the lower edge of the right posterior cusp was only a quarter of an inch lower than that of the left posterior cusp, but it was half an inch lower than the lower edge of the anterior cusp.

The root of the aorta is buried in the centre of the heart, and is therefore encircled by all the cavities of the heart and the two other great vessels. The crescentic edge of the anterior sinus is attached throughout to the central fibro-cartilage which forms the summit of the interventricular septum. The anterior sinus is covered in front by the *conus arteriosus* and, higher up, on the right side, to a varying extent, by the auricular portion of the right auricle, and on the left side by the pulmonary artery.

The left and right halves respectively of the right and left posterior flaps of the aortic valve are attached at their junction, and along their lower border to the anterior cusp of the mitral valve, and to the aponeurosis that is continuous with that cusp. At this situation the two posterior sinuses of the aorta are in front of the left auricle. (Figs. 49 and 50.)

The left half of the left posterior sinus is attached at its root to the muscular base of the left ventricle, and is covered, going from right to left, first by the auricular portion of the right auricle, and then by the inner or right wall of the pulmonary artery. The junction of the anterior to the left posterior flap of the aortic valve is usually a little in front of the junction of the posterior and the left anterior flaps of the pulmonary artery, so that a pin thrust through that artery at the junction of the flaps in question into the aorta, appears about the tenth of an inch behind the junction of those aortic flaps; but in one instance the pin, thus inserted, pierced through the junction of the aortic flaps as well as through that of the pulmonic flaps. The left or posterior coronary artery at its origin is, in one of my preparations, ·25 inch from the left edge of the left posterior cusp, and ·4 inch from its right edge, and I believe it will be found that this represents the usual position of the origin of the artery.

The relations of the right posterior sinus of the aorta are of remarkable extent and importance. The centre and right side of the root of that sinus is firmly attached to or incorporated with the central fibro-cartilage and fibrous septum of the heart that crown the interventricular

septum. To the left of this attachment to the fibro-cartilage, the right aortic sinus is united, as we have just seen, to the anterior cusp of the mitral valve, and it is seated in front of the left auricle. To the right and in front of this attachment, it is closely connected with the inner or left angle of the tricuspid valve. The right wall of the right posterior sinus, as it advances to join the right edge of the anterior sinus, is covered first by the inner or left wall of the right auricle, and finally by the inner or posterior wall of the arterial cone of the right ventricle.

This right aortic sinus is thus closely connected with every important part of the heart, except the pulmonary artery. The right and left ventricle, the right and left auricle, the mitral and tricuspid valves are all of them attached to or in contact with it: and the central fibro-cartilage of the heart, as we have seen, with which the base of this sinus is incorporated acts as a tie that binds together the allied movements of those parts.[1] The descending vena cava also comes into contact with the upper portion of this sinus.

Mr. Thurnam brought into notice, thirty-three years ago, the extensive and important bearings of the sinuses of the aorta, in especial relation to aneurism of those parts.

It is customary for authors on anatomy, following the original error of the great Valsalva, unfortunately repeated by Mr. Thurnam, and more recently by that great anatomist, Henle, to describe the aortic sinuses as being two of them anterior, and one posterior. I have examined the heart *in situ* in many bodies, with regard to this point, and I have always found those sinuses and the corresponding flaps of the aortic valve in the position I have described, one being anterior, and two posterior. A little consideration as to the known relation of these sinuses to other parts, the position of which is well ascertained and admitted, will show that two of these sinuses are posterior and lateral, and that only one of them is anterior.

The right and left posterior flaps of the aortic valve are attached in about an equal degree to the anterior mitral cusp, as is shown in drawings and many hearts now around me, and in Dr. John Reid's figure.[2] The anterior cusp of the mitral valve is on a level with the posterior wall of the root of the aorta, and it is therefore impossible that either of the aortic sinuses that are attached to that flap can be situated at the anterior aspect of the aorta; they must, indeed, both be posterior in position. Again, while the right or anterior coronary artery arises from the anterior aortic sinus, the left or posterior coronary artery arises from the left posterior sinus; and while the right artery advances to the right of the pulmonary artery, the left artery passes to the left behind the pulmonary artery. Further, the origin of the left coronary artery is nearer to the left or anterior and lateral edge than to the right or posterior edge of the left posterior sinus. I might adduce other points in illustration of what I have advanced, but these facts, which speak for themselves, are sufficient.[1]

[1] See Fig. 50.

[2] "Cyclopædia of Anatomy," vol. i. p. 588. See also Figs. 47, 48, and 49.

[1] Valsalva's original drawing (V. Opera, tab. ii. fig. 1; see Fig. A), in which the anterior and left posterior sinuses with their respective coronary arteries are represented in front of the root of the aorta, gives not a front but a side view of the aortic arch. The artery from which this drawing was taken shows the cut end of the vessel, and has evidently been removed from the body and placed upon its right side. The effect of this position would be to place the anterior and left posterior sinuses, each with its coronary artery, on the same anterior plane. Fig. B is a reduced copy of a similar drawing of the arch of the aorta after its removal from the body, given by Lower (Tractatus de Corde, tab. i. fig. 4) in which the two coronary arteries, as in Valsalva's drawing, spring from the front of the root of the aorta.

Nearly all the drawings of the root of the aorta that have been taken from the actual body, the artery being *in situ* (reduced copies of several of which drawings are given below), represent the sinuses in the position that I have described, two of them being posterior in situation and one anterior, and the right posterior sinus being the lowest of the three sinuses. I find it thus in Tiedemann's "Tabula Arteriarum," plate xix. (fig. E); John Bell's "Anatomy," vol. ii. p. 283 (fig. F); Charles Bell's Engravings of the Arteries, tab. ii. (fig. G); Mr. Quain's "Anatomy of the Arteries," anterior view, fig. 3, and posterior view, fig. 4, plate xlviii. (figs. H I); Pirogoff's "Anatomia Topographica," in eleven different views (figs. K L M N); and Braun's "Topographisch-Anatomisch Atlas" (figs. O P). Henle, in a much reduced figure of the aorta *in situ*, represents one anterior and two posterior sinuses (fig. Q), but he gives a series of drawings of the heart and aorta after their removal from the body (one of which I have given on a reduced scale, fig. D), in all of which the sinuses are represented and described as being two anterior and one posterior.

Anatomists, including Morgagni and Senac in former times, and, as I have said above, the respected names of Thurnam and Henle in our own day, have as a rule described two of the sinuses of Valsalva and their corresponding coronary arteries as being anterior, and one of them, or that which is destitute of a coronary artery, as being posterior.

On the other hand, Vesalius and P. Syl-

The error has, I believe, arisen and been perpetuated from the custom of examining these sinuses, not when the heart is *in situ*, but after it has been removed from the body. If the right ventricle with its arterial cone, and the ventricular septum are carefully removed without disturbing the position of the heart, and

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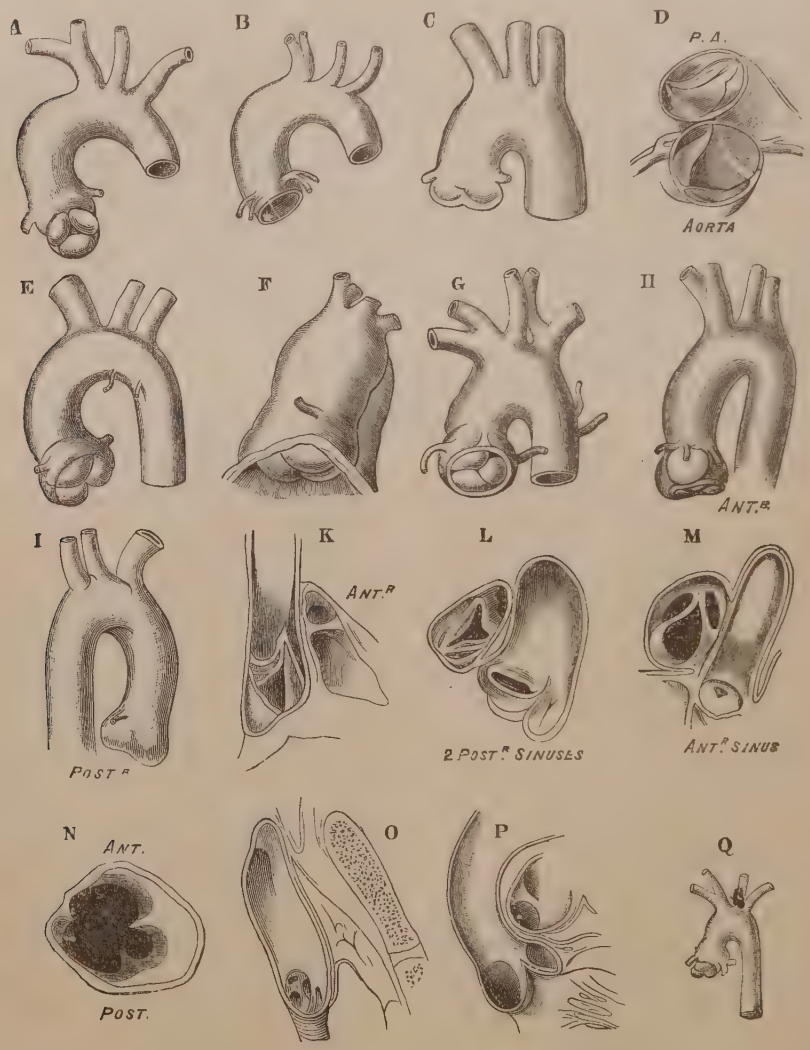
vius described the left coronary artery as arising from behind the posterior valve. Some authors give contradictory descriptions of the origin of the coronary arteries. Thus, Winslow in one passage says that there are two coronary arteries, "one of which is situated anteriorly, the other posteriorly" (vol. ii. p. 3); while elsewhere (p. 221) he says that "one of the vessels lies towards the right hand, the other towards the left, of the anterior third part of the circumference of the aorta." Portal ("Anatomie Medicale," vol. iii. p. 152) says



that the left coronary artery arises from the left posterior portion of the aorta; but elsewhere (p. 51) he states that two of the valves are anterior and lateral and the third is posterior, and that the right and left coronary arteries are situated above the two anterior valves.

The accurate Haller, "Elementa Physiologiæ," iii. 345, speaking of the aortic valve, says: "Situs alequantum differt, duæ enim superiori loco ponuntur, altera anterior, posterior altera; tertia inferior est. Earum eæ, quæ superiori loco ponuntur, sodales habent arterias coronarias, inferior nullum aortæ ramum vicinum habet." Here that great anatomist has given a correct description of

without injuring the anterior wall of the aorta at its origin, the true position of the aortic sinuses and of the flaps of the aortic valve may be readily observed.

The right and left posterior aortic sinuses advance forwards on either side, and finally curve gently inwards and forwards to complete the circle of the aorta by uniting at either end with the anterior sinus. The anterior portion of the left posterior sinus is concealed by the pulmonary artery, while the anterior portion of the right posterior sinus is readily exposed by pressing aside the auricular appendix. It is rather difficult to say which of the two posterior sinuses comes forward to the greater extent at their points of attachment to the anterior sinus; I think, however, that the right posterior sinus, which usually goes by the name of the posterior sinus, comes forward to a greater extent than the left posterior sinus, which usually goes by the name of the left anterior sinus. (Note 46.)

The Position of the Mitral Valve.—In seven instances the size and position of the mitral valve are given, and in three of them accurate details of its structure are represented. These points are further illustrated by preparations and dissections. (Note 46.)

The setting of the mitral orifice is muscular in its two posterior thirds, and fibrous in its anterior third. In these respects the mitral and aortic orifices balance each other. The setting of the mitral orifice is muscular behind, while that of the aortic orifice is muscular in front, the two openings being separated by the interposed anterior flap of the mitral valve and its short fibrous continuation to the two posterior aortic sinuses, and by the central fibro-cartilage of the heart. When the heart is boiled for a sufficient length of time this interposed fibrous partition softens and separates from its attachments, and the aortic and mitral apertures are thrown into one large irregular opening (see Fig. 52). The base of the ventricles then presents not four but three great apertures, the tricuspid, the pulmonic, and the mitral-aortic.

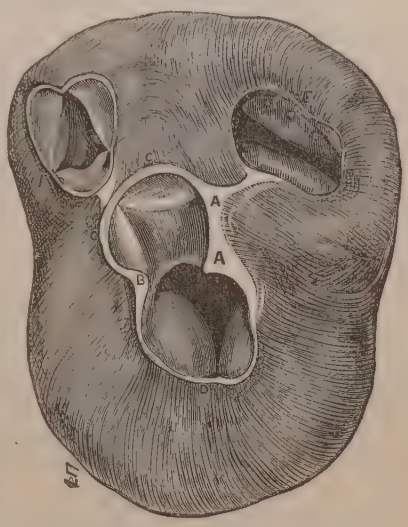
The apparatus of the mitral valve occupies the whole of the posterior part of the left ventricle, and when its anterior walls are removed, the whole of this apparatus is brought into view.

the situation of the flaps of the aortic valve and of the origin of the coronary arteries.

In our own day, Pirogoff and Mr. Heath describe the sinuses as being one of them anterior and two of them posterior. Bourgery (fig. c) curiously figures the coronary arteries and their sinuses as being both anterior; but he describes the anterior coronary artery as arising from the anterior sinus, and the posterior coronary artery from the posterior sinus.

The anterior cusp or flap of the mitral valve is alone visible in one of the three drawings giving the anatomical details of the valve, while in the two others the lower border of the posterior cusp is likewise brought into view.

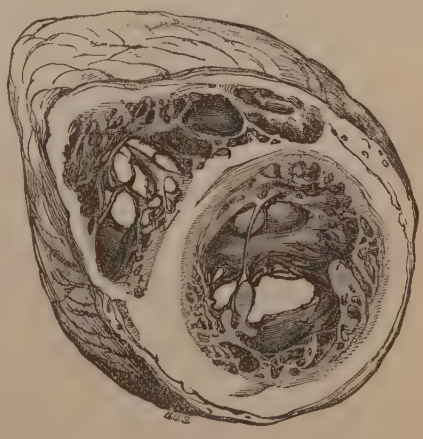
Fig. 52.



Calf's heart boiled, showing the aortic (C) and mitral (D) orifices thrown into one by the removal of the mitral valve, the lower A being the central fibro-cartilage, E the tricuspid orifice, and F the orifice of the pulmonary artery.

The whole apparatus of the valve takes an oblique direction from right to left and downwards. The right end or base of the apparatus of the valve corresponds with

Fig. 53.



Showing the mitral orifice, the anterior flap of the mitral valve, and the right and left posterior flaps of the aortic valve. Diastole of the ventricles.

the junction of the left auricle with the left ventricle, and its left end corresponds with the interior of the apex of the left

ventricle. The apparatus of the valve thus forms a long triangle, its base being at the base of the ventricle, its apex at the apex of the ventricle, its upper side being slightly curved upwards or outwards,

Fig. 54.

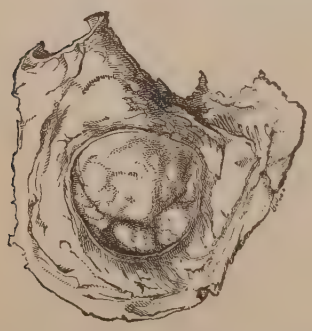
Systole of the left ventricle.

and its lower side being slightly bent inwards or upwards at its middle. The flaps, the tendinous cords, and the papillary muscles, which are connected by the cords to the flaps, form the three component parts of the valve. (Figs. 53, 56, 57, 58, and "Medical Anatomy," Plate VI.)

The convex base of the anterior flap of the mitral valve is attached on the one hand to the junction of the left ventricle to the left auricle, and on the other to the roots of the right and left posterior flaps of the aortic valve. This attachment of the mitral to the aortic valve is effected through the fibrous structure that extends from the base of one valve to the base of the other, and by the central fibro-cartilage of the heart, which forms a triple bond of connection that ties the mitral, the aortic, and the tricuspid valves to each other. (Fig. 48.)

When the mitral valve is shut, the anterior flap of the valve presents a convex edge, shaped like a horseshoe, which falls back upon and fits like a lid into the posterior flap of the valve, which flap, being crescentic in shape, presents a concave edge.[1] Each flap adapts itself to the other by a notched lip, made up of small hemispherical eminences. The eminences of one lip fill up the notches of the other lip. These eminences, thus seen on the auricular surface of the valve, are cells

Fig. 55.



Mitral valve shut; auricular surface; anterior or convex and posterior or crescentic flaps; ventricular systole.

Fig. 56.

Mitral valve shut; ventricular surface; anterior flap, with tendinous cords and papillary muscles; two posterior flaps of aortic valve; ventricular systole.

when seen on its ventricular surface, and as these cells are distended with blood when the ventricle contracts, and are exactly maintained in their places by the tendinous cords and papillary muscles, the distended cells or eminences at the opposite lips of the valve adapt themselves to and press against each other during the systole, so as to shut the valve. (Figs. 48, 55, 56, 58.)

The anterior flap is simple, and when closed is shaped like a three-quarters moon. The posterior flap is compound, and when closed is shaped like a quarter or crescent-shaped moon. The compound posterior flap is usually made up of one central and two lateral sub-segments, the latter being sometimes subdivided. These sub-segments adapt themselves so to each other, that the concavity of the crescentic border of the posterior compound-flap is preserved entire; for it would have been impossible, by means of one simple fold of membrane, to fill up without a break the whole of the crescentic border.

I need scarcely give a description of the arrangement of the tendinous cords in relation to the flaps of the valve, and of

[1] Figs. 48, 55.

the papillary muscles in relation to the cords and the flaps. It will be sufficient if I here say that they are so arranged that when the muscular walls of the ventricle contract, the papillary muscles, which are really semi-detached portions of those walls, also contract with equal steps; that as the walls shorten so as to approximate the base and the apex by a double movement to each other, the papillary muscles shorten to an exactly parallel degree; and that thus while they hold the flaps of the valve, through the medium of the cords, in apposition, they steadily draw the whole valve towards the apex, and the apex towards the valve, to exactly the same extent that the base and apex of the ventricle are drawn towards each other. The mechanical arrangements are complicated, for there are many parts to be adjusted to each other; but the principle on which those parts are adjusted to each other is simple, for it is by one single contraction of the whole single muscle of the left ventricle, made up in its component parts of walls, columns, and papillary muscles, that the base of the ventricle (including the mitral aperture and valve and the aortic aperture and valve) and the apex of the ventricle are approximated steadily to each other during the systole.

When the convex anterior flap of the mitral valve falls back upon and fills up

Fig. 57.

Mitral valve shut; posterior flap, with tendinous cords and papillary muscles.

Fig. 58.

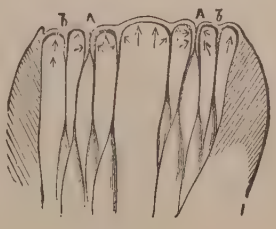


Diagram of the shut mitral valve, with the anterior cusp A A in close contact with the posterior cusp (*b*, *b*). The tendinous cords and papillary muscles are shown, the direction of the current and pressure of the blood being indicated by arrows.

the concave posterior flap of the valve, the anterior flap and its membranous continuation to the left and right posterior aortic flaps form a smooth scooped channel or hollow, along which the blood flows noiselessly from the ventricle into the aorta during the systole. (Fig. 53.)

The mitral orifice extends downwards, with an inclination to the left, immediately behind and below the aortic orifice; and, like that orifice, it looks towards the apex of the left ventricle, or to the left, downwards and slightly forwards. The line of direction of the mitral orifice, viewed from the front, is therefore from above downwards, with a slight obliquity from left to right. The upper and left boundary of the mitral orifice is about half an inch above the level of the lower edge of the right posterior flap of the aortic valve. The lower border of the mitral orifice is about three-quarters of an inch below the lower border of the aortic orifice. The upper or left edge of the mitral orifice is not so far to the left, while its lower or right edge is about as far to the right, as are the left and right edges respectively of the aortic orifice. The mitral orifice is situated deep behind the sternum, a little below the middle of that bone. Its upper or left boundary, in four instances, was on a level with the third cartilage, just within the left edge of the sternum; and its lower or right boundary was on a level with the fourth cartilage, behind a line drawn down the middle of the sternum. This is probably higher than the average position of the mitral orifice after death. In one other case the top of the mitral orifice was on a level with the lower border of the second space, its situation otherwise corresponding to that in the cases just described. In two other instances, the mitral orifice was comparatively low and was situated unusually to the right, its upper border being on a level with the lower edge of the third cartilage or upper border of the fourth space, behind the middle line of the sternum, and its lower or right border being on a level with the lower portion of the fourth space, or the top of the fifth cartilage behind the right edge of the sternum. As a rule, the mitral orifice occupied a space behind the left half of the sternum, extending downwards for more than one inch below the middle of the bone; but in occasional

cases it was present behind the right half of the bone.

The tendinous cords and papillary muscles of the mitral valve, as they extended to the left with an inclination downwards, retained, as a rule, their situation behind the space or cartilage that was on a level with their starting-point from the valve.

Thus in the four instances in which the upper rim of the orifice was on a level with the third cartilage, the upper or left cords lay behind the third left cartilage, and the upper or left papillary muscle behind the third space; and in the same instances the lower rim of the orifice was in two of them on a level with the third space, and in two of them on a level with the fourth cartilage; and in these two sets of cases the lower or right cords and papillary muscle lay respectively behind the third space and the fourth cartilage, with a final dip to the space or cartilage below.

In the other cases in which the position of the upper and lower edges of the mitral orifice were higher or lower in relation to the spaces and cartilages than in those just quoted, the upper and lower cords and muscles retained their bearing throughout in relation to the space or cartilage on the level of which they started, until they also usually made a final dip so as to occupy a relatively lower position.

In two of the instances there was a space of half an inch between the right and left papillary muscles, the width of the interior of the ventricle being an inch and a half; and in the other instance, in which the systole of the ventricle was more pronounced, the space between the muscles was the fifth of an inch, the width of the cavity being a little over an inch (1·2 inch).

In one instance, in which the heart and all its parts lay unduly to the right, and in which the flaps, cords and muscles of the valve took a very oblique direction downwards, the right papillary muscle was situated behind the left border of the sternum and the sternal half of the fifth cartilage, and the left papillary muscle crossed the third cartilage and space midway between the sternum and the junction of the cartilages to their ribs.

This instance was throughout exceptional in the position of the heart and all its parts, and the great vessels; but the other instances offer fair average examples of the position of the mitral valve. I need not, therefore, further analyze additional cases. It is sufficient to bear in mind that when the origin of the pulmonary artery is high or low in position, the aortic and mitral valves are also correspondingly high or low in position; and that when the ascending aorta and the origin of the pulmonary artery are far to the right or far to the left of their usual situation, the aortic and mitral valves are also correspondingly far to the right or far to the left of their usual situation.

The Tricuspid Valve.—The apparatus of the mitral valve occupies the whole of the posterior part of the left ventricle, but the apparatus of the tricuspid valve fills up the whole body of the right ventricle, the narrowing *conus arteriosus* being the only portion of the ventricle unoccupied by it. (Note 46.)

The reason for this diffusion of the apparatus of the tricuspid valve and this concentration of that of the mitral valve is obvious. It depends on the form of the two ventricles and the relation to each other of their apertures of ingress and egress.

The left ventricle is the central cavity of the heart, and is flask-shaped; and its walls on a transverse section are shaped like a ring, and surround a circular space, the mitral valve being behind (see Fig. 49). The right ventricle is applied upon the anterior and inferior walls of the left ventricle, which project into the cavity of the right ventricle and form its inner or posterior wall. The right ventricle on a transverse section is crescent-shaped, its inner wall being convex, while the inner aspect of its outer wall is concave or angular, for it presents at its lower border and outer aspect a projecting angle. The whole cavity of the right ventricle looked at in front is triangular in form, the base of the triangle resting on the central tendon of the diaphragm, its apex pointing to the top of the pulmonary artery, its right side being formed by the junction of the right auricle to the right ventricle and by the tricuspid orifice, and its left side by the septum between the ventricles.

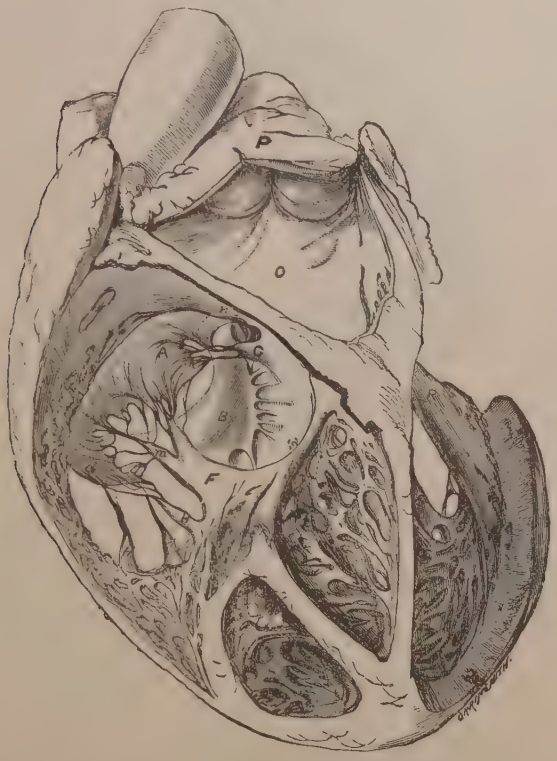
The three cusps of the tricuspid valve are visible when the cavity is opened, the anterior and inferior flaps being completely exposed while the posterior flap is partially concealed (Figs. 59, 60, 61).

The whole apparatus of the tricuspid valve, like that of the mitral valve, takes a direction from right to left; but while the apparatus of the mitral valve concentrates itself as it recedes from the flaps, the papillary muscles pointing towards the apex, and the whole structure forming a long triangle, the apparatus of the tricuspid valve spreads itself out as it recedes from the flaps, like the rays of a fan.

The anterior flap gives attachment at its upper edge to a group of cords which converge upon the small superior papillary muscle, which is incorporated with the posterior wall of the cavity at the lower portion of the arterial cone. The cords from the lower edge of the anterior flap converge upon the anterior papillary muscle, which muscle also sends a radiating series

of cords that attach themselves to the upper and anterior edge of the lower flap of the valve. The anterior papillary muscle is not immediately connected either with the front or the back wall of the ventricle, but is attached intermediately to both of them by fleshy columns. A strong and rather long column curves

Fig. 59.



Showing the tricuspid valve open, during the complete dilatation (diastole) of the right ventricle.

Figs. 59, 60, and 61 are views of the interior of the right ventricle and of a portion of the left ventricle: A, anterior flap; B, posterior flap; C, inferior flap; and *d*, one of the long sub-segments of the inferior flap of the tricuspid valve. F, anterior papillary muscle; G, superior papillary muscle; H H, inferior papillary muscles; *n*, sub-segment occupying the angle between the anterior and posterior flaps of the valve; *o*, *conus arteriosus*; P, pulmonary artery; R, upper or left papillary muscle, and S, lower or right papillary muscle belonging to the left ventricle and mitral valve.

backwards to be attached by outspreading roots to the posterior wall of the ventricle near the septum; while an interlacement of shorter and thinner columns advances forward and to the left, extending from the base of the anterior papillary muscle to the anterior wall of the ventricle, also near the septum (Fig. 59). Thus the roots of the anterior papillary muscle spread both backwards and forwards, the base of the muscle being, however, nearer to the front than the back of the ventricle. By this beautiful arrangement a purchase is given for this muscle to act upon the centre of the valve from the middle of the cavity.

The inferior flap of the valve is not formed, like the anterior flap, of one sheet of membrane, but is a compound flap, which is subdivided into four or five sub-segments, two of which are longer than the rest, which, by meeting together and adapting themselves to each other, fill up the large rounded space of the tricuspid orifice, at its inferior portion. The cords from these sub-segments converge upon a series of papillary muscles that are conveniently situated around the lower portion of the ventricle, some, or one, being seated in front, some below, and some behind. The inferior papillary muscles are attached, like the anterior papillary muscle, not immediately to the walls of the ventricle, but intermediately by interlacing fleshy columns. The posterior papillary muscles of this group, which are thus connected with the inferior flap of the tricuspid valve, are immediately attached to the inner or convex wall of the ventricle.

The posterior flap is attached behind by a series of radiating cords to the inner walls of the ventricle, sometimes by

means of small papillary muscles, sometimes by the immediate insertion of the cords into the walls.

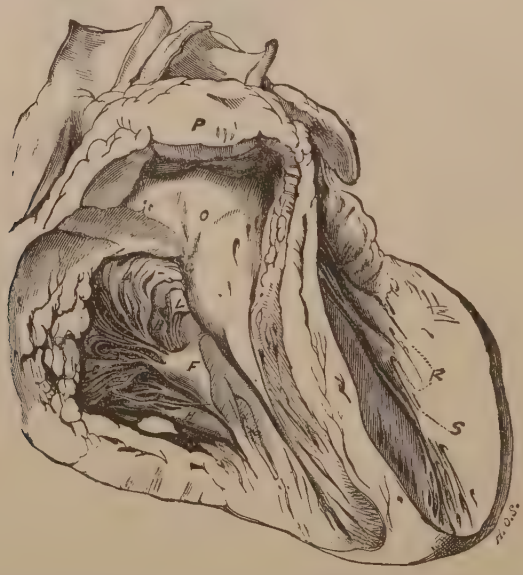
The upper portion of the tricuspid orifice is narrow and angular, while its lower portion is wide and circular; and thus,

Fig. 60.

Showing the tricuspid valve shut during the early period of the contraction of the right ventricle.

therefore, the simple anterior and posterior flaps, with the intervention of one anterior and one superior sub-segment, fill up the upper and narrow part of the orifice; while the inferior and compound sub-segments adapt themselves to the

Fig. 61.



Showing the tricuspid valve shut during the period of the complete contraction of the right ventricle.

large and rounded inferior portion of the orifice. (See fig. 48, p. 385.)

The whole of these segments of the valve meet together at the centre of the orifice, and hence arises the necessity for an array of papillary muscles, the points of which converge towards the centre of the valve, and that are attached at their

roots by fleshy columns that connect them with both the outer and the inner walls of the ventricle.

The tricuspid orifice is situated behind the lower portion of the sternum and in front of the mitral orifice and the left ventricle. The direction of the tricuspid orifice is from above downwards with a slight inclination from left to right. The upper boundary of the tricuspid orifice is immediately below the orifice of the aorta, and in front of the mitral valve.

The four great orifices of the heart—the pulmonic, the aortic, the mitral, and the tricuspid—are situated in that order, one above another, the pulmonic orifice being the highest and the tricuspid the lowest. The lower portion of each of the first three orifices is lower than the upper portion of the orifice below it. Thus the pulmonic orifice, when looked at in front, covers the upper part of the orifice of the aorta on its left side; the lower border of the aortic orifice is lower than the upper border of the mitral orifice; and in like manner, the lower two-thirds, or three-fifths, of the mitral orifice lie behind the upper half of the tricuspid orifice, the lower half of which is below the level of the lower edge of the mitral valve.

The posterior aspect of the tricuspid orifice is attached to the anterior wall of the left ventricle, not on a level with the mitral orifice, but about half an inch nearer to the apex. The wall to which it is thus attached is convex, and the posterior surface of the tricuspid valve where it fits upon the left ventricle is therefore concave. The shape of the tricuspid orifice is, in consequence, angular above, concave behind, convex in front, and rounded and broad below. The tricuspid orifice thus maintains the crescentic form of the cavity of the right ventricle when a cross section is made through its walls.

In five of the cases, the upper and left boundary of the tricuspid valve was situated about the third of an inch to the left, and its lower and right boundary about a third of an inch to the right of a line drawn down the middle of the sternum. In two instances the lower and right boundary of the tricuspid valve extended to the right of the right edge of the sternum.

The right transverse or auriculo-ventricular furrow which corresponds with the right edge of the right ventricle, as I have already remarked, is situated about half an inch to the right of the right edge of the tricuspid valve, and when therefore we know the position of the furrow, we can infer the position of the right edge of the valve. As we already seen (page 381) the transverse furrow was situated to the right of the right edge of the sternum in nearly three-fourths of the cases (36 in 51), at that edge in three of them, and to the left of that edge, behind the right half of the sternum, in one-fourth of them (12 in 51); and it extended in one instance for an inch and a third to the right of the right edge, and was situated in five instances behind the middle line of the sternum. The transverse furrow occupied every variety of position between these two extreme points. We may therefore infer that the right border of the tricuspid valve occupied every range of position between a line three-quarters of an inch to the right of the right edge of the lower portion of the sternum, and a line half an inch to the left of its middle line; the average situation of the right border of the valve being behind the right edge of the sternum.

In like manner we can infer approximately the position of the lower border of the tricuspid valve if we know the position of the lower boundary of the right ventricle. We have already seen that the lower boundary of the right ventricle varied in situation from an inch and a half below, to an inch and a half (1·4 inch) above, the lower end of the sternum. The lower border of the tricuspid valve is from half an inch to nearly an inch above the level of the lower boundary of the right ventricle, and we may therefore infer that the lower border of the valve varies in position from a point nearly two inches above, to a point three-quarters of an inch below the lower end of the sternum.

The top of the tricuspid orifice and valve was situated on a level with the third cartilage in three cases, with the third space in one, and with the fourth cartilage in two; its lower edge being in those cases on a level respectively with the fourth cartilage, the fourth space, and the fifth cartilage. In each of those cases the upper edge of the tricuspid orifice and valve was lower, and its lower edge was much lower than the corresponding edges of the mitral orifice and valve.

The tendinous cords and papillary muscles of the tricuspid valve, as they radiated to the left, slightly upwards, downwards and outwards, retained, as a rule, their situation behind the space or cartilage that was on a level with their starting-point from the valve. Thus, the inferior cords and muscles maintained their relative position throughout, behind the fourth space or fifth cartilage, which was on a level with the lower edge of the tricuspid valve, while the anterior group of cords and the anterior papillary muscles lay behind the fourth cartilage or the fourth space. The upper group of cords retained its relation to the third cartilage or space, or the fourth cartilage, on a level with which the upper edge of the valve was situated, but the superior papillary muscle radiated upwards to a somewhat

higher relative position than that from which it started, so that, as, for instance, in two cases, the top of the valve being on a level with the third or fourth cartilage, the superior papillary muscle was behind, respectively, the second or third space.

In the remaining instance, that in which the heart was placed to an unusual extent to the right, the flaps of the valve were situated behind and to the right of the right portion of the sternum, the valve extending from the level of the upper edge of the fourth cartilage to that of the lower edge of the fifth. The tendinous cords, and the papillary muscles took an oblique direction downwards, and they were seated almost entirely behind the right half of the sternum.

The position of the tricuspid valve corresponds with the position of the right ventricle, the valve occupying about the lower two-thirds of the cavity.

The Relation of the Lungs to the Heart.

The extent to which the lungs covered the heart varied much in different examples. In two instances the heart was almost concealed by the lungs, the edges of which were separated over the lower portion of the right ventricle by a mere chink, which widened out to three-quarters of an inch at the inferior border of the heart. In other instances the lungs had receded from before the heart to such an extent that almost the whole organ and the great vessels were exposed to view, though in no instance were they entirely uncovered.

The space where the heart comes to the surface, which is bounded above and at the sides by the lungs, and below by the liver and stomach, was sometimes triangular in shape (in 10 of 60 cases), but was usually four-sided (50 in 60).

The superficial "cardiac space" was triangular in shape in the two instances just noticed in which that space was very small, the width at the lower boundary of the heart being three-quarters of an inch; and in an instance of an opposite kind in which the base of the triangle, which always corresponded with the lower boundary of the heart, was four and a half inches wide. In this instance the lower boundary or base of the superficial space of the heart was wider than in any other. The base of the triangular superficial cardiac space presented every intermediate gradation of breadth between the extreme instances just noticed. This triangular shape is favorable to the covering of every part of the heart with lung except the right ventricle, for, while the anterior wall of the right ventricle was laid bare to a greater or less extent in these cases, as it was in every case under observation, in but one of them was the apex of the heart exposed, while in only two of them the right auricle, the aorta and pulmonary artery were also somewhat uncovered.

The superficial cardiac space was in all these cases actually triangular in shape, the lower limit or base of the triangle being the lower border of the heart. If, however, the lower boundary of the heart had occupied a lower position in relation to the adjoining margins and the lower boundaries of the lungs, then that space would have been four-sided in shape in the majority of these instances (6 in 10); for in them the inner border of the left lung, after it had left the heart, curved in a downward direction. If, therefore, the heart had not shrunk upwards in these instances, the superficial cardiac space would, like the other cases, have been four-sided in shape.

When, as is usually the case, the superficial space of the heart is bounded by four sides,[1] the heart, which moulds for itself a place between and within the lungs, comes forward to the surface at that part where the organ is massive and its walls are powerful. The inner edge of the right lung descends in a straight line behind the sternum, but the edge of the left lung leaves the right lung, and deviates to the left at a variable point. This deviating edge of the upper lobe of the left lung, which is suspended like a curtain above the upper margin of the superficial space of the heart, describes a double curve, first convex, where it leaves the right lung, and then concave, where it begins to dip downwards to form the outer edge of the space. It then, as I have already observed, again tends to curve inwards, and to form the tongue of lung that enfolds the apex of the heart. The breadth of the cardiac space, measured along its base at the lower boundary of the heart, varied from an inch and a half to four inches and a third (4·3 inch). The breadth of the lower boundary of the superficial cardiac space varied in three-fourths of the cases (37 in 50) from two to four inches; it was less than two inches in one-fifth of them (9 in 50), two-thirds of these being youthful, and it was above four inches in four of them.

The superficial cardiac space was, as a rule, narrower at its upper than at its lower boundary (in 35 of 50 cases); but

[1] I have grouped the remainder of the cases, amounting to fifty, under the common heading of those in which the superficial space of the heart was bounded by four sides, but in seven of these cases the space was almost triangular in shape, and in a few other instances irregularity in outline modified the typical four-sided form of the space.

sometimes this was reversed, the space being narrower below than above (in 10 of 50 cases). In a few instances (5) the space was of equal breadth above and below.

The lower boundary of the superficial cardiac space measured less than three inches in all but one of those instances in which it was narrower than the upper boundary of that space.

The inner borders of the right and left lungs were in contact with each other behind the upper portion of the sternum so as to cover the great vessels in three-fifths of the cases under examination (in 35 of 59). In the remaining two-fifths of the cases (24 in 59) a space varying in width from the eighth of an inch to an inch and a half was interposed between the inner borders of the right and left lungs at the upper part of the front of the chest. In one-third of these instances (7 in 24) the space between the edges of the lungs was less than the third of an inch, so that, practically, these cases may be added to those in which the edges of the lung were in contact, which brings up their proportion to three-fourths of the total number of cases observed (42 in 59).

The point of separation and divergence of the left and right lungs in these cases, including those in which the lungs were nearly in contact, varied from the level of the first intercostal space to that of the fifth cartilage. In three-fourths of the cases this point of separation varied in position from the level of the second space to that of the fourth cartilage.

In the seventeen cases in which the lungs were separated from each other over the great vessels to an extent ranging from almost half an inch to an inch and a half, the position of the point of divergence of the right and left lungs varied from the level of the first cartilages to that of the third, the level of the second cartilages and second spaces being the more usual situation of the point of divergence in this group of cases.

There was much variation in the relative size of the right and left lungs. The two lungs were about of equal size in more than one-fourth of the cases (17 in 59), the right lung was larger than the left in nearly one-half of them (27 in 59), and the left lung was larger than the right in one-fourth of them (15 in 59).

Although the right lung was so often larger than the left, yet the base of the left lung was lower at the side than that of the right lung three times more often than the reverse, the bases of the two lungs being on the same level in one-fourth of the cases (14 in 57).

When the right and left lungs met together behind the upper half of the sternum to form a covering over the great vessels, the margin of the right lung extended to the left of a line drawn down the middle of the bone more often than that of the left lung extended to the right of that line in the proportion of 35 to 15, while in nine instances the edges of the two lungs lay in contact behind the middle line of the sternum.

Below the point of separation of the two lungs, while the left lung deviated, as we have seen, extensively to the left, the right lung usually (in 42 of 60 cases) deviated at first very slightly and then more definitely to the right, so that at its lower anterior border the left inner margin of the right lung at the level of the lower boundary of the heart was usually situated to the right of the middle line of the sternum, the extent to which it did so varying from the tenth of an inch to an inch and three-quarters. In less than one-third of the cases (18 in 60) the left margin of the right lung, at or a little above, the level of the lower boundary of the heart, extended to the left of the middle line of the sternum.

When the superficial cardiac space was small, measuring less than two inches across, the inner margin of the right lung extended to the left of the centre of the chest in three-fifths of the cases (10 in 18). When, however, the space was of medium size (2 to 3 inches wide), the right lung passed to the left of the middle line in less than one-fifth of the cases (4 in 21); and when the space was large (above 3 inches wide), the right lung extended thus to the left of the middle line over the superficial cardiac space in only one-tenth of the cases (2 in 21).

The upper boundary of the superficial cardiac space, which is an important landmark to the clinical worker, is formed by the lower anterior border of the upper lobe of the left lung after it deviates from its point of separation from the right lung. I have already described the direction and nature of this curve.

The margin of lung, which thus forms the upper boundary of the superficial cardiac space, lay immediately behind one or other of the left costal cartilages or their spaces, and it generally took the downward direction of the cartilage behind which it lay, or was somewhat more inclined. It generally lay behind one cartilage or space, from the point at which it left the sternum to the point where it curved downwards to form the left border of the superficial cardiac space. It sometimes, however, took a more oblique direction downwards, and crossed from behind one cartilage to behind the next space below, and then, after crossing that space, it spent itself behind the next cartilage below. The upper boundary of the cardiac space varied in position from the level of the second left costal cartilage to that of the fifth, but it was most frequently

present behind the third or fourth cartilage or the fourth space, being thus situated in three-fifths of the cases (35 in 60).

In three of the cases the surface of the heart exposed below the lower edge of the left lung was a mere belt composed of the lower boundary of the right ventricle, this belt being from two inches to two inches and a half in diameter from side to side, and from a fifth to a little over one-half of an inch from above downwards. The heart had been lifted upwards behind the lungs by great distension of the stomach and intestines in these three cases, and the front of the cage of the chest had been also lifted upwards by the same agency, while the lungs had expanded downwards under the influence of the forward movement of the ribs.

Extent to which the Surface of the Heart is Exposed.

In every instance more or less of the right ventricle was uncovered. A very small portion of the right ventricle was exposed in the two instances in which there was a narrow longitudinal chink over the front of the heart, and in the three in which the exposed surface of the organ was a very narrow belt extending from right to left along the lower border of the ventricle.

In nearly one-half of the cases (25 in 60) the right ventricle was the only part of the heart that was exposed at the superficial cardiac space. In five other cases the apex of the heart was the only additional part brought into view by the lateral withdrawal of the lung. In almost one-half of the cases (25 in 60) the apex of the heart was in contact with the walls of the chest, the pericardium intervening; and in one-third of them (19 in 60) the higher portion of the left ventricle was also exposed to a greater or less extent. In only one instance was the whole of the narrow anterior portion of the left ventricle laid bare. In the rest of the cases, more or less of the upper portion of the left ventricle was covered by the edge of the left lung where it overlaps the front of the heart.

The right auricle was uncovered to a greater or less extent in one-fifth of the cases (11 in 59), and in all but three of these its auricular appendix was also apparent. In one instance the whole of the auricle was exposed. The tip of the auricle was just visible in eight additional cases.

The whole of the ascending portion of the aorta was exposed to view in nine instances, and it was visible on its right side in three, on its left side in four, and at its middle in one. Thus the aorta was more or less exposed in nearly one-fourth of the cases.

The whole of the pulmonary artery was laid bare in only one instance, but in eight other cases the right side of the vessel, and in five others the left side of the vessel, was respectively exposed. The arterial cone of the right ventricle was completely covered by the lungs in one-third of the cases (20 in 59). In certain cases (10) a very small portion of the cone was uncovered just below the point of separation of the right and left lungs. These cases may practically be added to those in which the cone was completely concealed, so that, with this reservation, it may be said that the cone was covered with lung in one-half of the cases (31 in 59). In several instances, only one-fourth of the arterial cone was exposed, while in one instance the whole of it was uncovered. Between these extreme cases there was every variety in the extent to which the cone was brought into view.

FRONT VIEW; DURING LIFE.

We have just seen that after death the healthy heart and great vessels, and the different parts composing them, present great variety in position; and that although perhaps in no two instances do those parts occupy precisely the same relative situation, yet in a considerable proportion of the cases, and within certain limits, they present a standard or average position.

During life in like manner the healthy heart and great vessels vary much in relative situation, yet those parts, within certain orderly limits, regulated and modified by the various demands of life, maintain a standard or average position.

It is evident that during life, when the heart is at work and in motion, sending blood to and receiving blood from the lungs and every part of the frame, the position of the heart and great vessels is different from what it is when observed in the dead body. A knowledge of the position of the heart and the great vessels during life, when in active motion, is essential to the clinical worker, and not merely that of the anatomy of the dead organ.

I shall here, therefore, endeavor to describe the average position of the heart and great vessels in the living frame, from the study of the situation of those parts after death and during life, and of the movements of the heart when in action, and when influenced by respiration.

When the exertions of the body are prolonged and powerful, the heart acts with corresponding power; it receives

and distributes more blood than when the body is at rest, and its size, and that of its great vessels, become enlarged. When, however, the body is in repose the heart's action is weakened; it receives and sends out less blood, and its size and that of its great vessels are diminished. The used power and the size of the heart, and the supply of blood to and from the organ, strictly balance the actual demands of the body, whether in action or at rest.

Under the like circumstances the lungs, answering to the demands on respiration, enlarge or lessen in size, and the volume of the cage of the chest is correspondingly larger or smaller, while the pitch of the diaphragm is lower or higher, so as directly to depress or elevate the heart. As the result, therefore, of these changes, thus induced by respiration, the heart, when it enlarges during exertion, is low and deep, and when it lessens during rest, is high and superficial.

In a corresponding manner, and for the same reasons, the heart is large, low, and deep in strong laboring men, while it is small, high, and shallow in weak youths of sedentary habits. In women and in children the heart is proportionally smaller and higher than in adult men; and in the scale of life, from infancy to old age, the heart tends proportionally to increase in size, and to become lower and deeper in position.

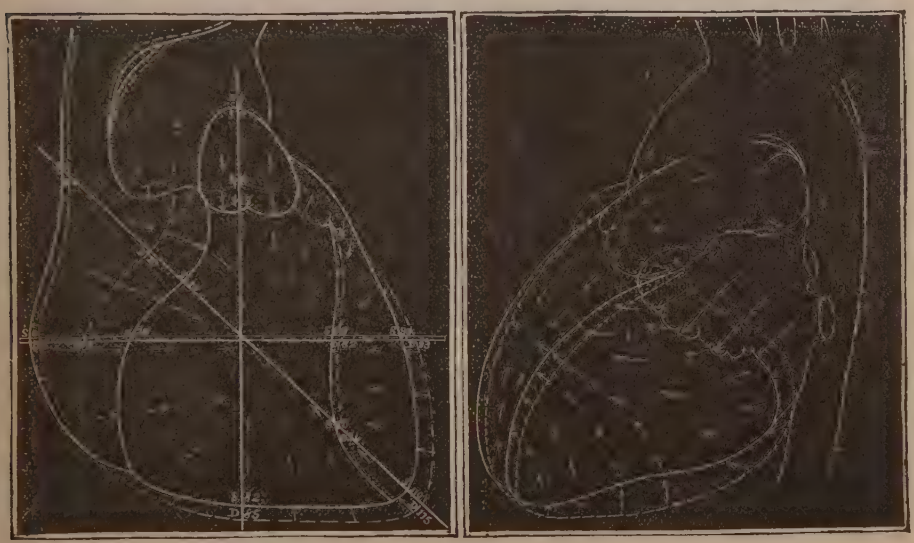
In order that we may have before us the movements of the heart and great vessels during the varied exercises of life, I shall, before describing the position of those parts in the living body, give a brief account of the action of the heart, of the currents of blood through the cavities of the heart, and of the movements of the heart caused by respiration.

Movements of the Heart. (See Figs. 62, 63, 68, 69.)

I have observed, with the valuable assistance of Dr. Broadbent, the movements of the heart in the dog and the donkey, when under the influence of chloroform; and from those observations, and the careful examination of the human heart in many subjects, I have constructed figures 62, 63, 68, and 69, representing the

Fig. 62.

Fig. 63.



Front view. Side view.

The *continuous* lines indicate the position of the outlines of the various parts of the heart during the *systole* of the ventricles; the *interrupted* lines indicate the position of the same parts during the *diastole* of the ventricles; the arrows point out the direction and extent of the movements of the walls of the heart during the *systole*. Fig. 62 shows the transverse, vertical, and oblique measurements in millimetres during the systole and the diastole.

heart in man in the opposite conditions of complete ventricular contraction and dilatation. In figures 62 and 63, the direction and extent of the movements of the walls during the ventricular systole are represented by arrows.

The appearance of the heart in motion is very striking. The ventricles, during their systole, contract from all sides upon their own centre and become wrinkled, and the arteries and veins on their surface are full and tortuous, while the auricles become purple, plump, and glistening. During the diastole, the aspect is re-

versed. The ventricles enlarge and become smooth, their superficial vessels almost disappearing, while the auricles shrink, and become pale and wrinkled.

The Systole of the Ventricles.—During the systole, the ventricles, when looked at in front, contract from all sides towards a given centre, which is situated on the right ventricle a little to the right of the septum, about midway between the origin of the pulmonary artery and the lower boundary of the ventricle, where it rests upon the diaphragm. The contraction of the right ventricle, owing to its position at the front of the heart, and its consequent complete exposure, is marked and vigorous. The whole right margin of the ventricle, at its juncture to the auricle, moves extensively from right to left; while its left margin, at the longitudinal furrow or septum between the ventricles, moves to a comparatively slight degree from left to right. At the same time the top of the ventricle, at the origin of the pulmonary artery, descends, while its lower border, where it rests on the diaphragm, ascends.

The point of rest towards which these various movements converge corresponds closely with the attachment of the anterior papillary muscle.

The right auricle and superior vena cava are distended, and the pulmonary artery is enlarged and lengthened simultaneously with the contraction of the ventricle. The auricle, which just before was wrinkled, becomes full; and its auricular portion and left edge move rapidly inwards, and to the left, so as to replace the ventricle. The movement of the auricular portion is remarkable. It suddenly enlarges and becomes purple, and its tip moves from the right to the left edge of the sternum, at the level of the third costal cartilage.

The vigorous contraction of the left ventricle is only visible at its apex and along its left border, since the rest of the cavity is hidden by the right ventricle. The apex has a revolving movement, upwards, forwards, and to the right. The left border of the ventricle, like the apex, moves forwards and to the right; but while the portion of the ventricle near the apex ascends, the portion near the base descends. The appendix of the left auricle, which during the diastole of the ventricle is scarcely visible, descends during the systole, and moves rapidly forwards and downwards, so as to replace the retreating ventricles, and to fill up the angle between them and the pulmonary artery.

When we remove the left ribs and look at the heart from the left side so as to obtain a profile view, the animal lying upon the back, we see that the whole left ventricle moves forwards during the systole, the posterior wall advancing much more than the anterior; and that the base of the ventricle descends, while the apex ascends, so that apex and base approximate. It is difficult to fix upon the precise zone of rest of the ventricular walls towards which the apex ascends and the base descends, but it is somewhere about the middle of the ventricle, nearer, perhaps, to the apex than the base. This region of stable equilibrium corresponds to a similar point of rest in the papillary muscles. Owing to this arrangement, the ventricles and their valves adjust themselves to each other during the ventricular contraction.

The left auricle, like the right, enlarges during the systole, and as the base of the ventricle then descends and advances, the ventricular attachment of the swollen auricle descends likewise, apparently, as it were, pushing the base of the ventricle before it.

When the left ventricle propels its contents into the aorta, the arch of the aorta is distended and lengthened, and its root, like that of the pulmonary artery, descends. The arch of the aorta enlarges both in length and breadth, and becomes tense and rigid. Its lateral enlargement is small, but its elongation is considerable; and its orifice, like that of the pulmonary artery, descends during the systole.

During the systole, the auricles and great vessels enlarge, and descend into the place just left by the retreating ventricles; there is, therefore, more blood at the base of the heart at the end of the systole than at the end of the diastole. Since, however, during the systole, both ventricles contract, the increase of the blood at the base probably balances its diminution towards the apex. During the pause which follows the dilatation of the ventricles, the blood continues to flow into the auricle, so that the amount of blood in the heart and great vessels is greater just before the ventricular systole than at any other period.

Movements of the Papillary Muscles.—That I might observe the action of the papillary muscles, I removed the anterior wall of the right ventricle when the heart was beating *in situ;* and I found that the tip of the anterior papillary muscle of the right ventricle contracted towards the septum during the systole.

I then removed the septum, so as to expose the two papillary muscles of the left ventricle, and I noticed that both the muscles, which during the diastole were wide apart, approximated and came close together during the systole. At the same time the muscles shortened towards their own centre, so that their tips and their tendinous cords descended to the left towards the apex of the ventricle, while their roots of attachment near the apex ascended to the right towards the base of

the ventricle. The fixed point towards which the two ends approximated corresponded apparently to the zone of rest, or stable equilibrium, in the walls of the ventricle, towards which the base and the apex of the ventricle approximate during the systole.

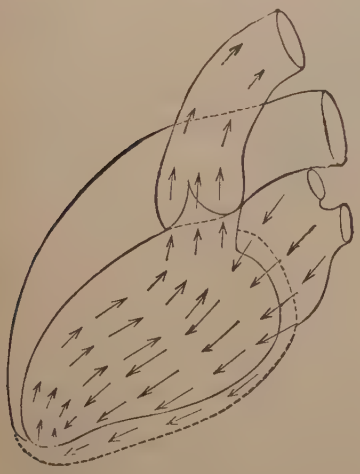
Action of the Mitral and Tricuspid Valves.—In order that I might see the movements of the mitral and tricuspid valves, I cut out the heart when beating vigorously, and immersed it in water. The ventricles contracted with force, and expelled the water from the great arteries during each systole. The jet from the aorta was six inches in length. The segments of the mitral and tricuspid valves were seen to come together at their notched and bead-like margins, so as to close the valves during the systole, and prevent the efflux of a drop of liquid.

At the beginning of each diastole the margins of the valves separated quickly from each other, so as to admit the flow of water freely into the cavity.

Direction of the Currents of Blood in the Cavities of the Heart. (See Figs. 64, 65.)

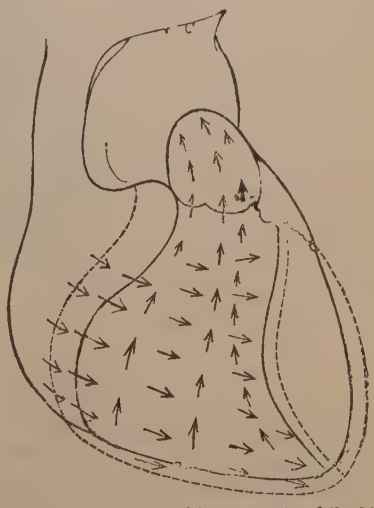
In the left ventricle, the aperture of entrance, the mitral orifice, is contiguous to the aperture of exit, the aortic orifice, the two orifices being separated by a membranous septum consisting of the anterior flap of the mitral valve. In the right ventricle, the aperture of entrance, the tricuspid orifice is at a distance from the aperture of exit at the pulmonary artery, the two orifices being separated by the muscular channel of the *conus arteriosus*. In the left ventricle the current of blood inwards, which descends during the diastole behind the anterior segment of the mitral valve, is parallel in direction to the current of blood outwards, which ascends during the systole in front of that segment. (Fig. 64.) In the right ventricle the current of blood inwards is at right angles to the current of blood outwards, since the blood enters the cavity from right to left, and leaves it from below upwards (Fig. 65). During the systole the stream of blood in the left ventricle takes a spiral direction towards the aortic orifice, in accordance with the direction of the aorta itself. The stream of blood in the right ventricle, as it ascends, mounts over the bulging septum, being restrained by the concave anterior wall of the ventricle. This upward stream, which narrows as it ascends, thus takes the curved direction upwards, backwards, and inwards of the *conus arteriosus* and the pulmonary artery. In the left ventricle, the anterior segment of the mitral valve and the right and left papillary muscles, form a hollow channel for the stream of blood, which, as it ascends to

Fig. 64.



Showing the direction of the currents of the blood in the left side of the heart.

Fig. 65.



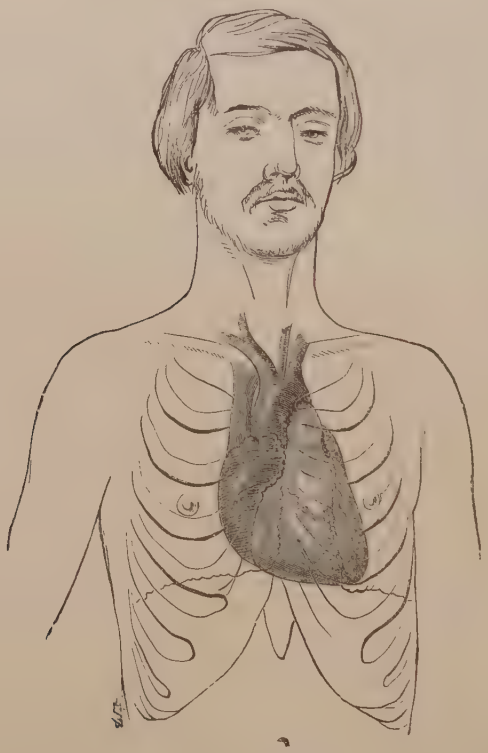
Showing the direction of the currents of the blood in the right side of the heart.

the aorta, presses upon the under surface of the valve. In the right ventricle the stream of blood, as it ascends, sweeps onwards at right angles to the under surface of the tricuspid valve, and rushes between and across the papillary muscles, and through the tendinous cordage that connects the muscles to the flaps of the valve.

THE MOVEMENTS OF THE HEART CAUSED BY RESPIRATION (See Figs. 66, 67.)

During inspiration the diaphragm in its descent draws downwards the fibrous sac and floor of the pericardium, and the whole of its contents. The heart rests upon the central tendon of the diaphragm which forms the floor of the pericardium, and it therefore necessarily rises and falls with the rise and fall of the diaphragm. The descent of the diaphragm is accompanied by the advance of the anterior wall of the chest, which produces the corresponding expansion of the lungs anteriorly. The central tendon of the diaphragm forming the floor of the pericardium presents an inclined plane, upon which the heart glides forwards and downwards during inspiration, under the combined influence of the descent of the diaphragm and the forward movement of the ribs and sternum. Whatever be the cause of the altered level of the diaphragm, whether it contracts and descends, as in inspiration, or is pushed downwards by fluid or tumors in the chest—whether it is raised during expiration, or pushed upwards by distension of the stomach and intestines, by fluid in the abdomen, by abdominal tumors, or by abscess or other affections of the liver; whatever, in short, be the cause producing the ascent or descent of the diaphragm, a corresponding ascent or descent of the

Fig. 66.



Showing the position of the heart and great vessels in relation to the walls of the chest, and the lungs in a healthy man *at the end of a forced expiration.*

heart must ensue. The only exception is the displacement downwards of the central tendon of the diaphragm by means of effusion into the pericardial sac, when the fluid interposes itself between the heart and the diaphragm, with the effect of pushing the diaphragm downwards and the heart upwards. An important part is played by the pericardium in the influence of respiration on the position of the heart. The central tendon of the diaphragm forms the base of the pericardium, upon which the heart rests as upon a floor. The aponeurotic structure of the pericardium, which takes its origin from the central tendon, ascends so as to form a strong fibrous pouch which envelops the whole heart, and gives off a fibrous investment to each of the great vessels as they enter or leave the pericardial sac. Through the medium of this aponeurotic structure, the diaphragm, during its de-

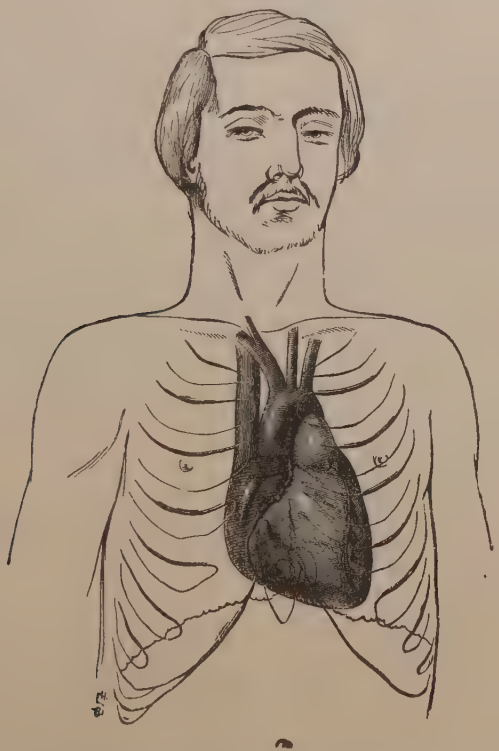
scent, acts so as to draw downwards the great vessels simultaneously with the heart.

The respiratory movements of the heart are vertical. The organ and all its parts and great vessels move downwards during inspiration, and move upwards during expiration. While, therefore, the vertical relations of the heart and great vessels to the parietes of the chest are altered, the lateral relations of the various parts of the heart and great vessels to each other are unaltered, and their relative positions to the surrounding organs are not materially changed.

While the diaphragm descends during inspiration, carrying with it the heart, the front and sides of the cage of the chest, formed by the ribs and sternum, ascend. The change in the position of the heart in relation to the ribs and sternum is, therefore, doubled in extent by the twofold operation of the descent of the diaphragm and the heart simultaneously with the ascent of the cage of the chest.

Inspiration, besides causing the descent of the heart, produces also a lengthening and general enlargement of the organ and its great vessels. The lengthening of the heart and its vessels tells with decreasing effect from below upwards. The descent of the great vessels in the neck is much, but not completely, restrained by the attachments of those vessels. The innominate, the left carotid and the subclavian arteries, and the ascending aorta are elongated and straightened to a considerable extent, and as less blood is sent into those vessels during inspiration than during expiration, they are lessened in width at the same

Fig. 67.



Showing the position of the heart and great vessels in relation to the walls of the chest, and the lungs in a healthy man *at the end of a deep inspiration.*

Note.—The lower boundary of the heart ought to have been somewhat lower in this figure.

time that they are increased in length. The enlargement of the cavities of the heart is limited to the right side. The right auricle receives in increased quantity the blood which has been stored up in the hepatic and portal vessels and the great veins during expiration. The space in the vessels of the expanded lungs for the reception of blood is increased; the blood is sent with greater ease through the pulmonary artery from the right ventricle, in consequence of the enlargement of the pulmonary capillaries, and is at the same time sent in greater quantity from that cavity, because its supply of blood, derived from the auricle, is materially in-

creased during inspiration. The venæ cavæ, the right auricle, the left ventricle, and the pulmonary artery are therefore enlarged both in length and width. The supply of blood to and from the right cavities of the heart, which is thus increased during inspiration, is then probably associated with a corresponding diminution in the supply of blood to and from the left cavities of the heart. The blood is retained in the pulmonary vessels in augmented quantity during inspiration. We may infer that less blood is sent then into the left auricle, and we know that less blood is sent then into the system through the arteries from the left ventricle, than during expiration.

The result of the various co-operating and contending forces which I have just considered are exhibited with, I believe, an approach to accuracy in Figs. 66 and 67, respecting the position of the heart and great vessels in relation to the cage of the chest and the lungs at the end of a forced expiration, and at the end of a deep inspiration.

The greatest change in the relative position of the heart during inspiration takes place at its lower bonndary, the descent of which is equal to that of the central tendon of the diaphragm, or at least one inch. The upward movement at the same time of the lower end of the sternum and the adjoining cartilages is about one inch also. The resulting change in the relative position of the lower boundary of the heart and the external walls ought to be, and I believe is, though I have not ascertained it by exact demonstration, about two inches. The ascertained change in the relative position of those parts is such, that the lower boundary of the right ventricle, at the end of expiration, is situated behind the lower end of the sternum, and at the end of inspiration, behind the lower end of the ensiform cartilage. The result during life, in a robust man, is that at the end of expiration, the impulse of the right ventricle may be perceptible to the left of the lower end of the sternum; while at the end of inspiration it is to be seen and felt beating with considerable force over, below, and to the left of the ensiform cartilage, or in other words, at the epigastrium. The heart has in fact descended into the space previously occupied by the liver and stomach, and instead of being protected at the part spoken of by a bony framework, is at the end of a deep inspiration only covered to each side of the ensiform cartilage by the abdominal muscles. The apex of the left ventricle descends to the same extent during a deep inspiration, or from the fifth rib to the seventh costal cartilage. The impulse at the apex, which at the end of expiration is often felt beating with force in the fourth intercostal space, is at the end of a deep inspiration quite imperceptible. I need not go through the whole of the details of the altered relative positions of the heart and great vessels in relation to the ribs and sternum during expiration, and at the end of a deep inspiration. They speak for themselves, and are exhibited in the accompanying figures. It will suffice, if I describe the altered bearings of the principal landmarks. A transverse boundary line drawn across the top of the right auricle and right ventricle corresponds with the attachment of the great vessels to the heart. This transverse line, which marks the position of the aorta above the right auricle, and the commencement of the pulmonary artery, extends at the end of expiration across the second intercostal spaces, and the intervening portion of the sternum a little below the manubrium; while at the end of inspiration it crosses the lower boundary of the third intercostal spaces, and the visible commencement of the aorta makes a corresponding descent behind the sternum. The top of the arch of the aorta which at the beginning of a deep inspiration is a little below the top of the manubrium, is, at the end of it, at or at a little above the lower end of that bone.

The vertical and forward respiratory movements of the heart explain the difference in the position of the heart in relation to the walls of the chest in weak persons with flat chests on the one hand, and in those who are full-chested and robust on the other. The relations of the heart and great vessels to the cage of the chest follow the type of expiration in the feeble, and the type of inspiration in the strong.

Front View of the Heart and Great Vessels in a Healthy Man with a well-formed Chest. (See Figs. 68, 69.)

The heart and great vessels occupy the central region of the chest. The lower boundary of the right ventricle is situated behind the ensiform cartilage, and is about half an inch or more below the lower end of the osseous sternum;[1] and

[1] All the works on the diagnosis of the diseases of the heart with which I am acquainted, whether published in this country or in Germany, represent the lower boundary of the heart as being situated above the lower end of the sternum. Several of these works have evidently taken their figures from Luschka's well-known drawing, which gives undoubtedly an accurate view of the heart and the surrounding parts in the dead body from which it was taken. It gives, however, on that very account, an inaccurate view of

the top of the arch of the aorta, at the origin of the innominate and left carotid arteries, is about half an inch or more below the upper end of the sternum.

The breadth of the heart is about one-half of the breadth of the chest. The heart, at its extreme limits, extends for a little more than one-third of its breadth into the right side of the chest, and for a little less than two-thirds of its breadth into the left side of the chest, or in that proportion to the right and left of a vertical line drawn down the middle of the sternum.

During the systole of the ventricles the proportion of the heart in the left side of the chest lessens, owing to the inward contraction of the left border of the left ventricle, while that in the right side of the chest increases, owing to the outward expansion of the right border of the right auricle.

The boundary line across the sternum, between the upper border of the heart and the lower limit of the great vessels, is on a level with the third costal cartilages.

The lower boundary of the heart extends, with a slight inclination downwards, from about half an inch below the lower end of the sternum to the fifth left space, just above or on a level with the upper edge of the sixth left cartilage. The lower boundary of the heart ascends during the systole of the ventricle, and descends during its diastole; it descends also during ordinary inspiration, and ascends during ordinary expiration for about the third of an inch. A deep inspiration may bring down the lower border of the heart to the lower end of the ensiform cartilage, and a forced expiration may raise it to or above the level of the lower end of the sternum.

The left boundary of the heart at its apex is situated to the left of the junction of the fifth rib to its costal cartilage, and behind or to the left of a vertical line drawn downwards from the left nipple.[1] The right boundary of the heart extends about an inch to the right of the right edge of the sternum.

The Right Side of the Heart.—The right cavities occupy the whole front of the heart with the exception of its left portion, where the left ventricle comes into view from behind the right ventricle to the extent of an inch in breadth. The transverse or auriculo-ventricular furrow forms the external apparent separation between the right auricle and right ventricle. The auriculo-ventricular furrow sweeps backwards and forwards to so great an extent, to the left during the systole, and to the right during the diastole, that it presents no fixed position during life, but ranges to and fro between certain limits. The upper end of the furrow may be situated at the left edge or at the mid-

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the relative position of the heart in the living man.

I have just stated that the lower boundary of the heart is situated behind the ensiform cartilage, about half an inch or more below the lower end of the osseous sternum, and have done so on the following grounds:—

(1) At the time of death the heart is raised by the elevation of the diaphragm during the final expiration. After death the heart contracts upwards towards its higher points of attachment, so as to leave an average space of half an inch between the lower boundary of the heart and the lower boundary of the *front* of the pericardium; that space being the exact measure of the upward shrinking of the heart after death. The lower boundary of the heart was situated behind the end of the osseous sternum in one-fifth, and below that point in two-fifths of my cases, while it was above that point in two-fifths of them. The lower boundary of the *front* of the pericardium, which marks the position of the lower boundary of the heart itself at the time of death, was behind the lower end of the sternum in one-fifth, and below that point (being situated behind the upper portion of the ensiform cartilage) in two-thirds of my cases, while it was above that point in only one-eighth of them.

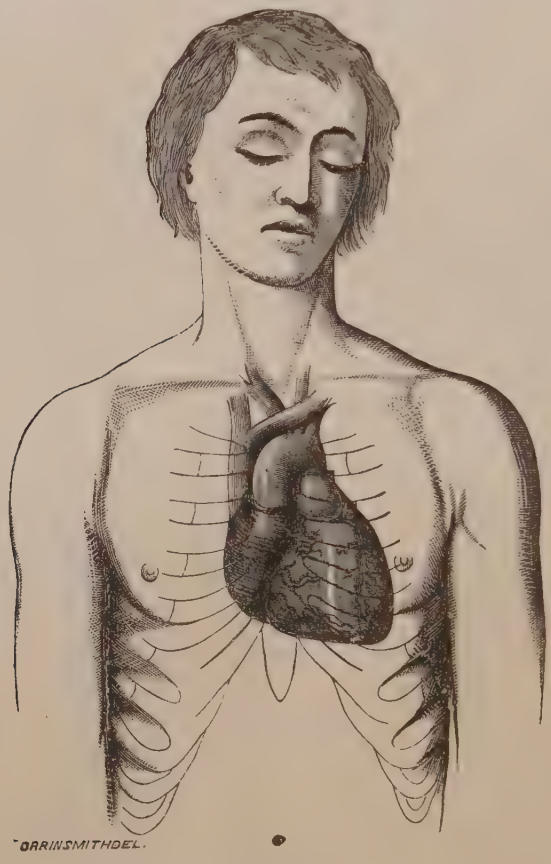
(2) We have already seen (pp. 371, 372) that there is a general correspondence between the relation of the lower boundary of the right ventricle to the end of the osseous sternum, and the relation of the lower border of the apex of the heart to the inferior edge of the fifth costal cartilage and rib. The inferior edge of the junction of the fifth cartilage and rib was on a lower level than the end of the sternum by from a quarter of an inch to an inch and a quarter in 60 out of 71 cases, was on the same level in 5, and was about that level in 6 instances. It is evident that, with few exceptions, the apex-beat could not be felt in the fifth space if the lower boundary of the heart were situated above the end of the sternum.

(3) The lower edge of the anterior portion of the right lung at its left border corresponds, as a rule, with the lower boundary of the heart at the same situation. In six cases the lower edge of that portion of the right lung was behind or on a level with the lower end of the sternum; in three it was above that point to the extent of half an inch; and in twenty it was below that point to an extent varying from a quarter of an inch to an inch and a half, or, in one exceptional case, two inches. We may therefore infer that the lower boundary of the heart was situated in two-thirds of these cases behind the ensiform cartilage, in one-fifth of them behind the lower end of the osseous sternum, and in only one-tenth of them above that end of the bone.

[1] I have made comparatively few observations as to the position of the left nipple in relation to the junction of the adjoining ribs to their cartilages and the left boundary of the heart at its apex.

dle line of the sternum, on a level with the third cartilage; and its lower end may be placed a little below and to the right of the lower end of the sternum, being behind the sternal end of the seventh cartilage, but it may extend for fully half an inch to the right of this position. The transverse furrow thus crosses behind the lower half of the sternum obliquely from left to right, and from above downwards. The upper third of the transverse furrow forms a true line of separation between the auricular appendix and the arterial cone of the right ventricle; but the lower two-thirds of the furrow lie about half an inch to the right of the tricuspid orifice

Fig. 68.



SYSTOLE.

Showing the position and relative size of the various cavities of the heart and of the great vessels during the ventricular systole in a healthy, well-formed man.

Note.—The fifth cartilages were unusually high in the body from which figures 68 and 69 were taken.

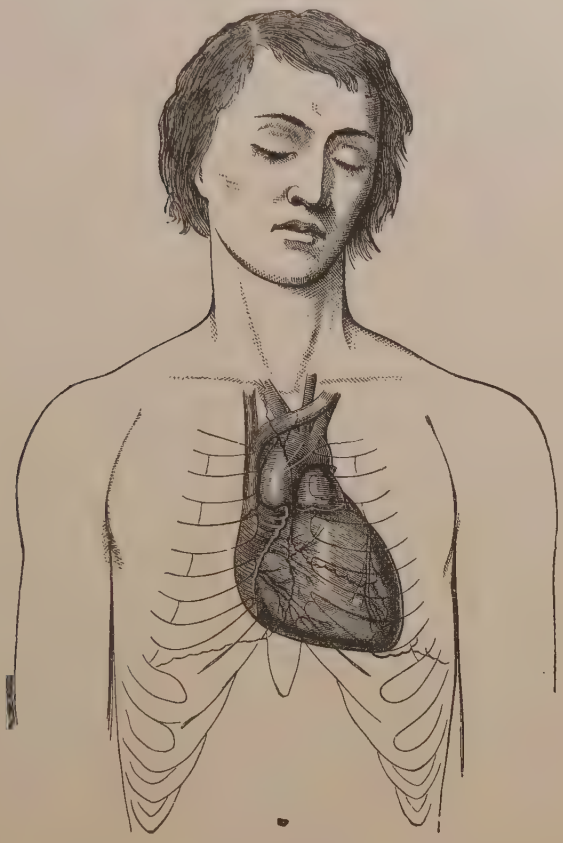
and the line of division between the right auricle and the right ventricle. The right transverse or "auriculo-ventricular" furrow is not therefore at this part of its course a true line of separation between the right auricle and ventricle, but is thrown half an inch to the right of that line by the presence there of the right coronary vessels, and the couch of fat in which they are imbedded.

The Right Auricle.—The right auricle is broad above, where it widens out into the auricular appendix, especially during the systole, and lies behind the middle of the sternum, reaching from its right often to its left edge, on a level with the third cartilages; and it is narrow below, where it appears to come to a point at the lower end of the transverse furrow, to the right of the lower end of the sternum. The real or internal breadth of the right auricle is, as I have just explained, greater than its apparent or external breadth along the line of the transverse furrow. When, therefore, the lower portion of that furrow is situated a little to the right of the sternum, the lower portion of the tricuspid orifice is covered by the lower

end of the sternum, a little to the right of the middle line of that bone. The right boundary of the auricle extends behind the right costal cartilages for about an inch beyond the right edge of the sternum.

The right auricle undergoes more change in form during the action of the heart than any portion of the organ. During the systole of the ventricles the auricle retains its length, but it becomes twice as wide, and its whole surface, instead of being pale and wrinkled, is purple, plump, and glistening. The ventricular border moves extensively to the left, so as to pass from the right margin to the middle line

Fig. 69.



DIASTOLE.

Showing the position and relative size of the various cavities of the heart and of the great vessels during the diastole of the ventricles in a healthy, well-formed man.

of the sternum, while its right border expands a little to the right. There is a slight descent of the upper and lower borders of the right auricle during the contraction of the ventricles. During the diastole of the ventricles these appearances and movements are reversed.

The Right Ventricle.—The right ventricle forms the solid muscular front of the heart, and is flanked to the right by the right auricle, and to the left by that small portion of the left ventricle that comes into view in front of the heart, and forms its left border.

The right ventricle, when exposed to view in front of the heart, presents a pyramidal shape. The base of the pyramid is formed by the lower boundary of the ventricle, which rests on the central tendon of the diaphragm, and extends, with a slight obliquity downwards and from right to left, from the right auricle to the apex of the left ventricle; its upper border is crowned by the pulmonary artery, which forms the apex of the pyramid: its left border is formed by the longitudinal furrow, which divides the right from the left ventricle; and its ostensible right border by the transverse furrow which apparently separates the right auricle from the right ventricle, the actual separation of those two cavities at the tricuspid orifice

being situated, as I have just stated, about half an inch to the left of the transverse furrow.

The right ventricle, in its vertical diameter or length, extends from the third left cartilage to the sixth, which are the cardiac cartilages. In its transverse diameter, or breadth, the right ventricle extends from the transverse furrow, at or to the right of the right edge of the sternum below, and somewhat to the right of the left edge of the sternum above, to the anterior longitudinal furrow, which is situated behind or a little to the right of the junction of the left costal cartilages to their ribs from the third to the fifth.

The length or vertical measurement of the ventricle is greater than its breadth or transverse measurement in the proportion of about four to three. The body of the ventricle forms about the lower two-thirds of the cavity extending from the fourth left cartilage to the sixth, and the *conus arteriosus* forms about the upper third of the cavity extending from the third left cartilage to the fourth. The arterial cone of the right ventricle narrows from below upwards until it ends in the pulmonary artery, and the breadth of the cone a little below the origin of the pulmonary artery in relation to that of the body of the right ventricle, is in the proportion of nearly three to five, or in other words, the width of the cone is nearly three-fifths of the width of the body of the right ventricle.

Owing to the arterial cone being so much narrower than the body of the right ventricle, especially at its right border, the transverse furrow extends further to the left at its upper than at its lower border by more than an inch. In consequence of this great deviation towards its upper end the transverse furrow presents a double curve, which, looking to the right, is concave above, where the rounded auricular appendix fits into the hollow profile of the arterial cone; and convex below, where it is situated half an inch to the right of the tricuspid orifice.

The longitudinal furrow takes a downward direction, with a slight inclination to the left, this inclination to the left increasing rapidly towards the lower end where it approaches the apex. In consequence of this, the longitudinal furrow also presents a double curve, which, looking to the left is convex above, concave below. The deviation to the left of the lower end of the longitudinal furrow is caused by the deviation to the left of the cavity of the right ventricle as it approaches the apex of the heart. The furrow between the ventricles turns to the left at its inferior extremity, and, so to speak, cuts through the apex of the heart. The apex of the heart is thus composed of the apex of the left ventricle and the adjoining left end of the lower border of the right ventricle.

During the contraction of the right ventricle its four sides approximate towards a point of rest or stable equilibrium, which is situated on the anterior wall of the cavity, over or close to the attachment of the anterior papillary muscle, a little to the left of the longitudinal furrow, and slightly nearer to the lower than the upper border of the ventricle. The movement of the transverse furrow to the left is extensive, and that of the longitudinal furrow to the right is slight; the downward movement of the upper border at the origin of the pulmonary artery is considerable, and the upward movement of the lower border of the ventricle is somewhat less. The right border of the *conus arteriosus* moves less to the left than the right border of the ventricle at the tricuspid orifice. At the same time the surface of the ventricle becomes wrinkled, and its coronary vessels start out from the surface and become tortuous. During the dilatation of the ventricle the reverse movements take place, its surface becomes smooth, glistening, and rounded, and the vessels on its surface cease to be prominent. (See Fig. 62, page 401.)

The Left Ventricle.—The left ventricle, where seen in front, comes into view to the left of the right ventricle, and forms the convex left border of the heart. The left ventricle forms here a comparatively long, narrow slip, extending from the third left space down to the fifth, and from the longitudinal furrow behind or to the right of the junction of the corresponding ribs to their cartilages, to the left border of the heart, which reaches up to or just beyond the left nipple. This visible anterior portion of the left ventricle is of the greatest width at and below its middle, behind the fourth space and the fifth cartilage. Above and below this region the ventricle narrows, coming to a point at the apex below, and above bearing to the right, where it is finally hidden by the appendix of the left auricle. The breadth of the anterior visible portion of the ventricle at its widest part is about one-fifth of the breadth of the heart.

The apex of the heart occupies the fifth space, its lower border being situated just above or behind the upper edge of the sixth cartilage and rib, and its left border being at or a little beyond a vertical line drawn down from the nipple.

During the contraction of the left ventricle the right and left borders of its visible anterior portion both move a little to the right, its base and upper portion descend, and its lower portion and apex ascend, both portions moving forwards and to the right. (See Fig. 63, page 401.)

The Appendix of the Left Auricle is situ-

ated behind the third left cartilage close to its junction with the third rib, and fills up the angle or space between the upper end of the left and right ventricles, at the top of the longitudinal furrow, and the left boundary of the origin of the pulmonary artery.

The left auricular appendix is much more prominent and extensive during the contraction of the ventricles, when its right and lower borders move respectively considerably to the right and downwards, and its left and upper borders move obliquely to the right and slightly downwards, than it is during the dilatation of the ventricles, when the auricular appendix shrinks inwards upon itself.

The Great Vessels.—The great vessels lie side by side, the ascending aorta being in the centre, the pulmonary artery to the left, and the superior vena cava to the right, behind the upper portion of the sternum and the adjoining costal cartilages, at and above the level of the third cartilage.

The Arch of the Aorta.—The root of the aorta, including the aortic orifice, valve, and sinuses, is hidden in the centre of the heart. The ascending aorta comes into view just above the appendix of the right auricle, on a level with the third costal cartilages, and is covered by the sternum, the right border of the artery being situated behind or a little to the left of the right edge of the sternum; and its left border, which is partially covered by the right border of the pulmonary artery, being about a quarter of an inch or less to the right of the left edge of the sternum. As the arch of the aorta ascends, it bears to the left, and at the point where it gives origin to the innominate artery, it is exactly behind the middle line of the sternum. From this point the transverse aorta ascends slightly until it gives origin to the left carotid artery, whence it curves backwards and slightly downwards, with an inclination to the left, and gives off the left subclavian artery, the last of its three great branches. The left carotid arises just within a line drawn downwards from the sternal end of the left clavicle, and the left subclavian just without that line. The part at which the innominate and left carotid arteries take their origin is the highest point of the arch, and is situated about three-quarters of an inch or rather less below the top of the manubrium, as far as the breadth of the innominate artery to the left of a line drawn down the middle of that bone, and in front of the lower portion of the body of the third or the upper portion of that of the fourth dorsal vertebra, which corresponds with the third dorsal spine, which is situated midway between the spines of the scapulæ. The transverse aorta, as it curves backwards, to the left and downwards, rests first on the front and left side of the trachea, and then upon the left side of the œsophagus, and is situated between the manubrium, just to the left of the middle line, from three-quarters of an inch or less below the top of the bone down to its lower end in front, and the left side of the body of the fourth and the upper portion of the fifth dorsal vertebra behind. The relations of the transverse aorta to the manubrium in front are very variable, but those to the dorsal vertebræ behind are less so.

The deep left border of the descending portion of the arch may be seen in a front view, and this border is situated in succession behind the left and lower portion of the manubrium, near its junction to the first rib, the first space and the sternal portion of the second left costal cartilage, and the adjoining portion of the sternum. The relations of this important portion of the arch will be considered when the side and back views of the heart and great vessels are described.

The ascending aorta just above the right auricular appendix descends slightly during the contraction of the ventricles; but the top of the arch, at the origin of the innominate and left carotid arteries, is scarcely moved during the contraction of the heart. Inspiration causes the descent of the ascending and transverse aorta and its great branches. This descent is slight during ordinary breathing, but is considerable on a deep inspiration. The inspiratory descent of the arch of the aorta is much less than that of the root of the aorta and heart.

The Pulmonary Artery.—The origin of the pulmonary artery is situated behind the upper portion of the third left cartilage, and its top lies behind the second left cartilage. As the artery ascends to the left of the ascending aorta, it occupies the second left space and cartilage for four-fifths of its breadth, and is covered by the left border of the sternum for the remaining fifth. The pulmonary artery, at its origin, is situated just above and within the appendix of the left auricle; and, as it proceeds on its course, it makes for the hollow of the arch of the aorta, through which it sends its right branch. Its direction is therefore much more from before backwards than from below upwards. The remaining course of the artery cannot be seen in front, and will be considered when the side and back views of the heart and great vessels are described.

During the contraction of the right ventricle the pulmonary artery descends at its origin to a considerable extent, and the higher parts of the artery also descend, but less and less from below upwards. At the same time the whole artery enlarges and lengthens. The pul-

monary artery descends also during inspiration, but to a less extent than the body of the right ventricle, and less at its upper part than at its origin.

The Superior Vena Cava.—The superior vena cava receives the right and left innominate veins a little below the level of the top of the arch of the aorta, behind the right portion of the manubrium, midway between the upper and lower end of the bone. The right innominate vein descends behind the sternal end of the right clavicle, and the left innominate vein crosses in front of the three great arteries, just at or above their origin from the arch of the aorta. The superior vena cava descends immediately to the right of the sternum behind the first space, the second cartilage and the second space, and it opens into the right auricle behind the third right costal cartilage.

The superior vena cava descends slightly at its point of entrance into the right auricle during the contraction of the ventricle. It descends also during the inspiration, but to a greater extent.

The Relation of the Lungs to the Heart in Front.—The lungs cover the great vessels and the whole of the heart except the more prominent portion of the right ventricle which is behind the cardiac cartilages.

The inner margins of the right and left lungs in front meet together behind the upper two-thirds of the sternum, the right lung, as a rule, passing to the left of the centre of the sternum, so as to encroach somewhat on the left side of the chest. The inner margin of the left lung separates from that of the right lung, and diverges to the left on a level with the fourth left costal cartilage. Thence the lower border of this portion of the lung extends to the left, lying behind the lower edge of the fourth cartilage or the upper border of the fourth space, and in front of the body of the right ventricle. Before this border of the lung reaches the longitudinal furrow and the junction of the cartilages to the ribs, it curves downwards, crossing within the fourth space and the fifth cartilage, where it again curves to the right, so as to form a hollow space for the lodgment of the apex of the heart. After crossing the fifth space the inner margin ends in the lower border of the upper lobe, which is situated behind the upper edge of the sixth cartilage and rib, where it soon ends in the septum that divides the upper from the lower lobe of the left lung. Owing to the outward and inward curve thus made by the inner margin of the left lung where it crosses the heart to form the left and lower border of the superficial cardiac space, a remarkable tongue of lung is formed by the inner and lower borders of the upper lobe of the left lung. This tongue of lung, owing to its free position just in front of the interlobular septum, wraps round the apex of the heart, being above, below, outside and in front of it, so as to adapt itself to every movement of the apex. When the apex advances it recedes, when the apex recedes it advances, and thus it allows free play to the apex at the same time that it softens the impulse of the apex upon the walls of the chest, and shields it, when it becomes again flaccid, and retires within its nest.

The inner margin of the right lung, after that of the left lung has deviated to the left, continues its course downwards, behind the sternum, being nearer to the left than the right edge of that bone. It thus completely covers the transverse furrow, the right border of the right ventricle, and the tricuspid orifice. This inner margin of the right lung inclines to the left before it reaches the lower boundary of the heart, where it soon ends in the lower margin of the right lung; which margin lies at first behind the upper part of the ensiform cartilage, then crosses behind the sternal portion of the seventh and sixth right cartilages, and afterwards takes its course to the right, behind or just above the sixth cartilage.

It is evident, from what has just been stated, that the lungs are moulded by a natural adaptation to the form and structure of the heart and great vessels. They thus cover the soft and yielding right auricle, which requires the additional protection of the soft covering in which it is thus imbedded; they thus cover the great vessels, which do not advance so far forwards as the body of the heart; they thus cover the circuit of the ventricles around the three sides of the superficial cardiac space; and they thus leave uncovered the most prominent and powerful portion of the right ventricle. Obeying this law of adaptation, the inner margin of the right lung extends inwards and to the left along its whole length, more than that of the left lung extends to the right; for the greater prominence of the pulmonary artery, of the *conus arteriosus*, and of the centre of the right ventricle, parts that are situated to the left of the middle line of the sternum, offers resistance to the free inward expansion to the right of the margin of the left lung. On the other hand, the less prominence of the ascending aorta, the soft and yielding character of the right auricle and its appendix, and the less prominence of the right border of the right ventricle, parts that are situated behind and to the right of the sternum, allow and even invite the more free inward expansion to the left of the inner margin of the right lung. The inner margins of the lungs, in short, advance freely where they

meet with the least resistance, and stop or even recede where they meet with the greatest resistance.

The Orifices and Valves of the Heart and the Great Arteries.—The orifices and valves of the heart may be considered in two orders: (1) As they are superficial or deep in situation, when the pulmonic and tricuspid orifices and valves would come first, and then the aortic and mitral orifices and valves; and (2) as they are ranged from above downwards, when the pulmonic orifice and valve come first in order, then the aortic, then the mitral, and last the tricuspid orifice and valve. I shall consider them in detail according to the first and most natural of those orders, namely, the superficial and deep orifices and valves, which are also the orifices and valves of the right or anterior and the left or posterior cavities. After doing so, I shall briefly indicate them, for the sake of their common connection, in their order, from above downwards.

The orifice of the pulmonary artery is the highest of the four orifices, and its anterior portion is situated mainly behind the third left cartilage, its right border being covered by the adjoining edge of the sternum. During the systole of the ventricles the anterior portion of the orifice of the pulmonary artery descends into the third space.

The root of the pulmonary artery consists of two anterior sinuses and one posterior sinus, and its valve consists of two flaps in front and one behind, each in its own sinus. The position of the anterior flaps is higher than that of the posterior flap. The anterior or superficial convex wall of the right ventricle is much longer than its posterior or internal convex wall, owing to its outer wall being a section of a much larger sphere than its inner one. When, therefore, the right ventricle contracts, its anterior and outer wall shortens and draws downwards the anterior flaps of the pulmonic valve to a much greater extent than the posterior and inner wall shortens and draws downwards the posterior flap. The result is that when the right ventricle is in a state of complete contraction, the anterior and posterior flaps of the pulmonic valve are nearly on the same level; and that when the ventricle is in a state of distension the anterior flaps may be an inch higher than the posterior flap. This is well seen in several of Pirogoff's vertical sections.

The tricuspid orifice, is the lowest as well as the most superficial of the four orifices, and is separated from the orifice of the pulmonary artery by the *conus arteriosus* of the right ventricle. In a healthy active man with a well-formed chest, the tricuspid orifice is situated behind the lower fourth of the sternum to the right of the middle line of that bone, its upper border being on a level with the lower edge of the fourth cartilage, and its lower border being behind the lower end of the sternum, and the articulation to it of the right sixth cartilage.

The tricuspid orifice is situated about half an inch to the left of the right transverse auriculo-ventricular furrow. It is impossible to assign accurately a fixed position to the tricuspid orifice, owing to its extensive movement to the left during the contraction, and to the right during the dilatation of the right ventricle. The limits of the range of this movement may, however, be defined to the right by a line a little to the right of the sternum, and to the left by a line a little to the left of the middle line of that bone, the orifice playing backwards and forwards behind, and to the right of the right half of the lower portion of the sternum.

The position of the flaps, the tendinous cords, and the papillary muscles of the tricuspid valve have been already described in detail.[1] It will, therefore, be sufficient to say here that the papillary muscles radiate like a fan upwards, outwards, and downwards from the cords and flaps of the valve; that the superior papillary muscle, when present, ascends behind the fourth cartilage; that the anterior papillary muscle takes the direction outwards of the fifth cartilage; and that the inferior papillary muscles descend behind the sixth cartilage.

The root of the aorta,[2] including its orifice, valve and sinuses, occupies the space between the pulmonic and tricuspid orifices. The root of the aorta, and the aortic vestibule, which is the channel or chamber with rigid walls that leads to it from the cavity of the left ventricle, project forwards in front of that cavity and of its mitral orifice, so that the orifice of the aorta, covered by the posterior wall of the *conus arteriosus*, interposes itself, as has just been stated, between the pulmonic and tricuspid orifices. By this arrangement the aortic orifice advances more nearly to the front of the chest, the shallow *conus arteriosus* being in front of the orifice, and the deep cavity of the right ventricle being below it. Hence the murmur of aortic regurgitation, and an intensified aortic second sound, and coincident doubling of that sound, are heard loudly over and to the left of the middle third of the sternum in front of the arterial cone and the root of the aorta; and feebly over and to the left of the lower third of the sternum, in front of the cavity of the right ventricle. The root of the aorta is somewhat overlapped above and

[1] See pages 394–398.

[2] I have already described the anatomical relations of the root of the aorta. (See pages 383–386.)

to the left by the root of the pulmonary artery, and is situated accordingly below and to the right of the pulmonic orifice, behind the left half or three-fifths of the sternum, on a level with the third space, the left portion of the aortic orifice extending beyond the sternum so as to lie within that space. The upper and left border of the aortic orifice, especially during the diastole, is seated behind the lower portion of the third cartilage, near the sternum; and its lower and right border, especially during the systole, is situated behind the middle line of the sternum, on a level with the upper portion of the fourth cartilage.

The root of the aorta descends considerably and moves to the left, so as to approach towards the apex during the contraction of the left ventricle, and at the same time the apex moves to a less degree upwards, and to the right, so as to approach towards the aortic orifice.

The mitral orifice is situated partly behind and partly below the level of the aortic orifice, its upper third or upper two-fifths being behind, and its lower two-thirds or three-fifths below the level of that orifice; and partly behind and partly above the level of the tricuspid orifice, its lower two-thirds or three-fourths being behind, and its upper third or fourth being above the level of that orifice. The mitral orifice is seated behind the left half of the sternum, at the upper two-thirds of the lower third of that bone, on a level with the fourth cartilage, the fourth space, and the upper portion of the fifth cartilage. It is impossible to assign a fixed position to the mitral orifice, for it, like the tricuspid orifice, plays to and fro during the contraction and dilatation of the ventricles. The limits of its movement may, however, be approximately defined by a line a little to the right of the middle line of the sternum on the one hand and a line corresponding to the left edge of the sternum on the other. I have already described the anatomical relations of the mitral valve,[1] and it will therefore be sufficient to state here that the left or upper and the right or lower papillary muscles, starting from their attachments through their tendinous cords to the flaps of the valve, concentrate themselves towards their roots at the apex, instead of radiating from the flaps upwards, outwards, and downwards, as in the instance of the tricuspid valve. The left or superior papillary muscle usually follows the course of the fourth cartilage and space, and the right or inferior papillary muscle that of the fifth cartilage, both muscles dipping downwards towards the lower cartilage or space as they approach the apex.

[1] See pages 391–394.

It may be gathered, from what has just been said, that each of the higher orifices overlaps in position the orifice immediately below it. Thus the pulmonic orifice at its lower or right edge is situated to a slight extent in front of the upper and left edge of the aortic orifice; the right posterior or lower flap of the aortic valve is situated in front of the upper third or two-fifths of the mitral orifice; and the lower two-thirds or three-fourths of the mitral orifice is behind the corresponding upper portion of the tricuspid orifice.

The position of the orifices and valves of the heart in relation to the deeper parts of the heart and of the chest, and to the spinal column, will be considered when the side and back views of the heart and great vessels are described.

The Position of the Heart and Great Vessels in Robust and Feeble Persons. (See Figs. 66, 67, 68, 69, 70.)

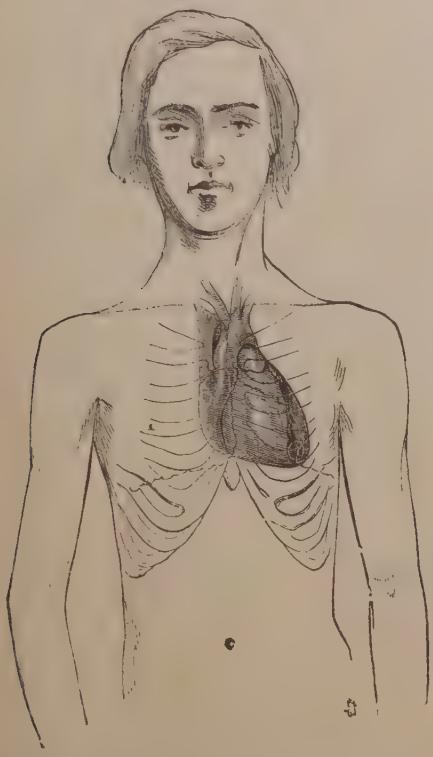
We have just seen that respiration materially alters the position of the heart and the great vessels, and that at the end of a deep inspiration the lower boundary of the heart may be two inches lower in relation to the walls of the chest than at the end of a forced expiration. Thus, the lower boundary of the heart is situated behind or even above the lower end of the sternum at the completion of a forced expiration; while it may be situated at the lower end of the ensiform cartilage at the termination of a deep inspiration. Again, the top of the arch of the aorta may be situated behind the upper end of the manubrium at the end of a forced expiration, and behind its lower end on the completion of a deep inspiration.

This great change is produced by a double agency, acting in opposite directions: one, the descent of the diaphragm which lowers and lengthens the heart and great vessels, and lengthens the lungs by lowering their base; the other, the ascent and advance of the walls of the chest in front. This combined downward movement of the heart and arteries, and upward movement of the sternum and cartilages, doubles the effect on the position of the organ in relation to the cartilages and sternum.

In robust persons, who lead an active and laborious life, the amount of reserved air constantly in the lungs is great, the chest is high, deep, and broad, and the heart and arteries are low in position in relation to the anterior walls of the chest. In such persons the chest and its organs present the form and position of inspiration, and they have therefore the *inspiratory* type of chest. (See Figs. 66, 67, 68, 69.)

In feeble persons, on the other hand, who lead an indoor sedentary life, the amount of reserved air constantly in the lungs is small, the chest is flat and narrow, and the heart and arteries are high in position in relation to the anterior walls of the chest. In such persons the chest and its organs assume the form and position of expiration, and they present the *expiratory* type of chest. (See Fig. 70.)

Fig. 70.



Showing the heart and great vessels in relation to the front of the chest and the lungs in a slender youth with a small chest.

In robust persons, such as sailors, miners, laborers, and smiths, the lower boundary of the heart may be situated quite an inch below the lower end of the sternum, so that the heart may be felt beating in the epigastrium to the left of the ensiform cartilage and the apex of the heart may be situated behind the sixth left cartilage or space. The lungs at the same time enlarge forwards and downwards, so as to interpose themselves between the heart and the walls of the chest, all but a small space bounded above by the fifth cartilage, on the right by the ensiform cartilage, and on the left by the sixth and seventh cartilages near their attachment to the sternum. The heart's impulse is, therefore, quite imperceptible over the front of the chest, that of the right ventricle being sometimes transferred, as I have just said, to the epigastrium, and the apex beat is lost, being enveloped in the folds of the enlarged lung. In such persons, also, the top of the arch of the aorta is low in position, being perhaps situated quite an inch below the top of the manubrium.

The position of the lower boundary of the heart and the summit of the arch of the aorta being unusually low, the position of every part of the heart and the great arteries is also correspondingly low. It is not necessary to describe the situation of the various anatomical points in detail, but it will be well to name that of the leading landmarks of the heart and great arteries.

The boundary-line across the third cartilage that indicates the upper border of the right auricle and ventricle and the lower limit of the great arteries may be shifted downwards to the level of the fourth cartilages. The position of the origin of the pulmonary artery in front being thus given, that of the aperture and valve of the aorta, being a degree lower and to the left, may be inferred, it being situated behind and a little to the left of the left half of the sternum, on a level with the fourth cartilage and the fourth space. The mitral and tricuspid orifices in their descending order take each of them a lower position, the mitral orifice being situated behind the lower fourth of the sternum, its upper boundary being on a level with the fourth space and its lower border, a quarter of an inch above the lower end of the sternum; and the tricuspid orifice being behind the lower sixth of the sternum and the upper portion of the ensiform cartilage.

In feeble, thin persons, of sedentary occupation, or in those who have only recently recovered from illness, the lower boundary of the heart may be situated behind the lower end of the sternum, or somewhat higher, and its apex may be present behind the fifth left cartilage, and may be felt, therefore, beating, not in the fifth, but in the fourth space. Each lung at the same time lessens at its base, and shrinks away from before the body of the heart, uncovering the apex and the left ventricle, the whole of the right ventricle, and a portion of the auricular appendix, of the pulmonary artery, and even of the ascending aorta. The heart's impulse is, therefore, diffused to an unusual extent over the front of the central part of the chest, from the second space to the fourth, and from the right of the lower portion of the sternum to the apex, being felt not only over the apex, but with considerable force over the right ventricle, where it is usually feeble or absent. A double pulsation may also be often felt over the pul-

monary artery, feeble and soft with the first sound, but sharp and sudden with the second sound. In such persons also the top of the arch of the aorta is high, being situated behind or even above the top of the manubrium.

The position of the other parts of the heart and great vessels is correspondingly high. The boundary-line between the upper border of the right auricle and ventricle and the lower limit of the great arteries may be on a level with the middle of the second space, behind which the origin of the pulmonary artery may be seated. The orifice and valve of the aorta, being a stage lower and to the left, may be on a level with the lower portion of the second space and the third cartilage, behind the left half of the sternum. The mitral orifice may be situated behind the left half of the sternum, behind and just below the central portion of the bone, its upper border being on a level with the upper edge of the third cartilage, and its lower border with that of the upper edge of the fourth cartilage; and the tricuspid orifice may be situated behind the right half of the sternum just below the centre of the bone, so that its upper border may be on a level with the lower edge of the third cartilage or the upper portion of the third space, while its lower border may be on a level with the fourth space.

In many well-formed women of active, healthy habits, the heart and great vessels maintain their proper position. But this is not so in the large class of women who work indoors with the needle, and in whom the chest is wont to be flat, the position of the heart being high.

The effect of tight stays is to lessen the descent of the diaphragm, and to increase, for the sake of compensation, the expansion and elevation of the upper part of the front of the chest. In such persons a double and opposite effect may be produced. The lower boundary of the heart in relation to the lower end of the sternum may be high, but the top of the aorta in relation to the higher costal cartilage may be low.

In children of both sexes the position of the heart in relation to the walls of the chest is high.

SIDE VIEW; AFTER DEATH.

LEFT SIDE. (Fig. 71.)

The ninth plate of my Medical Anatomy represents a side view, looked at from the left side, taken from the body of a robust well-formed man. In this body the lower boundary of the heart was situated behind the ensiform cartilage, an inch and a half below the lower end of the sternum.

In this instance the top of the manubrium was on a level with the middle of the body of the third dorsal vertebra, and the lower end of the sternum was on a level with the upper border of the ninth vertebra. The middle of the sternum corresponded in level to the lower portion of the body of the fourth vertebra; the lower end of the manubrium, to the lower portion of the fifth vertebra; and the top of the lower third of the sternum, to the middle of the body of the seventh dorsal vertebra. The ensiform cartilage was of great length, measuring nearly 3 inches (2·8 inches), and its lower end was about on a level with the upper border of the body of the twelfth dorsal vertebra.

This drawing and Plate X. of the same work show well the great anatomical importance of the somewhat neglected ensiform cartilage, especially to the clinical worker. The front of the diaphragm, and the floor of the pericardium, which is formed by the central tendon of the diaphragm, take their origin in part from the tip of the ensiform cartilage by means of a strong slip of muscular fibres. The lower boundary of the pericardium and of the heart, and the lower boundary of the diaphragm, and with it that of the cavity of the right side of the chest and the right lung, may be brought down on a deep inspiration almost to the extremity of the ensiform cartilage, when that point forms the lower boundary of the chest. In this drawing, the lower boundary of the pericardium and the lower margin of the right lung were situated an inch above the end of the ensiform cartilage, and nearly two inches below the lower end of the sternum, and the lower boundary of the heart at the apex, as I have already remarked, was an inch and a half below the level of the lower end of the sternum.

The top of the arch of the aorta at the adjacent origin of the innominate and left carotid arteries was in this instance four-fifths of an inch (·8 inch) below the top of the manubrium, and was on a level with the upper portion of the body of the fourth dorsal vertebra.

The lower end of the descending portion of the arch of the aorta was in front of the upper portion of the body of the sixth dorsal vertebra, and was on a level with a point a little below the lower end of the manubrium.

The top of the pulmonary artery was a little higher in position than that of the lower end of the descending portion of the arch of the aorta just described; the origin of the pulmonary artery was three-quarters of an inch below the centre of the sternum, and within the third space, and was on a level with the lower portion of the body of the seventh vertebra; and the

pulmonary artery occupied in its ascent the upper portion of the third space, the third cartilage, and the second space.

The top of the appendix of the right auricle was nearly half an inch below the centre of the sternum, and on a level with the cartilage between the sixth and seventh vertebræ. The top of the appendix of the left auricle, and the upper boundary of the left ventricle, which would be a little above the lower boundary of the orifice of the aorta, were about on a level with the middle of the body of the seventh dorsal vertebra behind, and the top of the lower third of the sternum, or about the fourth costal cartilage in front. The lower boundary of the left auricle, which would nearly correspond with the lower boundary of the mitral valve, was in front of the top of the ninth vertebra, and on a level with a point a quarter of an inch above the lower end of the sternum. The lower boundary of the posterior part of the left ventricle was in front of the top of the tenth dorsal vertebra, and about on a level with a point four-fifths of an inch below the lower end of the sternum; while the lower boundary of the left ventricle at the apex was on a level with the lower portion of the body of the tenth dorsal vertebra, and with a point about an inch and a half below the lower end of the sternum.

RIGHT SIDE.

The tenth plate of my Medical Anatomy represents a side view, looked at from the right side, taken from the body of a strong man with a well formed chest of the inspiratory type. In this body the heart was distended with water, and the lower boundary of the swollen right ventricle was situated behind the ensiform cartilage, three-quarters of an inch (·7 inch) above the tip of that cartilage, and an inch and a half (1·4 in.) below the lower end of the sternum.

The top of the manubrium in this instance corresponded in level with the lower border of the body of the second dorsal vertebra; the lower end of the sternum, with the lower border of the ninth vertebra; the middle of the sternum at the level of the third cartilage, with the body of the sixth vertebra; the lower end of the manubrium, with that of the fifth vertebra; and the upper border of the lower third of the sternum corresponded in level with the body of the seventh dorsal vertebra.

The commencement of the superior vena cava in this instance was on a level with a point below the middle of the manubrium in front, and with the body of the fourth dorsal vertebra behind; and the termination of the vein in the right auricle was in front of the cartilage between the sixth and seventh vertebræ, and on a level with a point half an inch below the middle of the sternum, and with the third space.

The top of the appendix of the right auricle was on a level with the middle of the sternum and the third cartilages in front, and the body of the sixth dorsal vertebra behind; the attachment of the lower boundary of the appendix to the body of the right auricle, at the transverse furrow, which corresponds closely to the upper boundary of the tricuspid valve, was on a level with a point an inch and a quarter above the lower end of the sternum in front, and the upper border of the eighth dorsal vertebra behind; and the lower boundary of the right auricle, which corresponds closely to the lower boundary of the tricuspid orifice, was on a level with a point half an inch below the lower end of the sternum in front, and the upper portion of the tenth dorsal vertebra behind.

The origin of the pulmonary artery and the upper boundary of the right ventricle were on a level with a point half an inch below the centre of the sternum and the third space in front, and with the lower border of the sixth vertebra behind; and the lower boundary of the right ventricle in front was situated behind the ensiform cartilage, an inch and a half (1·4 in.) below the lower end of the sternum, and three-quarters of an inch above the tip of the ensiform cartilage in front, and about on a level with the lower border of the body of the tenth dorsal vertebra behind. The lower boundary of the right ventricle was about three-quarters of an inch higher behind than in front.

The lower boundary of the pericardium was about an inch and three-quarters below the lower end of the sternum, and about half an inch above the tip of the ensiform cartilage.

Although I possess other drawings showing a side view of the heart and the other internal organs, these are the only ones that give the relation of the heart and its various parts to the walls of the chest in front and the spinal column behind. Both of these drawings were taken from the bodies of men of a robust frame, with a chest of the inspiratory type, and with a heart well developed and low in position. The relations of the heart to the front of the chest in all their variety have been already abundantly illustrated, and its relations to the spinal column will be further described when the position of the heart and great vessels looked at from the back is considered. Pirogoff gives numerous sections, both vertical and horizontal, showing the position of the various parts of the heart and great vessels in relation to the anterior walls of the chest

and the spinal column, and I therefore refer the reader to the notes describing those sections and two others that are figured in Braun's work. (Note 46; Note 47.)

SIDE VIEW; DURING LIFE.

In a Healthy Man with a well-formed Chest.

LEFT SIDE. (Fig. 71.)

The heart and great vessels occupy the space in the centre of the chest, between the sternum in front and the bodies of the dorsal vertebræ behind. The margins of the lungs fill up the unoccupied spaces in front of the great vessels and the heart; and the œsophagus and descending aorta are interposed between the heart and the bodies of the vertebræ behind.

It is evident that in the recumbent posture and during the ventricular systole, the heart would press backwards upon the œsophagus and the descending aorta so as to render swallowing difficult, and to interfere with the flow of blood to the lower part of the body, unless the heart were supported by some special contrivance. Such support is to be found in the walls of the pericardial sac. The floor of the pericardium is formed by the central tendon of the diaphragm, which is suspended in its place, in the middle of the partition between the chest and the abdomen, by means of the great converging circuit of the muscular fibres of the diaphragm, arising from the ensiform cartilage and the ribs; and is supported firmly from below by the liver and stomach. The heart rests upon the floor of the pericardium, formed by the central tendon of the diaphragm, and this supplies a smooth inclined plane, upon which the heart glides forwards and downwards during inspiration, and backwards and upwards during expiration, so as to adapt itself to the various modulations of respiration. The strong fibrous walls of the pericardium arise from the central tendon of the diaphragm. Those walls are endowed with a fibrous structure which is of special strength posteriorly, where it is firmly incorporated with the coats of the pulmonary veins as they enter the pericardium. A fibrous covering is also contributed by the pericardium to the inferior and superior venæ cavæ where they enter the sac, and to the pulmonary artery and ascending aorta where they leave the sac. In virtue of this arrangement the posterior wall of the pericardium supports the heart forwards and prevents it from making pressure upon the aorta and œsophagus, where they are situated immediately behind the left auricle and the base of the left ventricle. By the distribution also of the fibrous pericardium to the veins entering, and the arteries leaving the sac, and to the branches of those arteries in the neck, the central tendon of the diaphragm, when it descends during inspiration, draws intermediately upon the whole of those vessels so as to save them from dragging immediately upon the heart itself.

Fig. 71.

Side view, looked at from the left side, showing the heart and great vessels in relation to the walls of the chest and the spinal column.

The lower boundary of the heart is on a level with the lower end of the upper third of the ensiform cartilage and the upper edge of the sixth costal cartilage in front, and with the upper edge of the tenth dorsal vertebra behind. The top of the arch of the aorta, at the origin of the innominate, and the left carotid arteries, is on a level with the top of the middle third of the manubrium in front, and the lower edge of the body of the third or the upper edge of that of the fourth dorsal

vertebra behind. The horizontal boundary-line that divides the upper border of the heart from the origin of the pulmonary artery and the ascending aorta, is on a level with the third cartilage in front, and the body of the sixth dorsal vertebra behind. The heart therefore extends downwards from the body of the sixth dorsal vertebra to that of the tenth, and from the third costal cartilage to the sixth; and the great arteries extend upwards from the body of the seventh to that of the third or fourth dorsal vertebra behind, and from the level of the third cartilage to the top of the middle third of the manubrium in front.

The left auricle and ventricle occupy fully as great a proportionate amount of space at the left side of the heart as the right auricle and ventricle do at the front of the heart. The left ventricle occupies by much the largest share of the left side of the heart, and its double-convex cone-shaped outline is completely exposed to view from its base to its apex when the left side of the chest is looked at. The transverse furrow, which divides the left ventricle from the left auricle, follows a direction from above downwards and somewhat backwards. The left auricle rests behind on the descending aorta and the œsophagus; and that auricle, the transverse groove, and the mitral orifice are situated in front of the seventh and eighth dorsal vertebræ and the upper border of the ninth; and on a level with the sternal end of the third and fourth costal cartilages, the fourth space, and the upper edge of the fifth cartilage in front. The upper border of the left ventricle is nearly as high as that of the left auricle, but the lower boundary of the left ventricle which extends down almost or quite to the upper border of the tenth dorsal vertebra, is considerably lower than that of the left auricle, which reaches down to the lower border of the eighth or upper border of the ninth vertebra. The left auricle and ventricle take a direction from behind forwards, to the left and downwards, and as they have a similar inclination to that of the ribs, they, as well as the transverse furrow between them, are covered throughout by the fourth, fifth and sixth ribs. The left auricle and ventricle start from the back of the centre of the chest in front of the bodies of the seventh, eighth, and ninth dorsal vertebræ, and the left ventricle crosses from the back to the front of the chest with a definite leaning to the left, so that its apex points at the left fifth space. The left auricular appendix and the left pulmonary veins, where they enter the auricle at its higher part, are situated in front of the adjoining portions of the bodies of the seventh and eighth dorsal vertebræ and their intervening cartilage, and on a level with the third and fourth costal cartilages and the third space in front.

The anterior longitudinal furrow presents a convex outline, looking forwards towards the pulmonary artery at its upper third, and towards the right ventricle at its lower two-thirds; and a concave outline looking backwards and downwards towards the left auricular appendix and the left ventricle. The upper end of this furrow is in front of the body of the seventh dorsal vertebra and behind the third costal cartilage or space, and the lower end of the furrow at the apex of the heart is situated behind the lower border of the fifth space, and is on a level with the body of the tenth dorsal vertebra behind.

During the ventricular systole, the left ventricle and auricle change remarkably in form, size, and position (Fig. 63). The ventricle contracts and shortens, and the auricle expands and lengthens to a great extent. The base of the ventricle and the adjoining edge of the auricle, the transverse furrow and the mitral orifice advance to a considerable extent forwards, to the left and downwards away from the spinal column and towards the apex of the left ventricle. The apex at the same time moves forwards, upwards and to the right, towards the base, so that the base and apex of the ventricle both approximate towards each other, and towards a zone of rest in the walls of the ventricle, situated nearer to the apex than the base. The anterior wall of the ventricle, at the anterior longitudinal furrow, advances forwards and becomes more convex; while the posterior wall of the ventricle also advances forwards, but to a much greater extent, especially at its middle, where it becomes hollow, the apex pointing downwards; so that the posterior wall of the ventricle, previously convex, becomes concave towards the apex and convex at the base, thus presenting a double curve. All the systolic movements of the left ventricle converge forwards, towards the point of rest on the surface of the right ventricle, about its middle and near the septum.

During the ventricular systole the left auricle becomes greatly distended and expands upwards, forwards and downwards, along its upper, anterior and lower borders respectively, the amount of movement of the auricular appendix being greater than that of the transverse furrow. The posterior wall of the auricle which rests on the back of the pericardium remains stationary.

The right ventricle extends in front from the third cartilage to the sixth, and from the middle of the sternum to the lower portion of the upper third of the ensiform cartilage, and is on a level behind with the body of the seventh dorsal

vertebra at its upper boundary, and with the upper portion or middle of the body of the tenth dorsal vertebra at its lower boundary.

During the systole of the ventricles, the right ventricle advances, while the upper portion of its walls contracts downwards and the lower portion of its walls contracts upwards, those movements converging towards a point situated near the longitudinal furrow and the attachment of the anterior papillary muscle.

The pulmonary artery conceals the ascending aorta in the first half of its course, when we look at the left side of the heart. By removing the fat between the pulmonary artery and the left auricular appendix; the left posterior sinus of the aorta and the left or posterior coronary artery are brought into view, deep behind and beyond the posterior surface of the pulmonary artery. The mode in which the pulmonary artery in its progress backwards, and the ascending aorta in its progress upwards, cross each other, is now well seen. When the arch of the aorta is looked at in front, it does not present the appearance of an arch, since the left border of the ascending aorta is situated almost in front of the deep right border of the descending aorta; and the pulmonary artery covers the left edge of the ascending and almost the whole of the descending aorta, the deep left edge of which is alone visible in front. When, however, the left side of the arch of the aorta is looked at, its arched form is at once apparent, the ascending aorta forming the front, the descending aorta the back, and the transverse aorta the top of the arch.

The pulmonary artery as it ascends makes for the hollow of the arch of the aorta, through which it sends its right branch, and its direction is therefore much more from before backwards than from below upwards.

The origin of the pulmonary artery is situated just behind the third left cartilage, and is on a level with the body of the seventh dorsal vertebra. The upper boundary of the pulmonary artery, at the top of its point of bifurcation, which is also its most posterior portion, is situated in front of the lower portion of the body of the fifth, or the upper portion of that of the sixth dorsal vertebra, and on a level with the second costal cartilage; and the left and right branches of the pulmonary artery are situated in front of the body of the sixth dorsal vertebra, on a level with the second space.

The pulmonary artery in its course from before backwards and upwards presents a convexity on its anterior and upper surface, and a concavity on its posterior and lower surface, and is on a level with the third left cartilage and the second space. The posterior sinus of the pulmonary artery is somewhat lower in position than the two anterior sinuses, and is situated behind the upper portion of the third space. The left bronchus separates the left pulmonary artery from the left pulmonary veins.

During the systole of the ventricles, the whole pulmonary artery lengthens and enlarges. The origin of the artery moves to a considerable extent downwards and forwards, the higher parts of the artery sharing this movement, but to a less and less extent from below upwards. The two anterior sinuses of the pulmonary artery descend more during the systole than its posterior sinus, so that the anterior or higher valves are then more nearly on a level with the posterior or lower valve than during the diastole.

The arch of the aorta, like the pulmonary artery, lengthens and enlarges during the systole, so that the whole arch widens. The orifice of the aorta, which is situated at the centre of the heart, moves to a considerable extent downwards and to the left, the direction of its movement, like that of the mitral valves, being towards the apex. The walls of the ascending aorta also move downwards, but to a less and less extent from below upwards.

The position of the ascending, transverse, and descending portions of the arch of the aorta in relation to the sternum, the adjoining parts, and the spinal column has already been described.

The pulmonic, the aortic, and the mitral orifices and valves are situated in their relative position on an inclined plane, each being one above and behind the other in the order named, the orifice of the pulmonary artery being the highest and most anterior, the mitral orifice the lowest and most posterior, and the aortic orifice holding an intermediate position. The upper and anterior boundary of the pulmonic orifice and valve is behind the third costal cartilage and on a level with the lower third of the body of the sixth dorsal vertebra; and the lower boundary of the mitral valve is on a level with the fifth cartilage, and is situated in front of the lower border of the body of the eighth or the upper border of that of the ninth dorsal vertebra. The aortic orifice, being a stage lower than the pulmonic orifice, by which it is overlapped, is in front of the body of the seventh dorsal vertebra, and the intervertebral cartilage just below it. The mitral orifice is in front of the same intervertebral cartilage, the body of the eighth and the upper border of the body of the ninth dorsal vertebra. The position of the sternum and costal cartilages in relation to those valves need not be here repeated.

The position that I have assigned to

the various parts of the heart and great arteries is that which usually exists in a healthy, well-formed man, but those parts change in position during the systole and diastole of the ventricles, and during inspiration and expiration, in the manner and to the extent that I have already described. In those who are robust and possess a broad and deep chest of the inspiratory type, the position of the heart and arteries and of all their parts are lower, while in those who are slender and possess a narrow and flat chest of the expiratory type, the position of those parts is higher, than in the average man whom I have taken as an example. During inspiration the whole of the anterior walls of the chest ascend considerably, but the spinal column, owing to the deepening of the dorsal arch, descends to a small but definite degree, the descent of the upper being greater than that of the lower dorsal vertebræ, some of the lowest of which are stationary. While, therefore, during respiration, the change in the position of the cartilages and sternum in relation to the heart and arteries is doubled by the inspiratory ascent of those cartilages during the descent of the heart, and by the expiratory descent of those cartilages during the ascent of the heart; the slight respiratory movement of the dorsal vertebræ is in the same direction as the movement of the heart, that of both of them being downwards during inspiration, and upwards during expiration. The result is, that the position of the heart and great arteries in relation to the bodies of the dorsal vertebræ during respiration is more stable than their position in relation to the sternum and cartilages. For a twofold reason, the position of the great arteries in relation to the superior dorsal vertebræ changes less during respiration than the position of the heart in relation to the lower dorsal vertebræ. The first reason is the greater respiratory movement downwards and upwards of the higher than of the lower vertebræ. The second reason is the attachment of the descending aorta by means of the intercostal arteries to the spinal column, as well as that of the great branches of the arch to the head, neck, and arms, which hold the movements of the great arteries in check. The heart itself, on the other hand, is suspended so freely in the centre of the chest that it yields without restraint to every definite influence, being thus moved readily upwards and downwards by respiration, and by the distension and collapse of the abdomen, and from side to side by changes in position, or by effusion into or tumors in the chest, or by contraction or expansion of either lung singly.

RIGHT SIDE.

The position of the heart and great vessels viewed from the right side is much more simple than that of their position viewed from the left side. When the right side of the heart is looked at, the right auricle and ventricle, the descending vena cava, the ascending aorta, and the pulmonary artery are visible, but every other part is concealed. The relative position of the lower boundary of the heart, of the top of the arch, and of the boundary-line separating the great vessels from the heart is necessarily the same on the right side of the chest as on the left side. The upper boundary of the right ventricle is on a level with the body of the seventh, and its lower boundary with that of the tenth dorsal vertebra. The right ventricle occupies the anterior portion of the space between the sternum and the spinal column; and the right auricle, including its appendix, occupies the posterior portion of that space; so that its posterior surface is situated in front of the right side of the bodies of the dorsal vertebræ from the seventh to the upper portion of the tenth, the right pulmonary arteries and pulmonary veins and the right portion of the left auricle being interposed.

The tricuspid orifice is the most anterior and the lowest in position of the four orifices of the heart and great vessels, and is separated from the spinal column by the left ventricle and auricle. The tricuspid orifice is situated, as we have already seen, behind the right half of the lower fourth of the sternum, and is on a level with the bodies of the eighth and ninth dorsal vertebræ.

The descending vena cava is situated to the right of the ascending aorta and on a deeper plane. The commencement of the vein, at the confluence of the two innominate veins, is on a level with the body of the fourth dorsal vertebra, and it enters the right auricle behind its appendix in front of the body of the seventh dorsal vertebra, the right pulmonary artery being just above its termination, the superior right pulmonary vein just below it, and the œsophagus just behind it or to its left. The vein, as it descends, rests upon the right side of the trachea near and at its bifurcation, and upon the right bronchus.

BACK VIEW; AFTER DEATH.

I made observations some years ago on the position of certain parts of the heart and great vessels in relation to the spines of the dorsal vertebræ in eleven different bodies.

The top of the arch of the aorta was situated in front of a point below the spine of the second dorsal vertebra in one instance, just above the spine of the third dorsal vertebra in seven instances, and below the spine of that vertebra in three instances. In other words, in one instance the top of the arch was in front of the upper portion of the body of the third dorsal vertebra, in seven cases it was in front of its lower portion, and in three it was in front of the body of the fourth dorsal vertebra. The lower boundary of the left ventricle was on a level with the spine of the ninth dorsal vertebra in one instance, with a point just above that spine or below that of the eighth vertebra in eight cases, with the spine of the eighth vertebra in one, and above it in one. In other words, the lower boundary of the left ventricle varied in position from the level of the lower edge of the body of the eighth to that of the upper third of the tenth dorsal vertebra. In five instances the upper boundary of the left auricle was on a level with the spine of the fifth dorsal vertebra (in one), or just above that spine (in one), or just below that spine (in three); and its lower boundary was on a level with (in one), above (in one), or below (in three) the spine of the seventh dorsal vertebra. In other words, the upper border of the left auricle was situated in front of the upper part of the seventh dorsal vertebra, or just above it, and its lower border in front of the body of the eighth vertebra.[1]

BACK VIEW; DURING LIFE.

In a Healthy Man with a well-formed Chest. (See Fig. 72.)

When the back of the heart and great vessels is exposed, the left cavities of the heart are brought into view, the lower boundary of the left ventricle resting upon the floor of the pericardium, which conceals the under surface of the heart. When the floor of the pericardium is withdrawn, the under surface of the heart is visible from behind. The under surface of the heart inclines from behind downwards and forwards, and it presents posteriorly, the lower border of the left ventricle from base to apex; anteriorly, the lower surface of the right ventricle; and intermediately, the posterior longitudinal furrow.

The lower boundary of the left ventricle is on a level with or higher than the spine of the ninth and the upper portion of the body of the tenth dorsal vertebra; the top of the arch of the aorta at the origin of the innominate and left carotid arteries is in front of the spine of the third and the lower edge of the body of the third or the upper edge of that of the fourth dorsal vertebra, or it may be somewhat higher; and the boundary line between the heart and the great arteries, at the lower border of the division of the right and left pulmonary arteries and the upper border of the left auricle, is in front of the spine of the fifth and the lower border of the body of the sixth dorsal vertebra. The level of the boundary line between the heart and the great arteries is somewhat higher behind, where it follows the line of the lower border of the division of the pulmonary artery, than it is either in front or at the sides, where it follows the line of the origin of the pulmonary artery or that of the top of the right auricle.

The Left Auricle and Ventricle.—The left auricle and ventricle maintain the same relation to each other and to the spinal column at the back of the chest that the right auricle and ventricle do to each other and to the sternum at the front of the chest, but each portion of the left side of the heart bears more to the left behind, than the corresponding portion of the right side of the heart does in front.

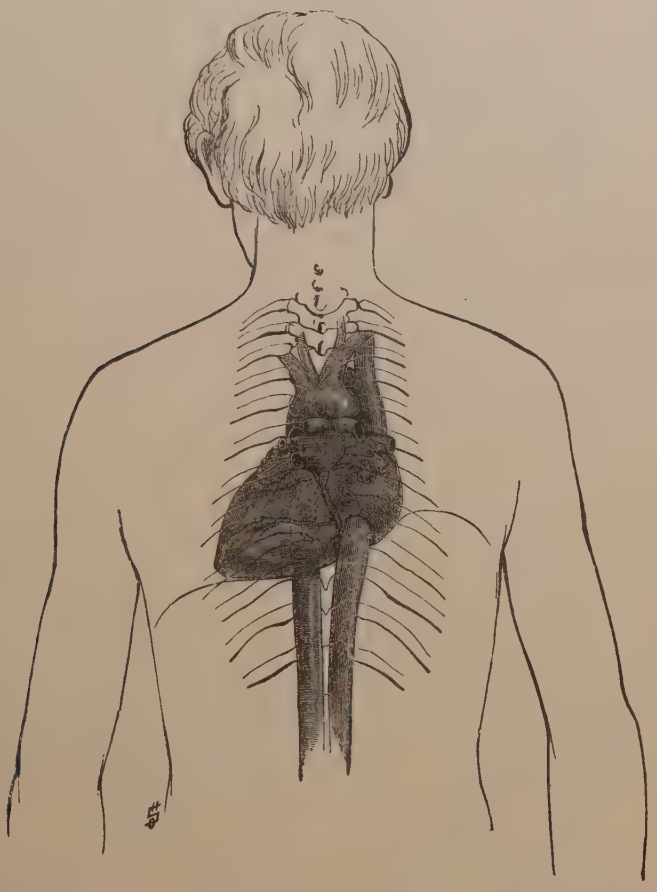
The left auricle at its upper and posterior portion, which includes the auricular appendix, is central, being situated in nearly about equal proportions to the right and left of the middle line of the spinal column. The auricular appendix, which is a semi-detached wing of the auricle, leaves the body of the auricle at its left upper corner and advances forwards and to the left, so as to fill up the deep furrow between the pulmonary artery and the base of the left ventricle. The lowest portion of the left auricle lies entirely to the right of the middle line of the spine, while the left ventricle lies almost completely to the left of it, and the transverse furrow where it separates the two cavities occupies an intermediate position, its upper portion lying considerably to the right, and its lower portion slightly to the left of the middle line of the spine. The left auricle at its anterior aspect lies, when at rest, almost entirely to the right of the middle line of the chest, but its left boundary moves to the left of the middle line when the ventricles contract, and to the right of that line when they dilate. The transverse furrow takes an oblique direction from above down-

[1] Note 46; Note 47.

wards and from right to left, and as it sweeps to and fro, from one side to the other and back again, during the contraction and dilatation of the ventricle, it occupies a position in front of the spines of the fifth, sixth, and seventh, and the bodies of the seventh and eighth dorsal vertebræ, and the upper part of that of the ninth.

The heart is attached to the roots of the lungs by the entrance of the right and left pulmonary veins into the upper part of the left auricle at either side of the spine. The left pulmonary veins are as a rule higher in position, and enter the auricle nearer to the centre of the spine than the right, while the right lower pulmonary vein is larger and lower in position than the left, the right lower vein being sometimes double. The greater size of the lower lobe of the right lung compared with that of the left, evidently accounts for the greater size of the right lower pulmonary veins. The higher position of the left side of the auricle, owing to the presence on that side of the base of the ventricle, and the general inclination downwards of the heart, its longitudinal parts following the line of the longitudinal furrows from right to left, and its transverse parts following the oblique direction of the transverse furrow from left to right, explain, I consider, the

Fig. 72.



Back view, showing the heart and great vessels in relation to the spinal column, the ribs, and the diaphragm.

lower position of the right than the left pulmonary veins. The pulmonary veins have, in short, more room to deploy on the right side of the left auricle, where no object interferes with their freedom, than on the left side of the auricle at its upper angle, where the veins and the auricular appendix are pushed up into a corner by the close proximity of the upper border of the left ventricle. The downward inclination from left to right of the upper boundary of the left auricle, between the left and right pulmonary veins, although quite definite, is very much less than the

downward inclination from left to right of the posterior transverse furrow. The right pulmonary veins are on a level with the spines of the fifth and sixth dorsal vertebræ, and the two left pulmonary veins, holding a higher position, are respectively just above the level of those two spines.

The left ventricle lies to the left of the spinal column, and extends in a direction to the left downwards and forwards, from its base at the back of the chest where it is in front of the spinal column on a level with the sixth and seventh dorsal spines, to its apex at the front of the chest where it is behind the fifth left intercostal space. The upper boundary of the left ventricle is more rounded and more inclined from above downwards than its lower boundary along the line of the posterior longitudinal furrow, where it is more nearly straight and horizontal.

The posterior and left border of the mitral orifice is situated about or fully half an inch to the left of the posterior transverse furrow. This orifice looks towards the apex of the left ventricle, or in a direction to the left, forwards and slightly downwards. Its superior or left angle is a little behind the auricular appendix, on a level with a point above the spine of the sixth, and with the middle of the body of the seventh dorsal vertebra, and about half an inch, more or less, to the left of the middle line of the spine. Its inferior or right boundary is on a level with the spine of the seventh, and the lower portion of the body of the eighth dorsal vertebra, and with a point between the scapulæ, just above their lower angles, and a little to the left or right of the middle line of the spine. The space between the mitral orifice and the apex of the left ventricle is occupied by the flaps of the mitral valve, their tendinous cords, and the papillary muscles, the apparatus of the mitral valve occupying the space at the back of the left ventricle between its base and its apex. The apparatus of the mitral valve is always in action. The transverse furrow and the mitral orifice oscillate to and fro extensively, moving to the left, forwards, and slightly downwards towards the apex during the contraction of the ventricle, and in the reverse direction during the dilatation of the ventricle (see Fig. 63, p. 401). The mitral portion of the left auricle and the base of the left ventricle necessarily share in the movements of the mitral orifice and of the transverse furrow in extent and direction, but the movements of the walls of both cavities, as they recede from the orifice, gradually lessen, and at a zone or transverse circuit of stable equilibrium around each cavity, the walls both of the auricle and ventricle maintain a state of rest. This zone of rest in the left ventricle is probably more near to its apex than its base, while the position of the zone of rest in the left auricle is probably to the left of and just below the termination of the right and left inferior pulmonary veins. The apex moves towards the zone of rest of the ventricle during the contraction of that cavity, but the upper and right boundary of the left auricle moves away from, or to the right of the zone of rest of the auricle during the dilatation of that cavity. Thus during the systole of the ventricle there is a movement, both of the base and the apex of the cavity towards a common centre, tending to its complete contraction; while during the same period the auricle dilates in all directions, and its left and right portions both diverge from the zone of rest of the cavity. The movement of expansion to the left, forwards and downwards, of the mitral portion of the auricle, is much greater than the movement of expansion to the right and upwards of the right portion of the auricle. During the contraction of the left auricle and the expansion of the left ventricle, the reverse movements take place at the mitral orifice, the transverse furrow, and both cavities at all points. The play of all these parts is constant, and they are always undergoing a series of regulated and co-ordinate changes in position. For this reason, although the range of movement of each part, as far as we know it, can be assigned within certain limits, yet the exact position of each part cannot be stated.[1]

The position of the mitral orifice, which is oblique in direction from above downwards, and from left to right, is, as I have just said, in front and to the left of the spines of the sixth and seventh dorsal vertebræ, and between the scapulæ, a little above their lower angles. This region forms a landmark for the position of the mitral orifice and valve over the dorsum. The left ventricle is situated to the left of this region, and extends below its level. During the diastole of the ventricle, the stream of blood from the auricle into the ventricle pours across this region in a direction from right to left, forwards and somewhat downwards. To the right of, and rather above this region, is situated the left auricle; and in cases of mitral incompetence, the reversed stream of blood pours across this region from left to right and somewhat upwards, as it regurgitates from the left ventricle into the left auricle.

In cases of mitral regurgitation, one

[1] I have frequently observed the movements of the heart in animals at the front and the side but never at the back of the organ, so that the movements of the left auricle described above have been derived from inference and not from observation.

might be led, *à priori*, to expect that the mitral murmur would be always audible over the back at the region of the mitral orifice, or midway between the scapulæ, just above the level of their lower angles. This is, however, not usually the case, and especially when the mitral murmur is soft in character, the lungs and chest are of full size, and respiration is free. This is, I believe, to be explained by the great space that intervenes, owing to the presence of the vertebræ, between the mitral orifice and the ear when applied over that region, by the extent to which the lungs envelop the heart and fill the chest backwards, and by the position of the descending aorta and the œsophagus between the mitral orifice and left auricle in front and the spinal column behind. When, however, the mitral murmur is grave, vibrating or musical in character and loud, and when the lungs and chest are contracted and respiration is limited, then the mitral murmur is audible over the back at the region of the mitral valve, and in many cases with great intensity.

The Right Auricle and Ascending Vena Cava.—The inferior and posterior portion of the right auricle, and the entrance of the ascending vena cava into that portion of the auricle are situated at the back of the heart. The right auricle is here separated at its upper boundary from the left auricle below the entrance of the lower right pulmonary vein by a septum, which often makes but little mark externally. The lower boundary of the right auricle is defined by the continuation backwards of the posterior transverse furrow between the base of the left ventricle and the right auricle, until it reaches the posterior longitudinal furrow. The posterior and inferior portion of the right auricle thus fills up the angle formed between the lower border of the left auricle and the base of the left ventricle posteriorly. This angle is formed by the downward prominence and thickness of the muscular wall of the left ventricle at its base.

The ascending vena cava penetrates the diaphragm on a level with the eighth or ninth dorsal spine, where it is situated nearly half an inch to the right of the descending aorta, and of the middle line of the spine; and after ascending to the extent of an inch or less, it enters the right auricle on a level with the seventh dorsal spine, about three-quarters of an inch to the right of the descending aorta.

The Under Surface of the Heart; the Longitudinal Furrow and the Right Ventricle.—The posterior longitudinal furrow divides the left ventricle behind from the right ventricle in front, on the under surface of the heart, and when that organ rests upon the floor of the pericardium, the transverse furrow and the right ventricle are hidden. When, however, the floor of the pericardium is lowered so as to bring into view the under surface of the heart, which inclines from behind, forwards and downwards, the posterior longitudinal furrow, and the under surface of the right ventricle beyond and in front of it, are rendered visible. The posterior longitudinal furrow, resting upon and adapting itself as it does to the floor of the pericardium, is comparatively horizontal in direction; but it is slightly convex near the base of the ventricle, owing to the shoulder formed there by the muscular walls. During the contraction of the ventricle, when its base and apex approximate, the transverse furrow changes in direction both toward the base and the apex. The furrow then becomes more convex than before at the base, because the base of the ventricle itself becomes more convex, and it turns or twists downwards in a peculiar manner towards the apex, because the apex itself twists downwards, so as to form a concavity towards that end. The longitudinal furrow then presents, therefore, an outline with a double curve.

The posterior longitudinal furrow at its auricular extremity comes very close to the posterior border of the heart, and I think that it is visible from behind at that point, even when the heart rests upon the floor of the pericardium. Thence the furrow advances forwards and to the left to the apex of the heart, where it divides the left ventricle from the right, and where it joins the anterior longitudinal furrow.

The under surface of the heart contracts gradually from its auricular portion or base, where it is wider than at any other part, to its apex, where it is narrower than at any other part. The under surface of the right ventricle is thus triangular in form, the base of the triangle being at the auriculo-ventricular furrow, and its apex at the apex of the heart. The posterior longitudinal furrow which is straight, forms the posterior side of the triangle, and the lower boundary of the right ventricle, which is somewhat convex, forms its anterior side. This lower boundary of the right ventricle at the front of the heart, which is on a level with the body of the tenth and the spine of the ninth dorsal vertebra, is lower in position than the lower border of the left ventricle at the back of the heart, which is situated in front of the cartilage between the bodies of the ninth and tenth dorsal vertebræ, or a little lower, and is on a level with the space between the eighth and ninth dorsal spines.

The apex of the heart is lower in position than the lower boundary of the right ventricle, and is on a level with the body of the tenth, and with a point above the spine of the ninth dorsal vertebræ, and with the lower angle of the left scapula.

The Great Vessels.—The position of the boundary line between the upper border of the heart and the lower limit of the great vessels is, as I have already stated, higher at the back than at either side or in front. The boundary line dividing the upper border of the left auricle from the lower border of the right and left pulmonary arteries is situated in front of the cartilage below the body of the sixth and the spine of the fifth dorsal vertebra; and the lower end of the descending portion of the arch of the aorta and of the vena cava, where it is lost behind the right pulmonary vein, are nearly on the same level.

The great arteries of the neck, the descending portion of the arch of the aorta, through the medium of the transverse and ascending portions of the arch, the right and left pulmonary arteries, and the right and left pulmonary veins, form in succession a series of central attachments for the heart, which are situated, so to speak, in tiers one below the other. To these must be added, but on a different plane, the descending vena cava. The heart is suspended forwards and downwards from these various attachments. Two of them, those formed by the pulmonary veins and the pulmonary arteries, connect the heart intimately with the roots of the lungs, so that the roots of the lungs and the heart at that position enjoy a common movement of descent during inspiration, and of ascent during expiration, a degree of movement that is measured by the respiratory movements of descent and ascent of the larynx.

The descending portion of the arch is maintained at its lower end in a fixed position in relation to the spinal column by the sixth intercostal arteries. The higher intercostal arteries, those which go to the third, fourth, and fifth intercostal spaces, arise in succession from the descending portion of the arch, in front, in their descending order, of the fifth and sixth dorsal vertebræ. These vessels all ascend from their point of origin to the spaces they respectively supply, the higher arteries making a greater ascent than the lower ones, because they have to reach a higher point in relation to their respective origins; and the right arteries mounting upwards to a greater extent than the left arteries, because they arise from a lower part of the aorta, owing to the right side of the descending portion of the arch being otherwise occupied by the passage behind it of the œsophagus, and under and in front of it, of the right bronchus. The intercostal arteries to the sixth spaces pass directly to the right and left from their point of origin. It is evident, therefore, that the lower end of the descending portion of the arch, which is braced down to the spinal column by the direct origin of the sixth intercostal arteries, is more fixed in position than its upper part, the intercostal arteries from which have a free ascent, and where the œsophagus is interposed between the artery and the spine; that the descending portion of the arch has less range of movement than the transverse portion, the great arteries from which are comparatively long and capable of being put on the stretch; and that the ascending portion of the arch enjoys a still more free play of movement than the transverse portion, for it is weighted at its root by the heart, and it is long, curved, and free from vascular connections.

The descending portion of the arch lies in front of the left half of the bodies of the fourth and fifth dorsal vertebræ, and that of the upper border of the sixth, on a level with the third and fourth, and the space between the fourth and fifth dorsal spines, and with the interscapular space at and below the spines of the scapulæ. This region forms a landmark at the back for the position of the descending portion of the arch of the aorta; over this region direct and even regurgitant aortic murmurs, especially if they are loud, grave, and musical, are often audible, and that with great intensity; and in this region, the signs of aneurism of the descending aorta most frequently betray themselves. It is sufficient if I allude here to the effect in such cases of the pressure of aneurism affecting this artery on the left recurrent laryngeal nerve, which winds underneath this portion of the arch on its way to the larynx; on the œsophagus, where it passes behind the artery; on the left bronchus, where it passes underneath it; on the left pulmonary artery, which is situated in front of the artery; on the bodies of the vertebræ, upon which it rests; and on the intercostal nerves that pass between and to the left of those vertebræ.

I have already described the position of the transverse aorta. The right and left pulmonary arteries are situated in front of the body of the sixth and the spine of the fifth dorsal vertebra, and they, as I have just said, form one of the two great points of attachment of the heart to the roots of the lungs. The principal points of clinical interest with regard to those arteries is the one I have just alluded to in relation to the pressure of aneurism of the descending aorta on the right or left pulmonary artery, which interferes with the supply of blood to the lungs; of the analogous effect of aneurism of the transverse aorta, below which the division of the pulmonary artery is situated; of aneurism of the ascending aorta on the right pulmonary artery, which often leads to secondary affections of the right lung; and on the effects of the pressure of an

intra-thoracic tumor or enlarged bronchial glands on either of those arteries.

The right pulmonary artery is somewhat lower in position than the left pulmonary artery, in the same way and for the same reasons that the right pulmonary veins are lower than the left pulmonary veins.

The descending vena cava is seen from behind, winding round the right side of the ascending aorta; and its great affluent, the left innominate vein, lies in front of the upper border of the transverse aorta and the great arteries that spring from it. Aneurisms of the ascending aorta tend therefore to make pressure on the descending vena cava so as to impede or arrest the flow of blood through that vein to the heart, and the same may be said with regard to the effects of the pressure of aneurisms of the transverse aorta in impeding or arresting the flow of blood through the left innominate vein.

The descending aorta, being attached by the intercostal arteries to the spinal column, is situated in front of the bodies of the dorsal vertebræ at their centre and left side, and it is therefore interposed between the mitral orifice and the base of the left ventricle in front and the spine behind. The œsophagus lies in front of the right side of the spinal column until it reaches the bodies of the eighth, ninth, and tenth dorsal vertebræ, which are on a level with the seventh, eighth, and ninth dorsal spines, where it gradually passes over the front of the aorta. It is thus interposed between the left auricle and the right side of the spinal column, and finally between the base of the left ventricle in front and the aorta and spinal column behind.

The right and left lungs at the back of the chest fill up the deep hollow in front of the angles of the ribs, and their inner margins overlap respectively the right and left borders of the bodies of the dorsal vertebræ.

The lungs at the back and both sides completely envelop the heart and great vessels, with the exception of those parts that lie at the very centre of the chest, in front of the anterior portion of the bodies of the dorsal vertebræ.

NOTES.

Note 1.—Pirogoff, in his valuable "Anatomia Topographica;" Braun, in his beautiful "Topographisch-Anatomischer Atlas;" and Le Gendre, in his "Anatomie Chirurgicale Homolographique," give drawings taken from sections of the frozen dead body representing the position of the internal organs. In this and the following notes I shall briefly describe the position of the heart as it is represented in those various drawings. I may remark that many of these drawings are evidently not of the size of nature.

Pirogoff represents vertical sections of twelve different bodies, the section being made either through the centre of the sternum in front and the spinal column behind or to the right or left of the centre. In these instances the front of the pericardium was lower in position than the front of the heart to an extent varying from ·8 or ·9 inch to ·02 inch. Between these two extreme instances there was every variety of difference from ·2 inch to ·7 inch, the average extent to which the front of the pericardium was lower than the front of the heart being ·4 inch, or less than half an inch.

These drawings of Pirogoff represent, which mine do not, the relation of the whole under surface of the heart to the floor of the pericardium. In two of them, the whole lower surface of the heart, including both ventricles and the longitudinal furrow between them, rested upon the pericardium; while in one of these, with healthy organs, the front of the pericardium was ·7 inch, and in another with ascites it was ·35 inch below the front of the right ventricle. In the latter case the fluid in the abdomen pressed the pericardium upwards into close contact with the heart, and elevated that organ. In four other cases the right ventricle rested upon the pericardium, while in all of these the interventricular furrow was separated by fluid from the pericardium from ·2 in. to ·65 in., and in three of them the left ventricle was higher than the pericardium from ·3 in. to ·4 in. In the six remaining cases, a film of fluid, varying from ·1 in. to ·5 in., separated both ventricles and the longitudinal furrow from the pericardium; in two of those cases the separation of the two surfaces was equal throughout; in two it was greater at the furrow than the ventricles, and greater below the left ventricle than the right; and in two it was greater below the right ventricle than the left.

Note 2.—Pirogoff represents the exact position of the lower boundary of the front of the heart in relation to the lower end of the bony sternum in five instances in which a vertical section was made through the centre of the sternum in front and the spinal column behind. In two of these instances the lower boundary of the heart was above the lower end of the sternum to an extent varying from ·8 in. to ·7 in., and in three of them it was below the lower end of the sternum to an extent varying from one inch to half an inch. He also gives thirteen cross sections of the body that show whether the lower boundary of the heart was higher or lower than the lower end of the sternum. In one instance the lower border of the heart was very much below, and in another it was very much above the lower end of the sternum. The latter case stood alone. The section was made through the lower margin of the nipples and the middle of the third space, and only a small piece of the ventricles towards the apex remained frozen in the pericardial fluid; the heart being absent from behind the centre of the sternum. The stomach and the œsophagus were enormously distended with

food, and both the stomach and the liver rose high into the cavity of the chest, above the level of the section. In eight other cases the section was made, as in this one, through the third cartilage, in nine others through the fourth, and in four others through the fourth space; and in all of these, amounting to twenty-one, the heart was present in the section of full size.

Braun gives vertical sections of the body through the centre of the sternum and the spine in two instances, in one of which the lower boundary of the heart is half an inch above, and in the other is an inch and a third below the level of the lower end of the sternum.

Note 3.—The position of the lower boundary of the pericardium in relation to the lower end of the sternum is represented by Pirogoff in the two groups of sections, vertical and transverse, referred to in Note 2. In two of the vertical sections the lower boundary of the front of the pericardium was above the level of the lower end of the sternum from the tenth to the third of an inch, and in three of them it was below the lower end from 1·2 in. to ·88 in. In the thirteen cross sections the lower border of the pericardium was above the level of the lower end of the sternum in only one case, and below it in twelve cases.

Note 4.—Pirogoff represents the position of the apex in relation to the fourth, fifth, and sixth spaces and cartilages in the two groups of vertical and transverse sections. In one of the vertical sections, a case of ascites, the apex was situated in the fourth space; in another, it was situated behind the fifth rib, and in a third behind the sixth rib; while of the cross sections, in five the apex was situated in the fifth space, in five behind the fifth cartilage, and in one behind the fourth cartilage or the third space. Five vertical sections also represent the relation of the lower boundary of the right ventricle to the cartilages and spaces, at a point intermediate between the lower boundary of the sternum and the apex; in two of these the inferior margin of the right ventricle was behind the seventh cartilage, in one behind the sixth cartilage, in one behind the fifth space, and in one behind the fifth cartilage.

Note 5.—Pirogoff, in the three vertical and eleven cross sections referred to in Note 4, shows the relation to the cartilages and spaces of the lower boundary of the pericardium below the apex. In two of the three vertical sections representing the apex, the inferior border of the pericardium was lower than the inferior border of the apex from two-thirds of an inch (·7 in.) to half an inch (·4 in.); and in the remaining one the pericardium fitted close upon the apex. In two of these cases the lower boundary of the pericardium below the apex was behind the sixth cartilage, and in the third, that affected with ascites, behind the fifth cartilage. In three of the cross sections the lower boundary of the pericardium below the apex was situated behind the sixth cartilage, in five of them it was behind the fifth space, in two behind the second cartilage, and in the remaining one behind the fourth cartilage. In two of the five vertical sections in which the relation of the lower border of the right ventricle to the cartilages and spaces is shown, the lower boundary of the pericardium below the ventricle was situated behind the seventh cartilage, in two behind the sixth space, and in one behind the sixth cartilage.

Note 6.—Pirogoff gives a series of deepening sections made downwards and from side to side, presenting a front view of the organs. In two of the more superficial of these sections there was an inclination of two thirds of an inch (·7 in.) from right to left extending from the lower boundary of the right auricle to the apex of the heart. In a third section, a case of ascites, there was no inclination, the apex being on the same level as the lower border of the right auricle. When the sections deepened, the inclination was still maintained, but the dip from auricle to the apex was less great. Thus in three sections in which the lower border of the left ventricle was exposed, the dip from auricle to apex was respectively one-half (·4 in.), one-third (·3 in.), and one-sixth (·15 in.) of an inch, the latter section being progressively deeper than the former. In like manner, but with a different effect, in two other sections of the case of ascites, the lower boundary of the apex was higher than that of the auricle, in one section by the fifth of an inch (·2 in.), and in a deeper section by half an inch (·5 in.)

Note 7.—Pirogoff shows the extent to which the heart extends to the left of the middle line of the sternum in four (or five) vertical, and in eighteen (or seventeen) cross sections. The heart extended to the left of the centre of the sternum from two inches to two and three-quarters (2·8 in.) in two-thirds of these cases (14 in 22); from an inch and a third (1·4 in.) to an inch and three-quarters (1·85 in.) in one-third of them (7 in 22); and three inches and a third (3·4 in.) in one additional instance.

Note 8.—Pirogoff indicates approximately the position of the top of the arch in five vertical and four cross sections. In two of the vertical sections the top of the arch appeared to be respectively a quarter and a tenth of an inch above the top of the manubrium, on a level in one with the top of the second, and in the other with the top of the third dorsal vertebra. In the three other vertical sections the top of the arch was three-quarters of an inch (·6 to ·8 in.) below the top of the manubrium, and on a level with the lower portion of the third dorsal vertebra. In one of the four cross sections the top of the arch at the origin of the innominate and left carotid arteries was on a level with the lower edge of the sterno-clavicular articulation, and with the lower border of the second or upper border of the third dorsal vertebra; while in three of them it was on a level with the first space, and in the individual cases respectively with the lower border of the second, the lower border of the third, and the upper border of the fourth dorsal vertebra. Braun gives two vertical sections, in one of which the top of the arch of the aorta was from a quarter to

halt an inch below the top of the manubrium and on a level with the third dorsal vertebra, while in the other it was more than an inch below the top of the sternum and on a level with the fourth vertebra.

Note 9.—The lower boundary of the heart was from two-thirds of an inch (·6 in.) to an inch below the lower end of the sternum in Pirogoff's three vertical sections in which the top of the aorta was three-quarters of an inch (·6 in. to ·8 in.) below the top of the manubrium; and was an inch and a quarter below the end of the sternum, reaching, indeed, to the tip of the ensiform cartilage in Braun's case, in which the top of the aorta was more than an inch below the top of the sternum. On the other hand, the lower boundary of the heart was three-quarters of an inch (·8) above the lower end of the sternum in one of Pirogoff's cases, in which the top of the aorta was above the top of the sternum, and was fully half an inch above that bone in Braun's case, in which the top of the aorta was from a quarter to half an inch below the top of the sternum.

Note 10.—Pirogoff shows in his vertical sections the position of the origin of the pulmonary artery in eight instances, and that of the top of the auricular portion of the right auricle in seven. The origin of the pulmonary artery was situated behind the second cartilage in one instance, and behind the fourth cartilage in another; in three cases it lay behind the third cartilage, and in one behind the second space; while in two it lay from two to two and a half inches below the top of the manubrium. The top of the right auricle was seated behind the second cartilage in two cases, behind the third cartilage in two, and below the top of the manubrium from an inch and a half to an inch and three-quarters in three. In one of the instances in which it lay behind the third cartilage, it was three inches below the top of the manubrium.

Note 11.—In five of Pirogoff's vertical sections referred to in Note 10 the vertical length of the pulmonary artery and the right ventricle is shown. In two cases the vertical length of the pulmonary artery was about half an inch, and in these two cases the vertical length of the right ventricle was respectively three inches (3·2 in.) and two and a third (2·3 in.). In the three other cases the vertical length of the pulmonary artery was about one inch (·9 in., 1·05 in., 1·2 in.), that of the right ventricle in those cases being about three inches (2 8 in., 3·1 in., 3·5 in.). In the three latter cases, in which the pulmonary artery was relatively long, the length of the ventricle to that of the artery was as three to one; while in the two others in which the vessel was short, the ventricle was from four and a half to six times the length of the artery.

Note 12.—In one of Pirogoff's transverse sections, referred to in Note 11, the top of the pulmonary artery was situated just above the second cartilage, and the artery, in its short upward course (·4 in.), was covered by the second cartilage; in another, the top of the artery lay behind the third cartilage, and the artery ascended within the third space. In the three other cases the artery took an intermediate and average position within the second space, its top being seated behind the second cartilage, and its origin behind the third cartilage, or, in one instance, the second space.

The origin of the pulmonary artery was the lowest in position, being behind the fourth cartilage, in the one among these five cases in which the vessel took the longest upward course (1·2 in.); while on the other hand, the origin of the artery was the highest, being behind the second cartilage, in the one in which the vessel was the shortest (·4 in.).

Note 13.—The arch of the aorta, measured from the point at which it came into view above the right auricle to the adjacent origin of the innominate and left carotid arteries, in Pirogoff's vertical sections, varied in approximate vertical length from about one inch to more than two inches (about 2·2 in.), its average length being about an inch and a half. In two cases, in which the vessel was short (about 1 in.) the vertical length of the arch, from the point at which it came into view, was less than that of the heart, measured from the same point, in the proportion of 10 to 25; while in three cases, in which the vessel was long (1·8 in., 2 in., 2·2 in.), the ratio of the length of the vessel to that of the heart was about 10 to 18.

Note 14.—Pirogoff shows the vertical length of the right auricle in six sections. In three of these the length of that cavity was two inches and three-quarters (2·6 in., 2·7 in., 2·8 in.); and in three it was from three inches and a third to almost four inches (3·3 in., 3·4 in., 3·8 in.).

Note 15.—Pirogoff represents the vertical length of the right ventricle in eleven cases. In two of these the cavity was two inches and a third (2·3 in.), and in one it was four inches in length. There was considerable variation in the other cases between these limits, the average length of the cavity in the eleven cases being three inches.

Note 16.—The great vessels occupied the upper half of the sternum, and the heart its lower half, in two of Pirogoff's and in one of Braun's sections. In one of Braun's sections the great vessels lay behind the upper third of the bone, and the heart beat behind its lower two-thirds; in three of Pirogoff's sections the great arteries were covered by the upper three-sevenths of the sternum, and the heart by its lower four-sevenths (1·5 in. to 2·1 in.; 2·7 in. to 3 in.; 1·4 in. to 2·3 in.); while in one of Pirogoff's the great vessels occupied the sternum to a greater extent than the heart in the proportion of eight to seven (3·1 in. to 2·7 in.).

Note 17.—The width of the healthy heart was one-half of the width of the chest in two of Pirogoff's cross sections (7·8 in. to 3·9 in. and 7·2 in. to 3·5 in.); it was one-third of that of the chest in four of them (7·4 in. to 2·4 in., 9·4 in. to 3·2 in., 9·2 in. to 3·2 in., 7·2 in. to 2·6 in.), and in six of them the proportion between the width of the heart and that of the chest varied from 10 to 3·9 to 10 to 4·6. In no instance was the breadth of the healthy heart greater in proportion than

one-half of that of the chest. In this respect Pirogoff's cases differ from mine, for, as I have said above, in one-third of my cases the width of the heart was greater than one-half of that of the chest (10 to 5 to 10 to 6·2). This may partly be accounted for that in Pirogoff's drawings the section was not as a rule made across the widest part of the heart, and that the breadth of the heart was measured from precisely opposite points; whereas in mine the measurement was taken from the point of the heart furthest to the left, which was near the apex, to the point of the heart furthest to the right, which was about the middle of the right auricle; and I need scarcely say that these points were never precisely opposite to each other.

Note 18.—In some of Pirogoff's sections the right ventricle and auricle were proportionally broad in relation to the front of the left ventricle when the heart itself was wide in relation to the width of the chest, while the right cavities were relatively narrow when the heart itself was relatively narrow. In other instances, however, it was the reverse, the heart being relatively narrow or wide, when the right cavities were respectively relatively wide or narrow.

Note 19.—In one of Pirogoff's cross sections the heart extended one inch and a tenth into the right side of the chest, and nearly three inches (2·8 in.) into its left side; while in another of them the heart occupied the right side of the chest for a little less than two inches (1·85 in.), and its left side for a little more than two inches (2·15 in.). In one of these extreme instances nearly three-fourths of the heart occupied the left side, and over one-fourth of it the right side of the chest; while in the other more than one-half of the organ lay in the left side, and less than one-half of it in the right side.

In twenty-five cross sections nearly two-fifths of the heart occupied, on an average, the right side, and fully three-fifths of it the left side of the chest (10 to 17). These sections were made across the heart at all levels, from just below the origin of the great vessels to a little above the lower boundary of the organ. In at least four instances more sections than one were made through the same body at different heights, and in these instances the heart, as a rule, lay more to the right in the higher than in the lower sections. This was due to the greater proportionate prevalence of the right auricle in the higher and middle sections; and of the right and left ventricles in the lower sections of the heart. There were, however, three marked exceptions to this rule, which seemed to be due to the greater extension of the right auricle to the right at its middle than at its higher region.

Note 20.—The right lung was more developed in front than the left in eight out of nine cases, in which two-thirds of the heart or more occupied the left side, and one-third of it or less the right side of the chest; and the development of the two lungs was about equal in seven out of eight cases in which two-fifths of the heart or more lay in the right side, and three-fifths of it or less in the left side of the chest, the right lung being, however, larger than the left in the two exceptional cases.

Note 21.—The breadth of the combined right auricle and ventricle in relation to that of the left ventricle as seen in front in fifteen of Pirogoff's cross sections, varied from 10 to 1·4 to 10 to 4·4, the average proportion being 10 to 3·3.

Note 22.—The auricular portion of the right auricle varied in breadth in Pirogoff's cases from nearly an inch and a half (1·4 in.) to four-fifths of an inch (·8 in.), its average breadth in ten cases being one inch.

Note 23.—The body of the right auricle varied in breadth in Pirogoff's cases from nearly an inch and a half (1·4 in.) to the fifth of an inch (·2 in.), its average breadth in twenty-one cases being two-thirds of an inch (·66 in.).

Note 24.—The left edge of the auricular portion of the right auricle extended almost to the left edge of the sternum (·1 in. from left edge) in one instance; almost or quite to the centre of the sternum, so as to lie behind its right half, in four instances; and in one instance it was covered by the right third of that bone.

Note 25.—The right edge of the right auricle extended to the right of the right edge of the sternum from the third of an inch to an inch, and, on an average, for two-thirds of an inch in sixteen of Pirogoff's cross sections.

Note 26.—The auricular portion of the right auricle was from one-third to two-thirds wider than the body of the auricle in five hearts represented by Pirogoff.

Note 27.—The width of the heart in ten of Pirogoff's sections varied from a little more than twice (22 to 10) to almost four times as great as that of the auricular portion of the right auricle; the heart being on an average fully three times as wide as the auricular appendix.

Note 28.—The heart was from three to nine times wider than the exposed portion of the body of the right auricle in twenty of Pirogoff's cases; the heart being on an average nearly six times as wide as the auricle.

Note 29.—The breadth of the right ventricle varied from four-fifths (10 to 12 5) to a little less than one-half (10 to 20·5) of the breadth of the heart in twenty-one of Pirogoff's drawings, sixteen of which were from cross sections of the body, and five from front views of the heart. The average breadth of the right ventricle in these drawings was two-thirds of the breadth of the heart (10 to 15), and in one-half of them (10 in 21) the proportionate width of the heart was at or above, and in one-half of them (11 in 21) it was below that average. The average proportionate breadth of the right ventricle in relation to that of the heart was 10 to 16 in the sixteen cross sections, and 10 to 14 in the five front views of the heart.

Note 30.—The breadth of the upper part of the *conus arteriosus* varied from one-half (10 to 20) to four-fifths (10 to 17·2) of the breadth of the right ventricle at its middle, in Pirogoff's five front views of the heart; the average width of the *conus arteriosus* being in those

cases fully three-fifths of that of the right ventricle (10 to 18·6).

Note 31.—The length of the right ventricle was equal to that of its breadth in one, and was greater than that of its breadth in four of Pirogoff's five front views of the heart, the average proportion of the length to the breadth of the right ventricle being in those four cases as 5 to 6 (10 to 11·75).

Note 32.—The breadth of the right ventricle in relation to the right auricle in Pirogoff's five front views of the heart varied from 10 to 2·2.

Note 33.—The breadth of the right ventricle varied from an inch and two-thirds (1·65 in.) to nearly three inches (2·9 in.) in sixteen of Pirogoff's cross sections, its average breadth being just over two inches (2·1 in.); while in his five front views of the heart, its breadth varied from two inches and a half to three and a third, its average breadth being almost three inches (2·9 in.). The cross sections were somewhat reduced in size, while the front views appeared to be of the natural dimensions.

Note 34.—In one of Pirogoff's sections the right ventricle extended further to the right than the left of the middle line of the sternum 1·4 in. to 1· in. to left); in one it occupied the right and left sides of the chest in equal proportions (1·2 in. to 1·2 in.); but in fourteen other sections the right ventricle extended more to the left than the right of the vertical centre of the sternum. In two instances six-sevenths of the ventricle lay to the left, and one-seventh of it to the right of the central line; but on an average, two-thirds of the ventricle occupied the left, and one-third of it the right side of the chest.

Note 35.—In three of Pirogoff's five front views of the healthy heart, the longitudinal furrow during its descent took a direction slightly to the left or outwards during its whole course, so that it was about half an inch more to the left at its lower than its upper portion; but in two of them the furrow curved first to the right for the third of an inch (·3 in.), and then to the left for, in one instance, the same, and in the other for a greater extent (·5 in.).

Note 36.—In one of Pirogoff's cross sections the right ventricle extended for only a quarter of an inch to the left of the sternum; but in every other instance that ventricle was covered to a greater or less extent by the costal cartilages. The exact extent to which they were so is not indicated, but I judged that in one-fifth of the cases (3 in 16) the right ventricle extended almost as far to the left as the junction of the cartilages to their ribs; that in one-fourth of them (4 in 16) the ventricle was covered by the two sternal thirds of the cartilages; that in two of them it extended to midway between the sternum and the ribs; and that in one-third of them (6 in 16) it was only covered by the sternal third of the cardiac costal cartilages.

Note 37.—The body of the right ventricle extended to the left of the middle line of the sternum from four-fifths of an inch (·85 in.) to two inches (2·1 in.), and on an average for an inch and a half (1·45 in.), in sixteen of Pirogoff's cross sections.

Note 38.—The right auriculo-ventricular furrow, starting from the right edge of the origin of the pulmonary, as it descended, extended to the right to an amount varying from one inch to one inch and four-fifths, and on an average for an inch and a half (1·45 in.), in Pirogoff's five front views of the healthy heart.

Note 39.—The breadth of the pulmonary artery at its origin varied from an inch to an inch and a half, and was on an average an inch and a quarter, in Pirogoff's five front views of the healthy heart; and in the same cases the breadth of the pulmonary artery a little above its origin varied from three-quarters of an inch to an inch and a quarter, and was on an average about one inch. The pulmonary artery was wider than the aorta in four of these instances, and narrower than the aorta in one of them.

Note 40.—The right edge of the pulmonary artery was covered by the sternum to the extent of the third of an inch in one instance, and the tenth of an inch in another, and the remainder of the artery, amounting to three-fourths of its diameter in one instance (·8 in., ·11 in.), and six-sevenths of its diameter in the other, occupied the second left space or the second costal cartilage.

Note 41.—The *approximate* breadth of the left ventricle as seen in front of the heart varied from almost half an inch (·4 in.) in two instances to an inch and one-fifth (1·2 in.) in three cases, and was on an average three-quarters of an inch in nineteen of Pirogoff's cross sections and five of his front views of the heart. The proportion that the width of the left ventricle at its anterior aspect bore to that of the whole heart in those cases varied from one-eighth (10 to 1·25) to one-third (10 to 3·2).

Note 42.—The apex was covered by the inner margin of the left lung to the extent of from half an inch to three-quarters in three of Pirogoff's cross sections, and to the extent of the tenth and the fifth of an inch respectively in two of them; while in two others the outer edge of the lung was not covered by the lung, which, however, was close to it; and in one other instance the apex was completely exposed, the left edge of the lung being ·6 in. to the left of the apex and ·3 in. to the left of the outer left border of the pericardium.

Note 43.—The ascending aorta varied in breadth from three-quarters of an inch (·7 in.) to an inch and a fifth (1·2 in.) in Pirogoff's five front views of the healthy heart, its average breadth being one inch.

Note 44.—The aorta was narrower than the pulmonary artery in four and wider in one of Pirogoff's cross sections.

Note 45.—The ascending aorta was covered by the sternum in four of Pirogoff's five cross sections showing that vessel, and of these instances, in three the artery was central and in one it inclined to the right. In the remaining case the ascending aorta extended a quarter of an inch to the left of the sternum, being present to that extent within the left second space.

Note 46.—Pirogoff, whose work is rich in illustrations of the root of the aorta, including

its valve and sinuses, represents those parts in eight cross sections, five vertical sections, made through the sternum or cartilages in front, and the spinal column or adjoining ribs behind, and two vertical sections made from side to side. In the eight cross sections the root of the aorta, including its sinuses and the flaps of its valve, was in part covered to a very varying extent by the sternum, and was in part situated behind the corresponding cartilage or space to the left of the sternum. In one of them four-fifths of the artery lay behind the sternum (·8 in.), and one-fifth of it extended to the left of that bone (·2 in.); while in one of them only one-fifth of the vessel (·8 in.) was covered by the sternum, while four-fifths of it occupied the adjoining third left space. There was every gradation between these two extreme instances; and, on an average, less than three-fifths of the root of the aorta lay behind the left portion of the sternum, and more than two-fifths of it behind the corresponding left cartilage or space.

The upper part of the root of the aorta, including its sinuses and the flaps of its valve, was situated in two of the cross sections on a level with the second space, its lower portion being on a level with the third cartilage; in three of them its upper portion was on a level with the middle or lower edge of the third cartilage, its lower portion extending to a greater or less extent to the level of the third space; in one of them its lower border was on a level with the upper half of the third space; and in two of them its upper portion was on a level with the third space, at and above its middle, while its lower portion extended to the level of the upper part of the fourth cartilage. In an additional cross section made through the third space the lowest portion of the right posterior flap of the aortic valve remained, showing its attachment to the anterior flap of the mitral valve.

Pirogoff shows the root of the aorta, including its sinuses and the flaps of its valve, in five vertical sections, of which, (1) two sections were made through the left costal cartilages in front, close to their articulation to the sternum, and the ribs behind near their attachment to the transverse processes of the vertebræ; (2) one through the left side of the sternum and the fifth and sixth cartilages near their attachment in front, and the bodies of the vertebræ behind; and (3) two through the centre of the sternum and ensiform cartilage in front, and that of the spinal column behind.

The relations of the anterior and left posterior flaps of the aortic valve were shown in three of those sections (1, 2), and those of the three flaps, including in addition the right posterior flap, in two others (3). In one section the top of the angle of junction of the anterior and left posterior flaps was situated behind the left third cartilage, in one of them the tenth of an inch (·1 in.) below its upper edge, and in another of them the third of an inch (·3 in.) above its lower edge. In two of them the lower boundary of the section of the aortic valve was half an inch (·5 in. and ·45 in.) below the lower edge of the third cartilage or about the middle of the third space. As, however, in these instances the right posterior flap had been removed, the lower boundary of the valve and of the origin of the aorta must have been about half an inch lower than the lowest point of the section, or behind the upper portion of the fourth left costal cartilage. In the third instance (2), in which also the inferior flap had been removed, the top of the angle of junction of the two superior flaps lay behind the sternum, three-quarters of an inch (·7 in.) below the lower end of the manubrium, or about on a level with the lower border of the second space; and the lowest portion of the section through the aortic valve was situated behind the sternum an inch and a half (1·5 in.) below the lower end of the manubrium, or about on a level with the top of the third space, so that in this instance the lower boundary of the aortic valve would be about on a level with the lower border of the third space. In these three cases the measurement of the section of the aortic valve, the lower portion of those valves being removed, varied from two-thirds of an inch in one instance (·6 in.) to almost an inch (·9 in.) in two instances. In the two remaining sections, however, in which the whole valve was exhibited, its measurement from above downwards amounted to a little over an inch (1·1 in.) in one instance, and to an inch and a half (1·5 in.) in the other. In one of these cases, in which the lower boundary of the heart was four-fifths of an inch (·8 in.) above the lower end of the sternum, the upper boundary of the aortic valve was situated about half an inch (·4 in.) above the middle of the sternum, or about on a level with the second space, and its lower boundary about three-quarters of an inch (·7 in.) below the middle of the sternum, or about on a level with the lower edge of the third cartilage or upper border of the third space. In another case, in which the lower boundary of the heart was situated behind the ensiform cartilage, about an inch (·95 in.) below the lower end of the sternum, the upper boundary of the aortic valve was situated behind the sternum four-fifths of an inch (·8 in.) below the middle of the bone, or about on a level with the lower edge of the third cartilage or upper border of the third space, and the lower boundary of the valve was situated behind the sternum, fully two inches (2·2 in.) below the middle of the bone, and two-thirds of an inch (·65 in.) above its lower end, or about on a level with the fifth cartilage. Keeping out of view this unusual case, it may be said that in Pirogoff's sections, on an average, the root of the aorta, including its sinuses and the flaps of its valve, was situated on a level with the third cartilage and the third space.

Mitral Valve.—In one of Pirogoff's vertical sections the top of the mitral valve was fully half an inch (·55 in.) and in another of them it was a third of an inch (·3 in) above the lower border of the right posterior flap of the aortic valve. In three other sections, the right inferior flap of the aortic valve had been removed, the other flaps being retained; and in one of these sections the top of the mitral valve was the third of an inch, in another it

was the fifth of an inch (·2 in.), and in the third it was about the tenth of an inch above the lower edge of the left posterior flap of the aortic valve.

The lower border of the mitral valve was about an inch below the lower border of the left posterior or the anterior flap of the aortic valve in the three instances in which the right posterior flap had been removed; and it was from fully half an inch to fully three-quarters of an inch below the lower edge of the right posterior flap in the two other instances. In one of Pirogoff's front vertical sections the top of the mitral valve was fully half an inch (·6 in.) above the level of the lower border of the right posterior flap of the aortic valve.

In two of Pirogoff's vertical sections, and probably in a third, the top of the mitral valve was about half an inch (·6 in.) below the level of the middle of the sternum, but it was an inch and three-quarters below that point in another instance in which the lower boundary of the heart was an inch below the lower end of the sternum.

In one of Pirogoff's vertical sections the top of the mitral valve was on a level with the lower edge of the third cartilage; and in three of them it was behind the third space, these occupying respectively the upper, the middle, and the lower portion of that space. If we combine the cases in which the vertical section was made through the cartilages with those in which it was made through the sternum, and estimate in the latter cases the approximate relative position of the valve to the cartilages by its position in relation to the sternum, we find that in two cases the top of the mitral valve was on a level with the lower portion of the third cartilage; in three, with the upper third of the third space; in two, with the middle or lower portion of the third space; and in one, with the fourth space.

In one of Braun's vertical sections (a woman aged 25), in which the lower boundary of the heart was half an inch above the lower end of the sternum, the top of the mitral valve was half an inch (·4 in.) below the centre of the sternum; and in another section (a soldier aged 21), the lower boundary of the heart was an inch and a fifth (1·2 in.) below the lower end of the sternum, and the top of the mitral valve was nearly an inch and a half (1·4 in.) below the middle of the sternum.

The lower border of the mitral valve was situated an inch and a half above the lower end of the sternum in one vertical section, and in two it was as low as half an inch above that point; while in three other vertical sections it was on a level with the fourth space, and in two with the fifth cartilage. If we group the two sets of cases together, it may be estimated that in four of them the lower end of the valve was behind the fourth space, and in four behind the fifth cartilage.

In one of Braun's vertical sections, from a woman aged 25, in which the lower boundary of the heart was half an inch above the lower end of the sternum, the lower boundary of the mitral valve was an inch and a half (1·4 in.) above that end of the bone; and in another, from a soldier aged 21, in which the lower boundary of the heart was an inch and a fifth below the lower end of the sternum, the lower border of the mitral valve was less than half an inch (·4 in.) above the end of the bone.

Pirogoff represents nine cross sections through the second space, the whole of which were above the mitral valve; four through the third cartilage, two of which were above the mitral valve, and two were made through the upper part of the valve; eight through the third space, one of which was above and one below the mitral valve, while five were made through the upper portion of the valve, and one through the middle of the mitral orifice; nine through the fourth cartilage, of which two were made through the upper portion and two through the middle of the valve, while five were made below the valve; six through the fourth space, of which one was made through the top of the valve, and three through its middle, while two were made below the valve; and seven through the fifth cartilage, of which six were below the valve, and one was made through the middle of the mitral orifice.

It is self-evident that, in these cases, the top of the mitral valve occupied the space or cartilage above that in which the section passed through the middle of the mitral orifice, and that the top of the valve was relatively still higher in those cases in which the section was made below the mitral valve.

Estimating the position of the top of the mitral valve approximately in these sections on this view, I consider that the upper boundary of the valve was situated in one case on a level with the second space; in nine, on a level with the third cartilage; in two, with the third cartilage or third space; in nine, with the third space; in two, with the third space or fourth cartilage; in three, with the fourth cartilage; in six, with the third cartilage or space or the fourth cartilage; and that in one instance the top of the mitral valve was on a level with the fourth space.

In these cases, on the basis of the calculation just made, it may be approximately estimated that the average position of the top of the mitral valve was about on a level with the upper half of the third intercostal space.

In the same transverse sections, on a similar approximate calculation, the lower border of the mitral valve was situated about on a level with the third cartilage in one instance; the third space in six instances; the fourth cartilage in two; the fourth space in four; the third space, fourth cartilage, or fourth space in six; the fifth cartilage in four instances; and below that cartilage in one.

The average position of the lower boundary of the mitral valve in these cases appears to me, from as close an estimate as I can make, to be about on a level with the lower edge of the fourth cartilage and the upper border of the fourth space.

Pirogoff represents the mitral valve or orifice in seven cross sections, and in all of them the anterior wall of the mitral orifice was situated more to the right than its posterior wall to an extent varying from one-third (·35 in.) to four-fifths (·8 in.) of an inch.

In four of these sections the mitral orifice was situated behind the left half of the sternum; and in three of them it was placed partly behind the left portion of the sternum, partly behind the cartilages and spaces to the left of that bone. In no instance was the anterior wall or border of the mitral valve seated to the right of the middle line of the sternum.

Tricuspid Valve.—In two of Pirogoff's vertical sections the top of the tricuspid valve was nearly the third of an inch (·3 in.), and in two others it was nearly half an inch (·4 in. and ·45 in.) below the level of the top of the mitral valve.

The lower border of the tricuspid valve was below the level of the lower border of the mitral valve from half an inch, in the first two cases noted above, to three-quarters of an inch (·65 in. and ·75 in.) in the other two cases.

The top of the tricuspid valve was situated, in one of Pirogoff's vertical sections, half an inch, and in two of them one inch, below the centre of the sternum; in another instance it was an inch above the lower end of that bone. In one of Braun's vertical sections, in which the lower boundary of the heart was high, the top of the tricuspid valve was on a level with the centre of the sternum.

The top of the tricuspid valve was on a level with the top of the third space in one vertical section, with the fourth space in another, and with the fifth cartilage in a third instance.

The lower border of the tricuspid valve was one inch above the lower end of the sternum in two of Pirogoff's vertical sections, and an inch and a half above that point in one of Braun's vertical sections, in which the lower boundary of the heart was above the lower end of the sternum; and it was a third of an inch (·3 in.) below the lower end of that bone in two of Pirogoff's sections, in which the inferior boundary of the heart was behind the middle of the ensiform cartilage. The lower border of the valve was on a level with the fourth space in one of Pirogoff's sections, and with the sixth cartilage in two of them.

Pirogoff represents four cross sections through the third cartilage, all of which were above the tricuspid valve; eight through the third space, four of which were above that valve, three were made through its upper portion, and one below it; nine through the fourth cartilage, of which three were above the valve, one was made through its middle, three through its lower portion, one through the bottom of the valve and one below it; six through the fourth space, of which one was above the valve, three through its upper portion, one through its lower portion, and one below it: and seven through the fifth cartilage, of which one was made through the middle of the tricuspid orifice and six below it.

Estimating approximately the position of the top of the tricuspid valve in these cross sections, I consider that the upper boundary of the valve was situated on a level with the second space, or third cartilage in one instance; with the third cartilage or space in two; with the third space in seven; with the third space or fourth cartilage in ten; with the fourth cartilage in three; with the fourth cartilage or space in four; and with the fourth space in two.

I think that we may estimate that in these sections the top of the tricuspid valve was on an average situated behind the lower portion of the third space, or the upper edge of the fourth cartilage.

In the same cross sections, and on a similar approximate calculation, the lower border of the tricuspid valve was about on a level with the third cartilage in one instance; with the third space in one; with the third space or fourth cartilage in one; with the fourth cartilage in one; with the fourth cartilage or space in six; with the fourth space in seven; with the fifth cartilage in three; and with the fifth cartilage or space or lower in ten.

The approximate average position of the lower boundary of the tricuspid valve in these transverse sections appears to me to be about on a level with the lower portion of the fourth space, or upper portion of the fifth cartilage.

Pirogoff represents the tricuspid orifice in eleven cross sections, and in all of them the anterior edge of the tricuspid orifice was more to the right than its posterior edge to an extent varying from a quarter (·25 in.) to four-fifths (·85 in.) of an inch.

The left edge of the tricuspid valve was situated more to the right than the right edge of the mitral valve in six of seven instances in which the section went through both valves, to an extent varying from the tenth to the third (·3 in.) of an inch; while in the seventh instance the left edge of the tricuspid was immediately in front of the right edge of the mitral valve.

In five of the eleven sections the tricuspid valve was situated behind the right half of the sternum; in one of them it was behind the right third of that bone; in one it lay partly behind the right portion of the sternum and partly to the right of it; in two it was central, occupying equally the right and left sides of the sternum, and in the remaining two it lay to the left of the middle line of that bone.

Note 47.—Pirogoff shows the relation of the sternum and costal cartilages in front to the vertebræ behind in twelve antero-posterior vertical sections and in sixty-two cross sections.

In five of the vertical sections the top of the sternum was on a level with the lower border of the body of the second or the upper border of the third dorsal vertebra, or the cartilage between these two vertebræ; in one of them it was on a level with the top of the fourth dorsal vertebra; and in one of them, an instance that stands alone, it was, according to Pirogoff's description, on a level with the upper portion of the first dorsal vertebra. This description is, however, evidently an accidental error, and I, therefore, for the first, read the second vertebra. In Braun's two vertical sections the top of the sternum was on a level with the cartilage between the second and third dorsal vertebræ.

I examined eleven human skeletons in the Museum of the Royal College of Surgeons, with the valuable assistance of Mr. Wright, of the Museum, and I found that in eight of them, including one in the Hunterian Museum, the top of the manubrium was on a level with the second dorsal vertebra,[1] the point varying from its upper to its lower border; and that in three of them it was on a level with the first dorsal vertebra.

In two of Pirogoff's vertical sections the top of the sternum was on a level with the lower border of the third rib, near the spine, in one of them it was on a level with the upper border of the fourth rib, and in one it was above the level of the first rib. In this last instance there was evidently an accidental error.

The lower end of the osseous sternum was on a level with the middle of the eighth dorsal vertebra in two of Pirogoff's vertical sections, in one of which the sternum and ribs had been elevated by a large accumulation of fluid in the abdomen; in one of them it was on a level with the middle of the ninth vertebra, and in one with the cartilage between the ninth and tenth vertebræ.

In Braun's two sections the lower end of the sternum was on a level respectively with the middle and lower border of the ninth vertebra.

In one of the skeletons in the Museum of the Royal College of Surgeons the lower end of the sternum was on a level with the seventh dorsal vertebra, in one with the cartilage between the seventh and eighth vertebræ, in three with the middle of the eighth vertebra, in two with the cartilage between that vertebra and the ninth, and in three with respectively the top, middle, and lower border of the ninth vertebra, the last instance being the skeleton in the Hunterian Museum.

The middle of the sternum which corresponds with its articulation to the third costal cartilages was on a level with the middle of the fifth dorsal vertebra in one of Pirogoff's vertical sections, with the cartilage between the fifth and sixth vertebræ in two of them, and with the middle of the sixth vertebra in another of them, and in Braun's two sections.

The bottom of the manubrium which corresponds with the second cartilage and with the lower end of the upper third of the sternum was on a level with the lower half of the body of the fourth dorsal vertebra in two of Pirogoff's vertical sections, and in two of them and in Braun's two sections with the middle of the fifth vertebra.

The lower end of the middle third of the sternum which corresponds as a rule with its articulation to the fourth costal cartilages was on a level with the lower half of the body of the sixth dorsal vertebra in two of Pirogoff's vertical sections and with the middle of the seventh vertebra in two of them and in Braun's two sections.

In one of Pirogoff's cross sections the sternum at the junction to it of the first costal cartilages was on a level with the upper border of the body of the fourth dorsal vertebra; in four of them the sternum at the spaces between the first and second cartilages was on a level respectively with the upper and lower borders of the second vertebra and the upper border of the third; the sternum was on a level—at the second cartilage, in three instances, with the fifth vertebra;—at the second space, in eight instances, with respectively the fourth, fifth, and sixth vertebræ—at the third cartilage, in four instances, with the top of the fifth vertebra in one and with the seventh in three;—at the third space, in eight instances, with respectively the cartilages between the fifth, sixth, seventh, and eighth vertebræ, and with the bodies of the seventh, eighth, and ninth vertebræ; at the fourth cartilage, in ten instances, with respectively the cartilages between the sixth, seventh, eighth, and ninth vertebræ, and with the seventh and eighth vertebræ and the top of the ninth;—at the fourth space, in six instances, with the cartilages between the sixth, seventh, eighth, and ninth vertebræ, and with the bodies of the seventh and eighth;—at the fifth cartilage, in eight instances, with respectively the lower border of the seventh vertebra, the upper border of the tenth, and the two intermediate vertebræ;—at the fifth space, in two instances, with respectively the eighth and tenth vertebra; and finally, in four instances, the lower end of the osseous sternum or base of the ensiform cartilage at the sixth cartilage, was on a level respectively with the lower third of the ninth vertebra, the cartilage between that and the tenth, and the upper border of the body of the eleventh dorsal vertebra.

The lower boundary of the front of the heart was situated in one of Pirogoff's vertical sections on a level with the middle of the body of the eighth or, according to an evidently erroneous reference, the seventh dorsal vertebra, behind, and the sixth cartilage in front; in two of them on a level with the cartilage between the eighth and ninth vertebræ; in three of them with the top, and in one of Braun's sections with the middle of the ninth vertebra; and in one of Pirogoff's sections with the lower border, and in one of Braun's with the middle of the body of the tenth vertebra. In the last two instances the lower boundary of the heart was situated behind the ensiform cartilage, an inch below the lower end of the sternum, in another instance it was half an inch below, and in two others from half to three-quarters of an inch above the lower end of the sternum.

The lower boundary of the pericardium was on a level in one of Pirogoff's vertical sections with the cartilage between the seventh and eighth dorsal vertebræ; in three of them with the cartilage between the eighth and ninth vertebræ; in one with the upper, and two with the lower portion of the ninth, and in one with the top of the eleventh vertebra.

In these cases the lower boundary of the pericardium was situated from a third of an inch above to fully one inch below the lower end of the sternum, and from a third of an

[1] The *body* of the dorsal vertebra is referred to here and elsewhere, unless it is otherwise specified.

inch above the level of the sixth cartilage to that of the lower portion of the seventh cartilage.

The top of the arch of the aorta was about on a level with the upper portion of the body of the third dorsal vertebra in three of Pirogoff's vertical sections, and with its middle in one of his and in one of Braun's sections, and with respectively the top and middle of the fourth vertebra in one of Pirogoff's and the other of Braun's vertical sections. In these seven cases the top of the arch of the aorta was about on a level with a point varying from a quarter of an inch above the top of the manubrium to an inch and a half below it. In one of Pirogoff's cross sections the top of the arch, at the origin of the innominate and left carotid arteries, was in front of the upper portion of the third dorsal vertebra, and in two of them the arch a little below its top was on a level respectively with the lower border of the third and the upper border of the fourth vertebra.

The top of the pulmonary artery was on a level with the cartilage between the fourth and fifth dorsal vertebræ in one of Pirogoff's vertical sections, with the space between the fourth and fifth ribs near the vertebræ in another of them, with that between the fifth and sixth ribs near the space in a third instance, and in a fourth with the lower border of the seventh rib at the same situation. In these four cases the position of the top of the pulmonary artery varied from the level of the middle of the second left cartilage to that of the lower border of the third.

The origin of the pulmonary artery was on a level with the body of the fourth dorsal vertebra in one instance, and with respectively the lower border of the fifth and the upper border of the sixth vertebra in two others of Pirogoff's vertical sections; and it was on a level with the top of the sixth vertebra or the cartilage above it in four of Pirogoff's cross sections.

The lower boundary of the body of the left ventricle, not including its apex, in three of Pirogoff's vertical sections was respectively on a level with the middle of the eighth, the top of the ninth, and the two upper fifths of the tenth dorsal vertebra.

The lower boundary of the body of the left ventricle was on a level with the upper border of the ninth vertebra in one of Braun's vertical sections, and with the cartilage between the ninth and tenth vertebræ in the other.

The section passed through the left ventricle a little above its lower border at the apex in Pirogoff's cross sections, in one instance on a level with the cartilage above the ninth vertebra, in another of them on a level with that vertebra, in two others with the cartilage below it, and in one on a level with the upper portion of the tenth vertebra.

The upper boundary of the root of the aorta, including its orifice, valve, and sinuses, at the attachment of the angle of junction of the anterior and left posterior flaps of the aortic valve, was situated in one of Pirogoff's vertical sections as high as the upper third of the fourth vertebra, in two of them it was in front of the sixth, and in one of them the upper portion of the seventh vertebra. The lower boundary of the root of the aorta, including the aortic orifice, valve, and sinuses, was on a level in two instances with respectively the middle and lower borders of the sixth vertebra, in one with the upper third of the seventh, and in one with the lower border of the eighth vertebra. In one of Braun's vertical sections the upper boundary of the root of the aorta was in front of the cartilage between the fifth and sixth vertebræ, and its lower boundary was in front of the cartilage between the sixth and seventh vertebræ; and in his other vertical section the lower boundary of the root of the aorta was on a level with the lower border of the seventh vertebra. The upper portion of the aortic valve, including the anterior and left posterior flaps and sinuses, was situated in three instances in front of the cartilage above the sixth vertebra, and the top and middle of that vertebra; in six instances in front of the top of the seventh vertebra or the cartilage above it; and in one in front of the body of that vertebra.

The lower portion of the aortic valve, or its right posterior flap, was situated in four instances in front of the middle or top of the seventh vertebra or the cartilage above it, and in one in front of the middle of the eighth vertebra.

The upper boundary of the mitral valve was situated in six of Pirogoff's vertical sections in front respectively of the middle of the sixth dorsal vertebra, the cartilage between the sixth and seventh vertebræ, the seventh vertebra, and in one instance the eighth; and its lower boundary was situated in three of his vertical sections in front of the eighth, and in one it extended down to the top of the lower third of the ninth vertebra. In one of Braun's vertical sections the mitral valve extended from the level of the cartilage below the sixth vertebra down to that of the upper third of the eighth, and in the other it extended from the cartilage below the seventh vertebra down to the upper third of the ninth vertebra.

The mitral valve was situated in front of the cartilage above the seventh dorsal vertebra in two of Pirogoff's cross sections, the seventh vertebra in probably nine of them, the cartilage between that vertebra and the eighth in two of them, and in front of the eighth vertebra in four of them.

The upper boundary of the tricuspid valve was situated in seven of Pirogoff's vertical sections on a level respectively with the upper and (in a case of ascites) lower borders of the sixth dorsal vertebra, the cartilage between that vertebra and the seventh, and the upper border of the seventh vertebra, the lower portion of the eighth vertebra, and the cartilage below it. The tricuspid valve in one of Braun's sections extended from the level of the top of the seventh vertebra to that of the middle of the eighth.

The tricuspid valve was on a level with the eighth vertebra in five instances, with the cartilage below it in two, and with the ninth vertebra in two.

MALPOSITIONS OF THE HEART.

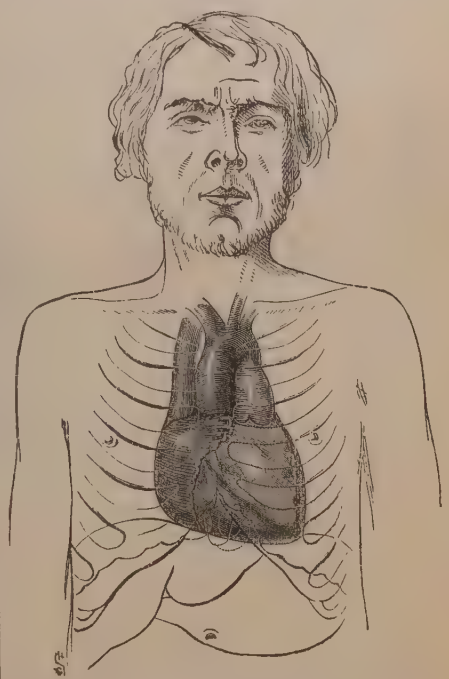
The displacements of the heart may be conveniently divided into the Vertical, Lateral, Forward, and Backward displacements.

The Vertical Displacements of the Heart.

Cases in which the Heart is Lowered.—The cause of the vertical lowering of the healthy heart is in all cases, with the exception of aneurisms of the arch of the aorta, an unusual lowering of the diaphragm. Pulmonary emphysema, bronchitis, and spasmodic asthma; croup, laryngitis, and laryngismus stridulus; collapse of the stomach and intestines; and aneurism of the arch of the aorta—all tend to lower the heart. To these may be added certain cases of mediastinal tumor, and pleuritic effusion into the left side during the middle period of its increase.

Pulmonary Emphysema, Bronchitis, and Spasmodic Asthma.—In Pulmonary Emphysema the right cavities of the heart and the pulmonary artery are greatly enlarged. The right ventricle often completely covers the left ventricle. The diaphragm is remarkably low, its standard position being often lower than it is in health at the end of the deepest possible inspiration. The enlargement of the lungs is so extensive that they cover the heart within the chest; and they are consequently everywhere interposed between the heart and the walls of the chest, with the exception of the border of the seventh costal cartilage (Fig. 73). The heart is invariably enlarged, the enlargement being almost limited to the right side. The venæ cavæ and right auricle are usually distended and of great size; the right ventricle is so much increased in volume that it almost or altogether conceals the left ventricle, its walls being hardened and hypertrophied; and the pulmonary artery is greatly increased in length and breadth. Notwithstanding the enlargement of the heart, its impulse is imperceptible over the walls of the chest; and in some cases its sounds are so muffled that they are scarcely audible over the usual cardiac region owing to the great development of the lungs in front of the heart. In no instance, however, is the heart absolutely covered by the dilated lungs. The central tendon of the diaphragm descends almost or quite to the level of the lower end of the ensiform cartilage, and necessarily draws downwards the enlarged heart. It is customary to speak of the displacement of the heart downwards as being caused by the expansion of the lung. This may be so in some cases, but as a rule the unusual descent of the heart, like that of the base of the lungs, is caused by the unusual descent of the diaphragm. The lower boundary of the right ventricle is brought downwards into the epigastrium, so that it is situated behind and to each side of the ensiform cartilage. In that position, and to the left of it, the heart is not covered with lung, and it is therefore in contact with the ensiform cartilage, with the neighboring margin of the seventh left costal cartilage, and with the intermediate abdominal muscles, the pericardium

Fig. 73.



Position of the heart and great vessels in Pulmonary Emphysema. The heart is displaced downwards, and is covered with the over-developed lungs. The apex-beat is imperceptible, but the impulse of the right ventricle is seen and felt in the epigastrium.

intervening. The result is that, as Dr. Stokes has pointed out, the impulse of the right ventricle may be felt in the epigastrium; and as the right ventricle is hypertrophied, "the heart may be felt pulsating with a violence that we would not expect from the examination of the pulse at the wrist, which is often small and feeble, while the impulses of the right ventricle are given with great strength."[1] The form of the chest, the great expansion of the lungs, the low position of the diaphragm, and the enlargement, elongation,

[1] Dr. Stokes on the Diseases of the Chest, p. 178.

and lowering of the heart and great vessels, all correspond, though to an exaggerated degree, with the condition of those parts at the end of the deepest possible inspiration in health. The presence of the impulse and sounds of the heart over the epigastrium, and their absence over the walls of the chest, are the signs that often first direct attention to the morbidly enlarged condition of the lungs.

In cases of severe bronchitis, the diaphragm is invariably lowered, the right cavities of the heart are enlarged, and the lungs are amplified. In those cases, therefore, as in emphysema, the heart is lowered, its impulse is obliterated over the intercostal spaces by the interposition of the lung, and the beat of the right ventricle is felt and seen in the epigastrium. The extent to which the heart is enlarged, lowered, and covered by lung is by no means so great in bronchitis as in emphysema.

When, as is often the case, the patient affected with emphysema is attacked by bronchitis, the extent to which the heart is lowered, and enveloped by the lungs is increased.

During an attack of spasmodic asthma, the diaphragm descends, the lungs are expanded to the utmost, and the impulse of the right ventricle is lowered into the epigastrium, just as in cases of true pulmonary emphysema. After the seizure is over, its effect upon the size of the lungs and the position of the heart does not immediately disappear. Gradually, however, the organs resume their healthy size and position. The asthmatic seizure that often attacks those affected with emphysema, is accompanied by an excessive amplification of the lungs and descent of the impulse; but in such patients the lungs and heart do not regain their normal size and position after the cessation of the attack, and in this important respect true spasmodic asthma is to be distinguished from the asthmatic seizure of a person affected with true pulmonary emphysema.

Croup, Laryngitis, Laryngismus Stridulus.—In all those cases in which there is excessive narrowing of the fauces, larynx, or trachea so as to contract the channels through which air is admitted into the lungs and render inspiration exceedingly difficult, the inspiratory efforts are laborious but ineffectual. Every muscle of respiration is brought into powerful action. The diaphragm descends as low as possible. The lungs are consequently lengthened and the heart is drawn downwards. As air, in spite of the labored breathing, can scarcely enter the air tubes, the lungs, being lengthened downwards, instead of expanding, collapse during inspiration, and the walls of the chest fall inwards. The lungs recede from before the heart, which is in immediate and extensive contact with the walls of the chest as well as with the ensiform cartilage. The heart is, therefore, in such cases to be felt beating with force over and to the left of the lower sternum and in the epigastrium.

Collapse of the Stomach and Intestines.—When the abdomen is unusually spare, the stomach and intestines being comparatively or quite empty, the abdominal organs shrink downwards, and the diaphragm is permanently lowered. This was well seen in the poor woman from whom fig. 74 was taken. She had been unable to swallow owing to cancer of the œsophagus for a fortnight before her death. Her emaciation was extreme. The stomach and intestines were absolutely empty of gas as well as of food. The liver, though not enlarged, had dropped downwards, so that its lower border rested on the bones of the pelvis. The diaphragm necessarily followed the liver and stomach in their descent, and as the result, the lungs at their base, and the heart where it rested on the diaphragm, were unusually lowered, and both organs were remarkably lengthened. The elongation of the ascending aorta and the pulmonary artery was very marked.

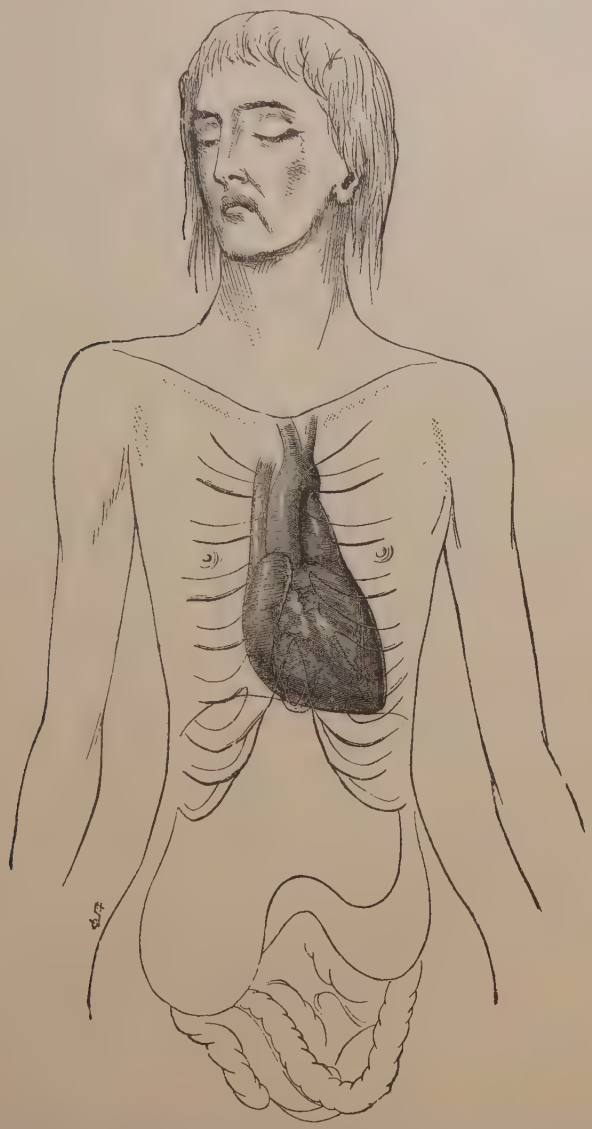
This was an extreme case, but in all instances of abdominal collapse, the diaphragm descends in exact proportion to the descent of those organs upon which it rests, and the lungs and heart are lengthened downwards to a corresponding degree. In some of those cases the transfer of the impulse from the intercostal spaces to the epigastrium may give rise to the suspicion of pulmonary emphysema on the one hand, or aneurism of the abdominal aorta on the other. In emphysema the chest is unduly developed, and the abdomen, instead of being retracted, is usually of more than average size. In aneurism of the lower thoracic or higher abdominal aorta, the impulse or pulsation in the epigastrium is strong during expiration, but it lessens and even disappears during a deep inspiration. In cases of abdominal collapse, it is the reverse, for the impulse in the epigastrium becomes lower and stronger when the patient takes a deep breath.

Aneurism of the Arch of the Aorta.—One would have expected *à priori* that aneurisms affecting the arch of the aorta, especially when they are of large size, would cause considerable displacement of the heart downwards. Dr. Townshend saw an instance of aneurism of the arch thrusting the heart downwards, so that it pulsated in the epigastrium.[1] I possess drawings taken from thirteen cases of aneurism of the arch of the aorta. In one of these the lower boundary of the right ventricle was situated more than an

[1] Cyclopædia of Medicine, ii. 391.

inch below the lower end of the sternum. In four there was effusion of blood into the left pleura, displacing the heart to the right. In the remaining seven instances the lower boundary of the right ventricle was from one-third to three-quarters of an inch below the lower end of the sternum. It is clear that in the majority of these

Fig. 74.



Position of the heart and great vessels in a case with *Collapse of the Stomach and Intestines*. The heart is displaced downwards, and covered with lung to the fifth cartilage. The apex-beat is present in the fifth space, and perhaps in the sixth, and the impulse of the right ventricle is seen and felt in the epigastrium.

cases, although the aneurism was in nearly all of them large, varying from three to five inches in diameter, the descent of the heart into the epigastrium was definite, but not proportionately great. In two of the instances there was cylindrical aneurism or dilatation of the ascending aorta. In these the transverse diameter of the aorta was only two inches, while its vertical measurement was four inches. They must, therefore, be included with the others in estimating the influence of aneurism of the arch of the aorta in displacing the heart downwards. The aneurismal sac displaces not so much the whole heart as those parts of

it upon which it makes immediate pressure, and which are subjected thereby to compression. This applies especially to the aneurisms of the ascending aorta, which amount to nine among my cases. In all of these the right ventricle, and in most of them the right auricle, were compressed from above downwards, the compression starting from a point at the top of the transverse furrow between those cavities, where the aorta comes into view. The difference in the vertical diameter of the right ventricle below the part in question and just below the pulmonary artery, amounted in one instance to two inches, the actual measurements being respectively three and five inches. As a rule the difference was much less, but this was mostly due to the right ventricle being compressed downwards in its whole breadth by the sac. In five of the cases the auricular appendix was displaced downwards and to the right.

The downward displacement of the apex in aneurism of the arch of the aorta is not considerable, being in fact mainly due to coexisting hypertrophy of the left ventricle. That condition, however, is not usual, except in those cases of cylindrical aneurism or dilatation of the ascending aorta, in which there is free aortic regurgitation, when the left cavity is greatly enlarged, and when the descent of the apex is much more due to that cause than to the aneurism.

Mediastinal Tumors.—Dr. Bennett gives a case of mediastinal tumor, which will be more fully noticed at page 449, in which there was considerable displacement downwards and to the right of the heart, which was seen and felt beating in the epigastrium.

Pleuritic Effusion into the Left Side.—In the middle period of these cases, when the fluid is steadily increasing, but has not yet reached to its height, there is displacement downwards and to the right of the heart, which may be felt beating in the epigastrium. A full account of such cases, and an explanation of their phenomena, will be found at page 443.

Cases in which the Heart is Raised.—Abdominal enlargement from gastro-intestinal distension, ascites, the presence of gas in the cavity of the abdomen, abdominal tumors, ovarian dropsy, aneurism of the abdominal aorta at the cæliac axis, and enlarged liver and spleen, all tend to elevate the heart. To these may be added certain cases of mediastinal tumors.

We have just seen that when there is collapse of the abdomen the diaphragm descends, drawing after it the heart and lungs. When there is distension of the abdomen, whatever be the cause, the reverse of this takes place. The diaphragm is raised, the cavity of the chest is shortened, and the heart and lungs are elevated and compressed upwards.

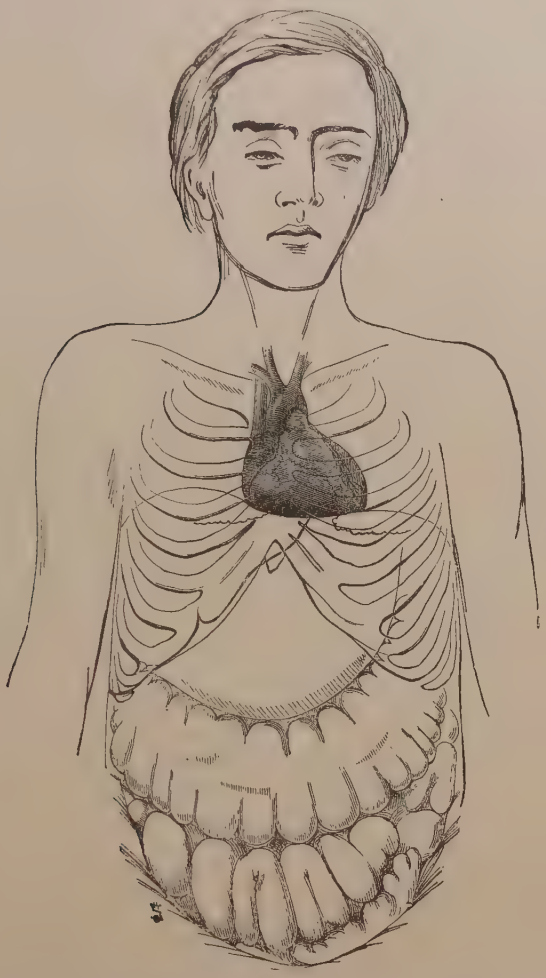
Distension of the Stomach and Intestines.—By far the most frequent, distressing, and often fatal cause of the elevation of the diaphragm and compression upwards of the heart and lungs, is the distension of the stomach and intestines with gas. The effect of this condition is well shown in Fig. 75, which was taken from a youth affected with diabetes, who, for months before his death, suffered from great abdominal distension. The cavity of the chest was materially lessened. The lower ribs, especially on the left side, were pressed outwards so as to restrain their movements, and the whole cage of the chest was elevated in front and at the sides. The heart and lungs were compressed upwards and lessened in size, so as to impede respiration and circulation.

When the abdomen is enlarged, it is enlarged in two directions, one outwards and downwards by the expansion of the walls of the abdomen, the other upwards by the elevation of the diaphragm. When the abdomen is extremely distended, the whole cavity becomes oval in form, or shaped like a balloon; the outer part of it presses outwards, and the upper part of it presses upwards. The cage of the chest is raised by this double movement of distension upwards and outwards. The wide irregular cone formed by the upper part of the swollen oval abdomen, acting upon the lower ribs, forces them asunder to the right and to the left, and lifts up the whole front of the cage of the chest. The more important effect of this distension of the abdomen is to lift up the diaphragm, and with it the heart at the centre of the chest, and the right and left lung on each side of it. When these organs are thus raised, as the walls of the chest in front of them, by which their relative position is measured, are raised also, the apparent elevation of the heart is much less than its real elevation. The heart and great vessels are compressed upwards, and displaced somewhat to the right, so that the heart takes a central position in the chest, while the great vessels often bear unduly to the right. The shape of the heart is altered. It is shortened from below upwards, and is proportionally though not actually widened. Its apex is especially tilted upwards, and instead of being, as in health, lower than the inferior boundary of the right ventricle at the end of the sternum, is higher than that point by from a third to one-half of an inch. It is to be observed that the heart and lungs are compressed upwards into the highest part of the cavity of the chest, and as that cavity is a cone narrowing from below upwards, those organs, to their great additional incon-

venience, are pushed up into the narrowest part of the space that they naturally occupy.[1]

Intestinal distension is usually present in peritonitis, and it becomes in many cases the most distressing symptom. As Dr. Stokes has shown, muscles are paralyzed by inflammation. The inflamed muscular coat of the intestines, being paralyzed, yields before the gaseous distension, which is no longer restrained by the peristaltic contraction of the intes-

Fig. 75.



Position of the heart and great vessels in cases with *Distension of the Stomach and Intestines.* The heart is displaced and compressed upwards, its impulse being present in the second and third spaces, and perhaps in the fourth.

tines. In peritonitis, abdominal respiration is suspended and the diaphragm is passive. It therefore yields without resistance to the upward pressure exerted upon it by the distended intestines, and the heart and lungs are compressed upwards to a greater degree than in those cases of abdominal distension in which the diaphragm retains its power. Distension of the stomach and intestines is very frequent in the dying. It was present to an excessive degree in either the stomach or intestines, or both, in 63 out of 122 dead bodies observed by me indiscriminately; and in 28 of these the stomach and intestines were very much distended. In such cases the abdominal distension, which is usually one of the

[1] For additional details as to this subject, see a lecture by the author on the "Influence of Distension of the Abdomen on the Functions of the Heart and Lungs," in the British Medical Journal for August 2, 1873, p. 108.

secondary effects of the original disease, produces compression of the heart and lungs, and thereby often hastens death or becomes its immediate cause. The introduction of the œsophageal tube from above, or of O'Beirne's tube from below, or the insertion of a small aspiration tube through the abdominal walls into the stomach, will in some of these cases give vent to the flatus and so produce material relief.

Many persons, especially those who have become stout, are subject to habitual distension of the stomach and intestines, with the effect of compressing the diaphragm upwards, curtailing its power to descend freely during inspiration, and so encroaching on the cavity of the chest. Those so affected do not, in many instances, suffer when they are at rest, but on any exertion respiration becomes hurried and difficult and the circulation of the blood is impeded. Such persons generally present themselves in two classes. One class complain of shortness of breath, the other of pain or distress in the heart when they make exertion, especially after a full meal. In many cases of angina pectoris, the distress is most easily excited after food. Some stout people are unusually subject to distress in breathing or in the heart, or both, from comparatively slight distension of the abdomen. In these persons the cavity of the abdomen is naturally incapable of great expansion owing to its walls being firm and resisting. The abdominal fulness, when it passes certain limits, cannot make way forwards and outwards, and the result is that the diaphragm is pushed upwards, and the lungs and heart are soon subjected to a distressing amount of pressure.

In dyspeptic persons, the most distressing symptoms induced by the fulness of the stomach after food are often referred to the heart. This is apt to be the case also whenever the stomach is greatly distended. The reason is obvious: the stomach is immediately subjacent to the heart, the diaphragm being interposed, so that the heart, in fact, rests upon the stomach. Whenever, therefore, the stomach is greatly swollen by an accumulation of gas and food, the heart is compressed upwards in an especial manner, and the distress experienced is often, therefore, almost limited to the heart. I do not, of course, lose sight of the additional physiological influence exerted by the stomach upon the heart through the medium of the eighth pair of nerves.

Ascites.—In ascites, the accumulation of the fluid is gradual. The patient is usually in bed, and the distress in breathing and in the heart experienced by the patient, owing to compression of the heart and lungs, is by no means proportionate to the amount of the distension. Indeed, those cases of ascites that suffer great distress in the organs of the chest usually have in addition distension of the stomach and intestines as well as enlargement of the liver. When this is so, a small amount of fluid in the peritoneal cavity will produce serious discomfort, and the removal even of a little of it by tapping will give immediate and unusual relief. Some years ago I had a patient in St. Mary's Hospital who was affected with aortic and mitral regurgitation. The heart was enlarged and the pericardium was adherent. He breathed with difficulty, owing to the great size of the abdomen, which was produced by the triple combination of great enlargement of the liver, distension of the stomach and intestines, and ascites. The quantity of urine was scanty, being about eleven ounces daily. The amount of fluid in the peritoneal cavity was small, but with the view of affording relief, tapping was resorted to. The intestines were so near the surface that an incision was made in the parietes of the abdomen, and the trochar and canula were introduced in a downward direction. At first only half a teaspoonful of fluid escaped, but by passing a female catheter through the canula, so as to press the intestines gently away from the end of the tube, about ninety ounces of serum were withdrawn. The relief to breathing was complete. The urine, before so scanty, now began to flow freely, and from fifty to eighty ounces were passed daily. By drawing off the fluid the extreme distension was relieved, and the ligature, so to speak, on the circulation, caused by the compression of the heart, was removed. Ultimately the fluid reaccumulated, and the patient died. The result was unfavorable, but the case was none the less instructive, for it demonstrated that the encroachment of the abdomen upon the chest checked the circulation of the blood, and so prevented the free secretion of urine.

In all cases of abdominal distension the seat of the impulse of the heart is a ready and exact measure of the extent to which the cavity of the abdomen encroaches upwards on the cavity of the chest. The progress of such distension, whether on the ascending or descending scale, may be exactly ascertained by noticing the varying position, upwards or downwards, of the impulse of the heart. It must, however, be borne in mind, that, when the heart and lungs are raised by distension of the abdomen, the walls of the chest in front of those organs is raised also, and that the apparent elevation of the heart, measured by its relation to the walls of the chest, is much less than its real elevation.

Escape of Gas into the Cavity of the Abdomen.—The escape of gas into the cavity

of the abdomen, owing to perforation of the stomach or intestines, produces rapid distension of that cavity and great elevation of the diaphragm and the heart and lungs, with the effect of inducing great distress in breathing and difficulty in the action of the heart.

Abdominal Tumors, even when they are of considerable size, rarely produce any material disturbance either in the action of the heart or in the performance of respiration.

Ovarian Dropsy.—The same may be said of cases of ovarian dropsy, even when the sac is of very large size, and rises upwards so as to encroach on the chest, unless that affection be accompanied by intestinal distension. In the female the walls of the abdomen are capable of great forward expansion, and the result is that large ovarian cysts as well as the gravid uterus at full time tend rather to protrude forwards so as to distend the abdominal parietes anteriorly, than to rise upwards so as to elevate the diaphragm and encroach upon the heart and lungs.

Simple Enlargement of the Liver and Spleen.—When the liver is universally enlarged, even when it assumes a very great size, it does not rise upwards, so as to raise the diaphragm and compress the heart and lungs, but it tends to grow downwards, so as to displace the stomach and intestines. The same may be said of the spleen in cases of leucocythemia, even when that organ attains to a very large size.

The result is, that simple enlargement of the liver or spleen does not as a rule encroach upon the chest so as to produce serious disturbance in the functions of the heart or lungs.

It is quite otherwise when the upper part of the right lobe of the liver is occupied by large abscesses or hydatid cysts or malignant growths. These morbid conditions produce a peculiar displacement of the heart upwards and towards the left subclavicular region, and I shall therefore consider them under the lateral displacements of the heart.

Mediastinal Tumor.—Dr. Bennett[1] gives a case of mediastinal cancer involving the bronchial glands and spinal column, in which the heart was found displaced, being drawn upwards. During life there was very little impulse to be felt or seen immediately to the left of the sternum just above the nipple.

The Lateral Displacements of the Heart.

Pleuritic effusion, empyema, and pneumo-thorax of one side of the chest; hemorrhage into either cavity of the chest from the rupture of an aneurism of the aorta; thoracic tumors; aneurisms of the arch of the aorta; aneurisms of the abdominal aorta at the cæliac axis; and large abscesses or hydatid cysts or malignant tumors in the upper part of the liver; all tend to displace the heart towards the side of the chest opposite to that which is affected. Contraction or cirrhosis of one lung with adhesions of the pleura tends to displace the heart towards the affected side. To these may be added lateral curvature of the spine and congenital transposition of the viscera.

The lateral or transverse displacements of the heart, which are sometimes called dislocations, unlike the displacement of the heart upwards by the encroachment of the cavity of the abdomen upon that of the chest, do not as a rule produce much distress in the heart itself or disturbance of the circulation. The lateral displacements of the heart are, however, valuable and decisive indications of disease, since by the evidence they afford they often render our diagnosis accurate and certain.

Pleuritic Effusion, Empyema, Pneumothorax.—The effusion of serum into either cavity of the chest, owing to pleuritis, acute or chronic, is the usual cause of the lateral displacement of the heart.

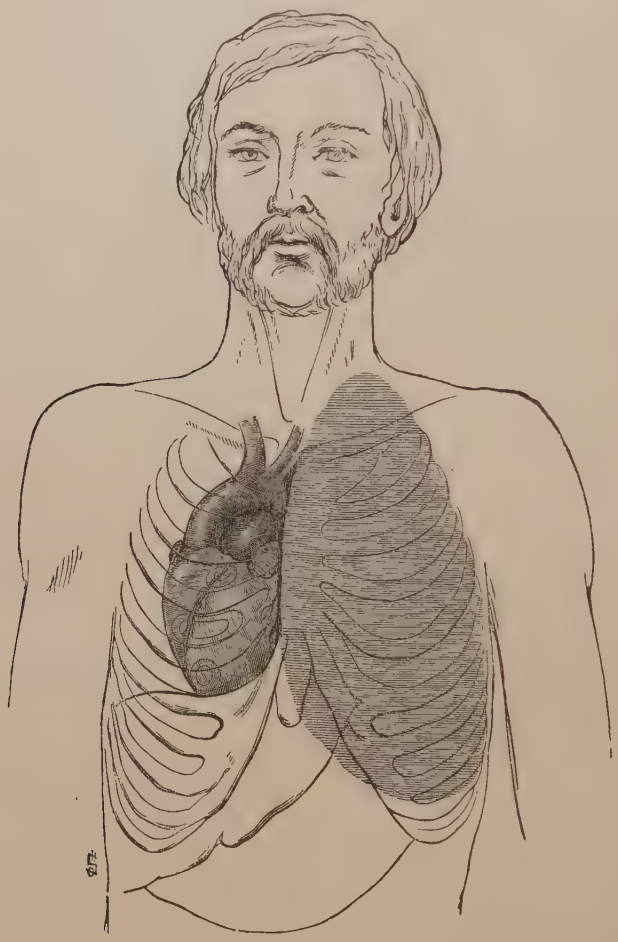
When extensive effusion takes place into the left side, the heart is pushed over towards or into the right side of the chest, as may be seen in fig. 76. This figure, unlike the others, does not represent an actual case, but is a diagram, made from drawings of six cases, one of effusion of serum into the pleura, one of empyema, and the four others of extensive effusion of blood into the left pleura from the rupture a thoracic aneurism. In one of these the clot measured three pints and a half.

The displacement of the heart from the increasing effusion of fluid into the pleura is usually gradual. It may, however, be rapid, and Dr. Walshe states that thirty-six hours will sometimes suffice for the heart's impulse to find its way beyond the right nipple. When the quantity of fluid is so small as to occupy only the back part of the left side of the chest, the heart is scarcely displaced. When the fluid increases the left ventricle and its apex are at first thrown a little forwards, and towards the centre of the chest. The pressure of the effused fluid is not made directly upon the heart, but upon the strong fibrous sac of the pericardium, and through its medium, upon the heart. If the heart had no sac of its own, and was present without restraint in, say, the left cavity of the chest, it would not be forced forward and to the right when the left cavity of the chest is filled with fluid, but it would, I consider, gravitate backwards owing to its own dead weight, and sink to

[1] Intrathoracic Tumors, p. 127.

the back of the cavity, just as the liver sinks to the back of the fluid in cases of ascites. The presence of the pericardium completely prevents such a state of things. The accumulated fluid distending the left cavity of the chest presses equally in every direction. It displaces the ribs backwards, forwards, and especially outwards, so that they draw the lower end of the sternum somewhat to the left; it displaces the left wing of the diaphragm, the spleen, stomach, and left lobe of the liver downwards and to the right; and it displaces the pericardium and the heart and great vessels inwards and to the right. The lower end of the pericardium at its

Fig. 76.



Position of the heart and great vessels in cases of *Pleuritic Effusion into the Left cavity of the Chest*. The heart is displaced into the right side of the chest, its impulse being felt in the third, fourth, and fifth spaces.

attachment to the central tendon of the diaphragm is stretched downwards by the traction upon it of the lowered left wing of the diaphragm to which it is attached by its central tendon.

The apex forms throughout the lowest part of the heart, and it describes a segment of a circle or arc as it sweeps round from its natural position in the left side of the chest to the position of extreme deviation to which it may attain in the right side of the chest. When the apex describes this curve, instead of being raised by the resistance offered by the abdominal organs, it is lowered during the first two-thirds of its course. The reason for this is obvious. The fluid in the left pleura, which displaces the pericardium and the heart to the left, displaces at the same time, as I have just explained, the left wing of the diaphragm and its central tendon and the subjacent organs downwards, forwards, and to the right. Under these circumstances, as the central tendon

forming the base of the pericardium is lowered, there is a free space downwards into which the apex of the heart, suspended from the arch of the aorta, necessarily drops, so that it may be felt beating in the epigastrium over, beyond, and even below the ensiform cartilage. At length, however, the heart, as it advances further into the right side, meets with increasing resistance from the solid convexity of the liver; and the heart, consequently, again rises, so that it is at length about as high on the right side as it is in health on the left. The displaced heart may indeed attain to a higher position if it deviate still farther to the right, when, as in a case of Wintrich's,[1] it may approach the axilla, and be felt beating from the second to the fourth spaces.

Information of some diagnostic value is to be obtained by observing the position of the heart in comparatively early stages in cases of pleuritic effusion, at a time when the impulse of the apex has already moved from its natural position and is on its way towards the central line. To quote Dr. Stokes, we observe, first, that the apex strikes in a situation about midway between its natural position and the upper portion of the ensiform cartilage.[2] It is not, however, until the apex beat presents itself in the epigastrium that much notice is taken of the altered position of the heart. In four of my cases of displacement of the heart towards the right from effusion into the left side of the chest, the apex presented itself in the epigastrium, being in one of these behind the lower end of the ensiform cartilage, and in two behind its middle. As Dr. Townshend remarks, in speaking of empyema in the left side, the heart is thrust from its natural position down into the epigastrium, where it may be seen and felt beating. There is no difficulty in distinguishing the impulse of the apex from that of the right ventricle in the epigastrium. When the latter is present the whole heart has been lowered, owing to the lowering of the diaphragm. This may occur, as we have already seen, in cases either of pulmonary emphysema, or croup, or with collapse of the stomach and intestines, when the presence of *pulmonary* resonance over the left side will at once enable us to distinguish the case. In cases of pleuritic effusion the existence of dulness, and in those of pneumothorax the presence of amphoric resonance, over the whole of the left side, and the absence of impulse to the left of the sternum, will generally suffice to make the case clear. Cancerous tumors occupying the whole of the left side may also give rise to displaced impulse and to general dulness on percussion, when that disease cannot be distinguished from pleuritic effusion or empyema on those grounds alone. In cases of pneumonia of the whole of the left lung, it is possible that owing to the enlargement of the pneumonic lung from consolidation and the development of the right lung to compensate for the disablement of the left lung, the impulse of the apex may disappear from the walls of the chest, while that of the right ventricle may descend into the epigastrium. In such cases, however, the impulse is comparatively slight, and it always extends rather to the left than the right of the ensiform cartilage, while in cases of pleuritic effusion the impulse is usually strong and marked, and tends rather to the right than the left side of that cartilage. As soon as the seat of the impulse disappears from the left side of the chest and extends to the right of the sternum, every difficulty of the kind just stated vanishes.

As the heart passes over from the left to the right side of the chest it gradually and necessarily turns over upon itself, hinging, so to speak, upon the vessels by which the heart is attached to the lungs and the system, so that the right auricle is hidden, all but the top of its appendix, and instead of the right ventricle being in front of the left ventricle, all but its left border, it is the reverse, for the left ventricle hides a large portion of the right ventricle (see Fig. 76). The part of the right ventricle exposed is, however, not that near the apex, but that near the pulmonary artery. The ascending aorta and pulmonary artery change their direction; they move to the right at their respective origins, but higher up they are retained in their places, the arch of the aorta at the end of its transverse portion, and the pulmonary artery at its bifurcation. The aorta and pulmonary artery, therefore, present not a front but a profile view, with a direction to the right.

I published a case with a diagram showing the position of the internal organs in the "Provincial Medical Transactions" for 1844 (p. 162), in which effusion in the left side of the chest was limited to the lower two-thirds of the cavity, owing to the upper lobe of the left lung being adherent down to the third rib. In this case the heart was simply displaced to the right, the front of the organ being still occupied by the right ventricle, and its right and left sides by the right auricle and the left ventricle. This case shows that the heart does not turn over upon itself so as to present the left ventricle instead of the right in front, unless the fluid presses upon the left side of the pericardium for its whole length, so as to bear upon the great vessels as well as upon the body of the heart.

[1] Krankheiten der Respirationsorgane.

[2] Dr. Stokes on the Diseases of the Heart and Lungs, p. 500.

The impulse to the right of the sternum is sometimes limited to the fourth and fifth intercostal spaces, while sometimes it is also present over the third and even the second space. In the latter case the impulse is double, and is due to the pulsation, followed by the second beat coincident with the second sound of the pulmonary artery or aorta, or both. When pulsation is present in the first, second, and third right spaces, and also in the normal position to the left of the sternum, the case is one of aneurism of the aorta; and the distinction of this impulse or pulsation from that of displaced heart presents therefore no difficulty.

Wintrich[1] states that sometimes, when the effusion is in the left side, the heart is displaced backwards (and to the right) being covered by lung, when the displacement of the heart can by no means be discovered. He saw one such case in which an able clinical physician mistook the disease for pericarditis with very great effusion.

When effusion of fluid takes place into the right cavity of the chest, the heart is displaced towards the left side. As the impulse, however, is already seated on that side, the change in position of the impulse of the heart is not nearly so marked or diagnostic as in cases in which the heart is displaced to the centre or right side of the chest, owing to effusion into the left side. Important information, however, is to be obtained in such cases from the position of the impulse on the left side.

In a patient under my care who had extensive effusion into the right pleura, the impulse was felt in the sixth space, two inches farther to the left, and somewhat lower than the natural position. In two cases of seropurulent effusion in moderate quantity into the right pleura, of which I possess drawings, the heart was displaced to the left, and lowered to a slight extent. In one the apex of the heart was situated behind the seventh rib, more than an inch to the left of the natural site, and nearly an inch lower. In the other, the displacement of the heart downwards and to the left also existed, but to a less degree.

Since the above was in type I have seen three cases of extensive effusion of fluid into the right side of the chest. In two of these cases the apex-beat was felt as far to the left as about the seventh rib, the position of the impulse being somewhat lower than natural. In the third case, a young woman, whom I saw through the kindness of Dr. Wane, the amount of fluid in the right side of the chest was very great. The impulse of the heart was not perceptible to the right of the mamma, but prevailed along its upper left border from the third or fourth to the seventh space where it was unusually low in situation. There was a double impulse over the great arteries at the left upper border of the mamma, and doubling of the second sound, the second of the two sounds being that made in the pulmonary artery. There was also a loud mitral murmur around the region of the apex. A large quantity of fluid was drawn off, by means of a glass syringe through a fine tube, by Mr. James Lane, who performed the same operation for the two other cases. I watched the position of the impulse when the fluid was being withdrawn, and noticed that it soon disappeared from the seventh space, and more slowly from the sixth, the beat moving steadily to the left and somewhat upwards. When the full amount of fluid had been withdrawn, the impulse was present in the fourth and fifth spaces, and perhaps in the third, being situated to the right of the mamma. The doubling of the second sound at once disappeared, and later I believe that the mitral murmur also vanished. In the drawing of an instance of great cylindrical dilatation or aneurism of the ascending aorta, in which there was considerable effusion of fluid in the right side of the chest, the heart, which was greatly enlarged and lowered in position, was displaced to the left as far as the ribs would allow, the apex extending to the seventh space, fully two inches below the level of the lower end of the sternum.

In two cases related by Dr. Gairdner[1] of effusion into the right pleura, the apex-beat in both was displaced to the left; in one (p. 329) the impulse probably retained its usual level, being displaced about one inch to the left. In the other (p. 354), before paracentesis, the apex-beat was felt in the fifth space, one inch and a half to the left of the normal site; after the operation it was present in the fourth space. In this case the impulse was probably lowered. Dr. Townshend, who was the first to observe the displacement to the left in such cases, felt the apex striking against the stethoscope between the fourth and fifth ribs in the axilla in two cases of empyema of the right side.[2] It is evident, then, that when considerable effusion takes place into the right side the apex-beat is always pushed further to the left, and that it is usually lower, sometimes on the same level as, and sometimes higher than the natural position. I attribute the lowered position of the impulse to two causes, the displacement downwards of the central tendon of the diaphragm by the effusion, and the inspiratory lowering of the diaphragm to

[1] Krankheiten der Respirationsorgane, p. 255.

[1] Clinical Medicine.

[2] Cycl. of Med. vol. ii. p. 38.

enlarge the left lung, and so to compensate for the disuse of the right lung.

I do not find that the displacement of the heart from empyema differs in any respect from that caused by the effusion of serum into the pleura.

In pneumothorax of the left side, the displacement of the heart is the same as in cases of fluid effusion into the pleura. In general, fluid is combined with the air in those cases, but air without fluid will produce displacement of the heart, and it must do so when it is in sufficient quantity to distend the sac of the pleura, press down the diaphragm, and so push the pericardium and the heart over to the opposite side. Dr. Douglas Powell[1] relates a case in which the right side of the chest was filled with air, and the right border of the heart was situated to the left of the left sterno-clavicular line.

Wintrich[2] states that displacement of the heart takes place in pneumothorax as in pleuritic effusion; the only difference being that in pneumothorax the heart is more frequently displaced from before backwards.

Hemorrhage into either Cavity of the Chest from the rupture of an aneurism of the aorta displaces the heart, as a rule, to the opposite side, in the same manner, and to the same extent, the quantity of fluid being alike, as in cases of pleuritic effusion. Two circumstances, however, tend to modify this result, one, the size and position of the aneurismal sac; the other, the lessening of the size of the heart that may be induced by the hemorrhage. Mr. Sidney Coupland[3] gives a case in which a diffuse aneurism of the thoracic and abdominal aorta ruptured into the left cavity of the chest, which contained twenty-four ounces of clot. During life the apex was tilted upwards, and was felt beating in the fourth space, one inch within, and on a line with the left nipple.

Contraction or Cirrhosis of the Lung with Adhesion of the Pleura.—When pleuritis with effusion, whether chronic or acute, ends in the permanent condensation of the lung, fibroid thickening of the pleura, and binding adhesions, the whole of the affected side contracts and the ribs are crowded together. That side of the chest, however, is not obliterated; it is still much larger than the condensed lung, and the result is that if, for instance, the right be the affected side, the heart is permanently drawn over into the right side.

Dr. Stokes was the first to draw attention to the displacement of the heart to the right side, in consequence of the absorption of an effusion into the right pleura.[1]

When the left is the affected side, the heart may be drawn quite over into the left side, the right auricle being situated to the left of the median line. This is well seen in Fig. 77, which was taken from a man in whom, owing to the complete contraction of the left lung, the heart entirely occupied the left side of the chest in front, no portion of the left lung being interposed between the heart and the walls of the chest. The heart is raised towards the infra-clavicular region and the axilla, and the ribs fit closely upon the heart from the second to the fifth. In this man the impulse must have extended from the first intercostal space to the fourth.

It may be observed that here also, as in displacement of the heart into the right side, the heart revolves upon itself and turns over, but in the reverse direction. In displacement into the right side, the left ventricle and auricle are situated in front, the right ventricle being partially and the right auricle all but its tip being wholly concealed. In displacement to the left, the right ventricle entirely hides the left side of the heart. The aorta and pulmonary artery are twisted to the left, both venæ cavæ are completely exposed when the right lung is turned aside, and are situated behind the sternum, and the whole heart seems to turn to the left upon the two venæ cavæ as upon a hinge or pivot.

In cirrhosis of either lung the heart is drawn towards the affected side. Dr. Hilton Fagge[2] relates a case of cirrhosis of the right lung in which the impulse was seen and felt two inches below and one inch to the left of the right nipple. The heart deviated more to the right during life than after death, when the apex was two inches to the left of the middle line, being situated between the fifth and sixth (cartilages); and one-half of the heart was to the left, and one-half of it was to the right of the middle line. Dr. Greenhow[3] gives a case of contraction of the right lung, the precise condition of which was unknown, observed by him during life, in which the heart was displaced very far to the right and upwards, and was felt beating in the third and fourth spaces over an area of three inches by three and a half, of which the right nipple formed the central point.

Dr. Wilks[4] communicates a case of cirrhosis of the left lung, in which that lung was contracted and hard, and had to be

[1] Path. Trans. xix. 77.
[2] Krankheiten der Respirationsorgane, pp. 344, 347.
[3] Path. Trans. xxiv. 54.

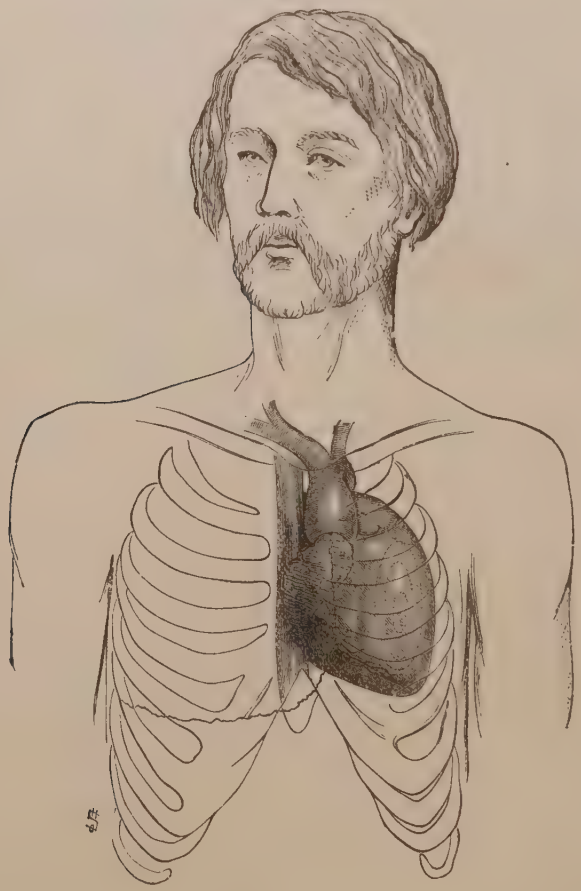
[1] On the Diseases of the Chest, p. 501.
[2] Path. Trans. xx. 35.
[3] Ibid. xix. 159.
[4] Ibid. viii. 39.

cut out. The right lung was enlarged, and was the only organ observable on removing the sternum. The heart was drawn towards the left side, "owing to the pericardium being firmly united to the pleura."

Dr. Bastian[1] gives an analysis of thirty cases of cirrhosis derived from various sources. The heart was much displaced towards the affected side in twelve of these, and slightly in three; while in three of them there was no displacement, and in the remaining twelve there was no notice of the position of the heart.

When the left bronchial tube is obliterated by compression, by its own con-

Fig. 77.



Position of the heart and great vessels in a case with *Contraction of the Left Lung*. The heart and great vessels are drawn completely over into the left side of the chest, so that it is much farther to the left and higher in situation than in the healthy chest. They are partially covered by the right lung, but not at all by the left, and the impulse of the heart is present in the second, third, and fourth spaces, and perhaps in the fifth.

traction, or by the admission of a foreign body, the left lung shrinks, the left side contracts, and the heart is displaced towards the clavicle and axilla, exactly as in cases of complete contraction with adhesions of the left lung. Dr. Stokes publishes a case of Dr. Mayne's of aneurism arising from the front of the transverse portion of the arch of the aorta, which extended downwards towards the left lung, compressing and flattening the left bronchial tube. The left side of the chest was less than the right by two inches, the ribs were crowded together, and the heart was displaced towards the left axilla.[1]

There are many cases of partial contraction of a portion of the upper lobe of the left lung, whether from phthisis, cirrhosis of the lung, gangrene of the lung, or other cause, in which the upper part

[1] Path. Trans. xix. 47.

[1] Dr. Stokes on Diseases of the Heart and Aorta, p. 566.

of the heart and the great vessels, especially the pulmonary artery, are drawn upwards and to the left towards or into the former seat of the contracted portion of the lung. In such cases the presence of the pulmonary artery, elevated in position and drawn to the left, may be immediately ascertained by its peculiar double impulse. I cannot say that I have strictly observed the analogous displacement of the ascending aorta towards the seat of the upper lobe of the right lung, in cases of contraction of that lobe, but I have noticed cases of this class in which the vessel evidenced itself by very loud superficial first and second sounds, which communicated themselves to the ear, if not to the hand, like a double shock or impulse. Dr. Stokes has given an interesting account of the displacements of the heart from the diminished volume of the lung, in his work on Diseases of the Heart, p. 458.

Intra-thoracic Tumors.—Large cancerous growths in the cavity of the chest, when they press upon the heart without penetrating into its structure, necessarily displace it in the direction of the pressure. The heart is simply pushed aside by the tumor, and its displacement is in no way influenced by the relation of the heart to the central tendon of the diaphragm.

"In the year 1856 I saw," writes Dr. Cockle, in his paper on intra-thoracic cancer, "a case of intra-thoracic cancer occupying the whole of the left side of the chest, and encroaching slightly on the right side, in which the tumor carried the heart before it as far as the right nipple. The impulse was felt pulsating between the second and third ribs, and down to, and at a later period beyond, the right nipple."

Dr. Bennett[1] relates the case, communicated to him by Dr. Sutton, of a little girl, in whom the entire left side was occupied by a mass of medullary cancer which had pushed the heart considerably to the right. During life the heart was displaced and was felt beating at the right nipple. The diagnosis was "*very great effusion into the left pleural cavity*," and the chest was twice punctured.

In a case published by Dr. Andrew,[2] in which a large malignant growth occupied the upper lobe of the left lung, the heart was displaced downwards and to the right. Dr. Bennett[3] gives a case of cancer of the anterior and posterior mediastinum involving the anterior portion and root of the right lung on which the heart was pushed downwards and towards the right side, so that rather more than half of the organ was to the right of the median line. A fortnight before death there was manifest and considerable displacement of the heart, which was beating in the epigastrium. Dr. Douglas Powell[1] relates a case in which the left cavity of the chest was occupied by a solid mass, displacing the heart to the right, and the lung posteriorly. After death it was found that this tumor was intimately connected with the heart at its left and posterior aspects. I might cite other cases of intra-thoracic tumor, published by Dr. Townshend, Boerhaave, quoted by him, and others, in which the heart was displaced.

On the other hand, cases are recorded in which there was little or no marked displacement of the heart, although the extent of the disease was great.

Dr. Graves and Dr. Stokes[2] have published a well-known instance of this disease, in which there was found, in place of the right lung, a solid mass, weighing more than six pounds. It encroached upon the left side of the chest, enveloping and nearly concealing from view the pericardium, great vessels, and trachea. Notwithstanding the extent and position of the disease, the heart pulsated in its natural situation.

Dr. Wilks describes a case in which the whole right lung was converted into one mass of medullary cancer, which protruded into the pericardium, ran along the great vessels at the base of the heart, and pierced the auricles of the organ itself. The superior cava was almost destroyed by the cancer, the inferior vena cava was closely surrounded by it but was free, the right pulmonary artery was a mere slit in the midst of it, and it had entered the heart through the pulmonary veins. There is no notice of displacement of the heart, although it is stated that the sounds of the heart were very feeble.

Dr. Quain[3] exhibited before the Pathological Society an encephaloid mass of the size of a large cocoa-nut, which was situated between the root of the left lung and the heart. When the patient was first seen, six weeks before his death, the heart was little displaced. Afterwards effusion took place into the left side, and the heart became much displaced towards the right side.

It is evident from these cases, that a large intra-thoracic tumor occupying one side of the chest may in some instances displace the heart into the opposite side, while in other instances, in which the tumor is equally large, there may be no displacement of the heart whatever. The reason is obvious. In those instances in

[4] Intra-thoracic Growths, p. 100.
[2] Path. Trans. xvi. 51.
[3] Loc. cit. p. 92.

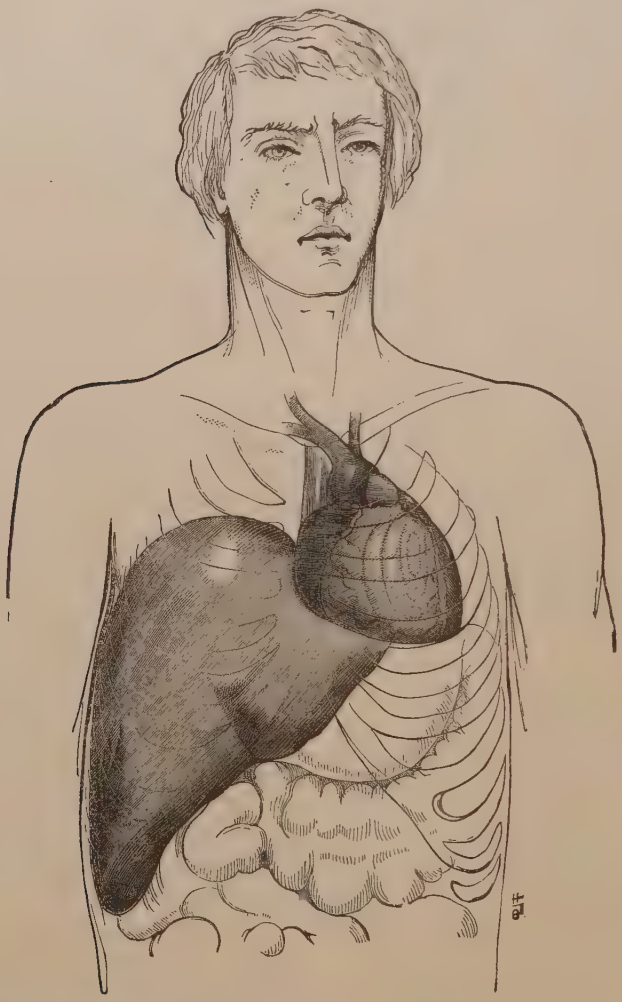
[1] Path. Trans. xxiv. 28.
[2] Dr. Stokes on the Diseases of the Chest, p. 371.
[3] Path. Trans. viii. 54.

which there is no displacement, the cancer penetrates or surrounds the organ, without pushing it aside.

It is evident, then, that the displacement or non-displacement of the heart, and the mode and extent of its displacement, in instances in which there is complete dulness of one side, may sometimes help us to discover whether the case is one of intra-thoracic cancer or of simple effusion into the pleura.

Large abscesses, hydatid cysts, or malignant tumors in the upper or convex portion of the Liver.—The patient from whom Fig. 78 was taken was affected with jaundice. On post-mortem examination several large abscesses were found in the upper portion of the liver, where it ascends into the right side of the chest. He also had peritonitis, and excessive intestinal distension. The whole diaphragm was raised, and with it the heart was pushed upwards

Fig. 78.



Position of the heart and great vessels in a case with *Large Abscesses in the Upper portion of the Liver.* The heart and great vessels are displaced extensively upwards and to the left towards the left axilla, so as completely to occupy the left side of the chest. The impulse is present in the second and third left spaces.

and to the left in a remarkable manner. The liver encroached upon the right side of the chest to such an extent that its highest point was on a level with the lower edge of the second rib. The convexity of the liver consequently encroached on the left side of the chest as well as the right, and carried the heart, resting upon its upper surface, completely over into the upper portion of the left side of the chest.

If this figure be compared with Fig. 75,

in which the diaphragm is excessively raised by means of distension of the stomach and intestines, it will be seen that while in both the diaphragm is raised to an excessive degree, there are important points in which they differ materially from each other. In that figure as well as in this we find that the abdomen is distended, the diaphragm is pushed upwards, the lower ribs are prominent, and the heart and lungs are pressed upwards and lessened in size, being encroached on by the abdominal organs. In universal distension of the abdomen, the heart, while it is compressed upwards, retains a central position, as it rests on the central tendon of the diaphragm. It deviates rather to the right than to the left. But in those cases in which there are large abscesses or hydatid cysts, or cancerous growths in the upper portion of the liver, the heart, as it is pushed upwards, deviates extensively to the left, and occupies a space to the left of the upper half of the sternum, behind the first, second, third, and fourth ribs. It is to be remembered that in this case there was peritonitis and great intestinal distension, consequently the compression of the heart upwards was effected by a double cause.

The deviation of the heart to the left side of the chest from extensive abscesses in the upper portion of the liver, differs thus from the deviation caused by effusion of fluid into the right side of the chest—in effusion into the right side of the chest, the heart and the impulse at the apex are either lowered or only slightly raised; while in cases with abscesses in the upper portion of the liver they are pushed upwards, being above the fourth rib. The position of the heart in enlargement of the liver from abscess, and in great contraction and adhesions of the left lung, corresponds very closely. (Compare Figs. 77 and 78.) In both the heart and great vessels are situated behind the second and two or three upper ribs, in both the heart is pushed entirely into the left side, the venæ cavæ being behind the sternum. But in the following respects they differ. In enlargement of the liver from abscesses, the anterior aspect of the heart is unchanged; the left upper ribs are widened apart and the ribs on both sides are raised and pushed outwards; the dulness on percussion is more extensive on the right side than the left, especially behind, and the heart and its impulse scarcely appear below the fourth rib. In contraction of the left lung, these conditions are reversed. The heart turns upon the venæ cavæ as upon a hinge over towards the left, the right auricle and both venæ cavæ being completely exposed, and the left ventricle being hidden by the right; the ribs are crowded together, the whole of the left side of the chest being contracted; there is dulness on percussion over the whole left lung, while the whole right side of the chest is very resonant, the area of resonance being increased, owing to the encroachment of the right lung upon the left side of the chest to the left of the sternum; and the impulse of the heart is felt down to the fifth rib.

Extensive effusion in the pericardium in *acute* pericarditis is an additional cause of displacement of the heart towards the axilla. Of this displacement I shall speak in the article on pericarditis.

Displacement of the Heart Forwards.

Dr. Hope relates a case in which the thoracic aorta, extending from an inch below the left subclavian artery down to the diaphragm, was enlarged into an aneurismal sac which lay across the spine, and projected on the right side three inches beyond the vertebræ without reaching the ribs, while on the left it extended to the ribs, causing destruction of three and caries of two or more of them, and at last formed a considerable tumor on the back. This tumor necessarily compressed the heart forwards against the front of the chest. The impulse of the heart was exceedingly vigorous, and was double, consisting of a diastolic as well as a systolic impulse, each of a jogging character. It was agreed that there must be considerable hypertrophy of the heart to account for so strong an impulse, and yet the organ was found by Mr. Cæsar Hawkins, who drew up the autopsy, only "slightly enlarged and thickened."[1] Dr. Hope quotes without reference, a case mentioned by Dr. Todd, in which the heart was pushed forward and outwards, and, as it were, compressed against the ribs by an enormous aneurism of the thoracic aorta. The sounds of the heart were so modified by this compression as to lead to the erroneous diagnosis of concentric hypertrophy.

I possess a drawing taken from a case of extensive aneurism of the abdominal aorta at the cœliac axis, in which the aneurismal sac extended upwards, behind the diaphragm, in front of the lower dorsal vertebræ, so as to displace the heart forwards and probably somewhat upwards.

Displacement of the Heart Backwards.

When abscesses or tumors form in the anterior mediastinum, behind the lower

[1] Dr. Hope on the Diseases of the Heart, p. 447.

portion of the sternum, the heart must be displaced backwards.

The displacement of the heart backwards is also induced by the very extensive effusion that gradually takes place into the pericardium in cases of chronic pericarditis.

Wintrich states, as we have already seen, that sometimes when there is pleuritic effusion in the left side, the heart is displaced backwards and to the right, so that its displacement can by no means be discovered.

LATERAL OR PARTIAL ANEURISM OF THE HEART.

By Thomas Bevill Peacock, M.D., F.R.C.P.

Under this term it is proposed to treat of the partial or lateral sacculated dilatations, in contradistinction to the general enlargements of the cavities of the heart, to which, and especially in France, the term aneurism has also been applied. The partial aneurisms differ, however, from the latter forms of the disease, not only because they involve only a portion of the parietes of the cavity, but also in that the structure of the muscular walls is always more or less altered in the seat of disease.

The real aneurismal tumors affect only the left cavities of the heart, the left ventricle and auricle, or the corresponding arterial and auriculo-ventricular valves. The immunity thus possessed by the right cavities has been variously explained by different writers. Breschet, who thought that the aneurismal dilatation was almost always, if not invariably, situated near the apex of the left ventricle, and that its production was due to the laceration of the inner portions of the ventricular walls, supposed that the non-occurrence of the disease in the right ventricle was owing to the greater relative power of its walls at the apex. Dr. Thurnam referred the freedom of the right ventricle from disease to the peculiar action of the valves at the right auriculo-ventricular orifice, by which, when the ventricle becomes distended, the aperture is incompletely closed so as to allow the reflux of the blood into the right auricle. He also contended that the term aneurism should be restricted to the dilatations of the cavities of the heart through which arterial blood circulates; while the term varix should be applied to the similar enlargements of the venous cavities, so as to maintain the analogy between the affections of the two sides of the heart and those of arteries and veins. Rokitansky considers the dilatations of the right side of the heart as not truly aneurismal, and ascribes the occurrence of the real aneurisms only on the left side to the greater frequency of endocarditis in that situation. There seems good reason to believe that the proneness to inflammation of the lining membrane of the left cavities, is mainly influential in causing the occurrence of aneurism on the left and not on the right side of the heart; but it is also probable that the greater tension to which the walls of the left ventricle are exposed, with the variations of pressure exerted by the column of blood in the arteries, materially conduces to the disease. Certainly when from any cause any portion of the parietes is rendered less resistant and more readily expansible, the pressure of the blood will tend rapidly to expand the weaker part so as to form a distinct sac.

In the following notice I shall treat first of aneurisms of the left ventricle, then of those of the left auricle, and lastly of valvular aneurisms.

ANEURISM OF THE LEFT VENTRICLE.

The occasional occurrence of partial aneurismal dilatations of the heart similar to those which are of such frequent occurrence in the arteries, was first shown by the case recorded by Galeatti in 1757; and it is a curious coincidence that in the same year the condition was brought to the knowledge of John Hunter by the occurrence of a case, the preparation of which is contained in the Museum of the Royal College of Surgeons, and of which the description was found by Dr. Thurnam[1] in his MS. Catalogue. In 1759 a case of the kind occurred to Walter, which was published in 1785,[2] and in 1793 an-

[1] Med.-Chir. Trans. vol. 21, 1838.

[2] Nouv. Mém. l'Acad de Berlin.

other specimen preserved in Dr. Hunter's Museum, was described by Dr. Baillie and figured by him in the plates which appeared in 1799. Corvisart met with a case in 1796, which was published in 1806. Hodgson described one in 1815,[1] Zannini in 1816,[2] Rostan in 1820,[3] and Shaw in 1822.[4] Sir A. Cooper, in his Lectures published by Tyrrell in 1825, said that he had met with three cases of which two were contained in the Museum of St. Thomas's Hospital. In 1827 the first memoir on the subject was published by Preschet,[5] in which the particulars of ten cases were collected, including one communicated to him by Cruveilhier in 1816, two by the Berards which appeared in a Paris Thesis, and one by Dance, together with the case of the celebrated Talma and the description of a specimen in the museum of the Faculty by Breschet himself. In the same year two other cases of the kind were described by Adams in Dublin,[6] and by Johnson in this country.[7]

In 1830, Dr. Elliotson, in his Lumleian Lectures, described another case, of which the preparation is now in the Museum at St. Thomas's, and referred to sixteen cases as on record at that time. In 1829 two additional cases were narrated by Bignardi and Reynaud,[8] in 1832 a third was published by Hope, and in 1833 a fourth by Lobstein. In 1834 a notice of the subject was given by Ollivier,[9] in which he referred to the cases collected by Breschet, together with those of Adams, Bignardi, and Reynaud. In 1835, Dr. Thomas Davies referred to the disease, and stated that there were two specimens in the Museum of the late Mr. Langstaff. In the same year, Bouillaud treated of the subject in a section of his work, detailing the more important observations recorded by Breschet and Ollivier, with two more recently published cases by Choisy and Petigny. In 1838 Dr. Thurnam contributed a memoir to the Medical and Chirurgical Society,[10] which was then completely exhaustive of the subject and still leaves little to supply, and affords the best description of the pathology of these affections which has appeared. In this memoir he related seven new cases, of which three were drawn from the MSS. of John Hunter in the possession of the Royal College of Surgeons. He further referred to five other specimens previously undescribed, which he had found in different museums. In 1842 a short notice of the subject was published by Rokitansky, in his work on Pathological Anatomy; and in 1843, Dr. Craigie contributed to the Edinburgh Medical and Surgical Journal a valuable memoir,[1] detailing the particulars of twenty-two of the cases up to that time recorded, all of which had, however, been previously referred to by Dr. Thurnam, together with a very interesting example which had occurred in his own practice. In 1846 a case was described by myself;[4] in 1850, Dr. Halliday Douglas[3] related the particulars of four cases; and in 1852, M. Cruveilhier discussed the subject in his Pathological Anatomy, illustrating his views by reference to various examples which had fallen under his own notice.

Since the publication of Dr. Thurnam's memoir, numerous observations have been placed on record, so that I have had no difficulty in collecting forty-three fresh cases, together with brief notices of others not fully reported. Of this number fourteen are contained in the Bulletins of the Société Anatomique of Paris, two in the Mémoires of the Société de Biologie, and sixteen in the Transactions of the Pathological Society. With the cases collected by Dr. Thurnam, fifty-eight in number, those on record must at present exceed one hundred, and I have seen references to several others, the particulars of which I have not been able to obtain.

Nature and Mode of Origin.—Breschet, as the name, *false consecutive aneurism*, which he gave the affection, indicates, regarded the real aneurisms of the heart as originating in rupture or ulceration of the lining membrane of the ventricle and some portion of the muscular walls, the result of softening from inflammation or atheroma. Reynaud showed that in his case the dilatation originated in disease of the endocardium; and Cruveilhier pointed out that in some cases the whole of the structures of the ventricular walls were dilated,—and apparently in consequence of the muscular fibres having undergone conversion into a fibroid structure, which was less resistant to pressure and more readily admitted of expansion.

Dr. Thurnam to some extent adopted the views of the pathologists who had preceded him, and contended, that, while the aneurisms did in some cases originate in rupture or softening of the lining membrane and muscular walls of the ventricle, they more frequently were connected with

[1] Diseases of Arteries and Veins, p. 84.
[2] Italian Translation of Baillie's Morbid Anatomy.
[3] Sur les Rupt. du Cœur, Obs. v.
[4] Manual of Anatomy, vol. i. p. 251.
[5] Rep. Gén. d'Anat. tome 3me p. 181.
[6] Dublin Hospital Reports, vol. iv.
[7] Med.-Chir. Rev. vol. xv.
[8] Journal Hebd. de Méd.
[9] Dict. de Méd. tome viii. p. 303.
[10] Transactions, vol. xxi. In Dr. Thurnam's paper references will be found to all the cases here named, published up to the period of its appearance.

[1] Vol. lix. p. 381.
[2] Edin. Med. and Surg. No. 169.
[3] Monthly Jour. of Med. Sc.

the changes in the endocardium and muscular substance pointed out by Reynaud and Cruveilhier, and consisted in dilatations of the whole of the structures constituting the parietes of the ventricle. He also thought that these changes were probably often referable to inflammation, and that in some cases the formation of coagula in the cavity of the ventricle might cause the expansion of the ventricular wall in the seat of deposition. He further contended that the aneurisms of the heart presented all the several forms which are met with in similar affections of arteries. Rokitansky regards the aneurisms of the heart as always depending upon inflammatory processes, either of an acute or chronic character. In the first or acute form of the affection, the disease originates in recent inflammation of the endocardium and probably also of the contiguous muscular substance, and the consequent laceration or breaking down of the inflamed surface under the pressure of the blood. In the other variety, the dilatation is the more remote result either of inflammation of the endocardium and a somewhat thick layer of the muscular substance, or of the whole thickness of the wall of the ventricle during endo- and peri-carditis. In this form the muscular fibres become replaced by fibroid structure, the endo- and peri-cardium are blended with the altered tissue, and the parietes become expanded under the pressure of the blood. The first of these forms corresponds therefore with the false consecutive aneurism of Breschet; the second with the true aneurism of Reynaud, Cruveilhier, and Thurnam. While adopting Rokitansky's views as to the inflammatory origin of the cardiac aneurisms, there is no reason to deny the correctness of the analogy contended for by Dr. Thurnam, between their various forms and the different varieties of arterial aneurisms. It is, however, very doubtful how far the coagulation of the blood in the cavities of the heart gives rise to partial dilatation. Such coagula form, it is well known, chiefly on the right side, in which the aneurismal dilatation does not occur; and the clots which Dr. Thurnam has described and figured, might as probably have originated in the already dilated part as have given rise to the dilatation.

It is obviously only by the examination of incipient aneurismal sacs, or those of small size, that we can form a correct judgment as to their original modes of development. Confining his assertion only to such cases, Dr. Thurnam states that of twenty-eight out of the fifty-eight cases which he collected, twenty-two originated in dilatation of the structures entering into the composition of the walls of the heart; while in six there was solution of continuity of the endocardium and inner stratum of muscular fibres. Of the forty-three cases which I have myself collected, in thirteen the data are imperfect or the disease is very far advanced; of the remaining thirty, in twenty-five the sac was lined by endocardium, which is stated to have been opaque, thickened, indurated or ossified in eleven cases;—and in four the lining membrane was destroyed. In sixteen of these cases the subjacent muscular structure had undergone the fibroid degeneration and was more or less attenuated, in one of them to such an extent as to present only a trace of the altered tissue; in five the muscular substance was thinned but not otherwise altered; and in seven cases it was wholly wanting and the sac was only bounded by the endo- and peri-cardium. Both series of facts, therefore, show that in the cases in which satisfactory opinions as to the mode of origin of the sacs can be formed, they are usually at first of the true form, or that in which all the structures are expanded.

From several specimens which I have had the opportunity of examining, either in the recent state or as preparations, the following may be stated to be the progressive changes in the development of the true aneurisms.

1. In the earliest stages in which the affection can be recognized, we observe thickening and opacity of the endocardium, with slight dilatation of the corresponding portion of the walls of the ventricle, and attenuation of the muscular substance without any marked alteration of its texture.

2. In a more advanced stage there is thickening and opacity of the endocardium, and conversion of a more or less thick stratum of the muscular substance into a dense, yellowish or whitish colored fibroid tissue intermixed with the muscular structure. The parietes of the ventricle in the seat of disease have become more atrophied, and the cavity presents a more marked dilatation.

3. At a still later period, together with the thickening and opacity of the endocardium, this membrane becomes intimately blended with the subjacent tissue, so as to be no longer separable from it. The muscular substance throughout the whole or the greater part of the thickness of the ventricular parietes, is converted into dense, pale-colored fibroid tissue. The attenuation of the walls of the ventricle is greater, and the dilatation of the corresponding portion of the cavity, if occupying the outer surface of the heart, occasions a more or less marked prominence externally.

While these changes are in progress in the parietes of the ventricle, others are proceeding in its interior. The dilated portion of the cavity becomes, especially

if it be somewhat circumscribed and bounded by a tolerably defined margin, the seat of coagula. These are at first thin, loose, and dark colored, subsequently they become more firm and paler; and at length the sac is found more or less completely filled by coagula, of which the outer portions are distinctly laminated and decolorized, and often adherent to the altered endocardium. As the partial dilatation of the ventricular cavity increases and forms a more or less decided prominence externally, the visceral pericardium becomes implicated in the disease. At first it is only slightly opaque and presents a rough surface, from the existence of small granular concretions of fibrine; these become thicker and coalesce, and finally constitute a distinct layer of false membrane; and at length adhesions are formed between the visceral and reflected pericardium over the seat of the aneurism, or more rarely uniting the whole or a large portion of the membranes; often also, when there are not entire adhesions, the surface of the heart displays large white patches. In the cases in which there are evidences of the existence of more general pericarditis, it seems probable that the alterations in the ventricular walls upon which the aneurismal dilatation depends, have, as stated by Rokitansky, proceeded from without, and have involved the inner portions of the parietes secondarily.

With the gradual expansion of the aneurismal sac, the lining membrane and part or the whole of the muscular layers may be eroded or destroyed, so that the cavity may come to be bounded by the pericardium, with or without a portion of altered muscle; the aneurism thus assuming the "*false consecutive*" form.

Pathologists have within the last few years described the occurrence of fibrinous deposits in the walls of the heart. The Pathological Transactions contain various instances of the kind, originating either in acute inflammatory action, or resulting from an altered condition of the blood. In some cases this fibrinous material may undergo imperfect organization, giving rise to the fibroid degeneration of the muscular tissue which, as above shown, so frequently precedes the formation of the true aneurisms. In other cases the deposit breaks down and destroys the involved tissue, so as to give rise, under the pressure of the blood, to a kind of sac, to which the term "*false aneurism*" may be applied.

It should, however, be stated that Rokitansky regards the acute or originally false form of aneurism, as of decidedly less common occurrence than that in which the whole of the tissues are expanded, and his conclusions are confirmed by the observations of others. Various cases originating in the former mode are, however, on record. One such was reported by Dr. Pereira, in which the cavity was situated at the base of the septum of the ventricle; and another is related by Mr. Shillitoe and myself in the Pathological Transactions. In both these cases there was considerable disease of the adjacent parts and the patients rapidly sank; and such is probably generally the case in similar instances. It is by no means uncommon in connection with endocarditis of the aortic valves to find smaller or larger excavations in the ventricular walls at the base of the septum, which, were life sufficiently prolonged, might probably become aneurismal sacs. Cases of the kind have at different times been exhibited at the Pathological Society by the late Mr. Avery,[1] Dr. Bennett,[2] and myself.[3]

In some cases it has been supposed that an extravasation of blood, or the formation of an abscess in the substance of the ventricular walls, producing a laceration or erosion extending into the cavity, may give rise to the formation of an aneurismal sac; and instances affording examples of aneurisms probably so originating have been referred to by Cruveilhier. I have also myself described a case in which, in connection with general pericarditis, an abscess had formed in the septum of the auricles, which opened into the base of the left auricle and origin of the aorta. In this instance the aortic valves were also extensively involved and the patient died rapidly, but it apparently formed an instance in which an abscess in the cardiac walls might have been followed by aneurism. The case is more fully reported by Dr. Craigie, in whose practice it occurred. It is also highly probable that in some cases lacerations of the internal portions of the muscular walls of the ventricle connected with fatty degeneration, may lead to the formation of the false consecutive aneurisms.

I have already mentioned the conclusions deduced from the cases analyzed as to the parts constituting the walls of the sacs. Dr. Thurnam has also given particulars of their contents. He states that in twenty-three cases they contained a greater or less amount of laminated coagulum; in nineteen simple amorphous clots; and in twenty-three cases they had been found empty. Of my own series of cases, nineteen contained old coagula, which were more or less decolorized, laminated, and in some cases adherent to the lining membrane of the sac; three displayed old and recent clots combined: and seven contained only recent coagula. The

[1] Path. Trans. i. p. 72.
[2] Ibid. p. 59.
[3] Ibid. ii. p. 49.

condition of the sac has not been reported in most of the remaining cases. In twenty-one of the first collection of cases the aneurismal walls, and especially when the sacs formed distinct tumors, had been strengthened by adhesions of the pericardium; in other instances there were loose false membranes on the pericardium without adhesions; and in seven cases the layers of pericardium were universally adherent. In the second series, eleven out of thirty in which the condition of the pericardium is named in the reports, displayed adhesions over the projections of the aneurisms; in five there were white patches and adhesions in the seat of disease or elsewhere; in one the adhesions were almost entire; and in three instances the two layers of pericardium were universally attached.

Seat of Disease.—M. Breschet supposed that the aneurismal sacs were nearly always situated at the apex of the left ventricle. Dr. Thurnam was led to qualify this opinion, and showed by the analysis of the cases which he collected, that, while the partial dilatations are of more frequent occurrence at the apex than elsewhere, they do occur in all parts of the ventricular walls. Of fifty-seven cases in which the description was complete, in twenty-seven the sac was situated at or near the apex; in twenty-one in different parts of the base; in fifteen in the intermediate parts of the ventricle; and in three in the septum. Of forty-two of the more recent cases, in fourteen the sacs were situated at the apex; in eleven near the base; in eight in the middle of the ventricle, at the anterior, outer, or posterior part; and in six in the septum. In three instances there were two or more sacs in the same case. In one of them one sac was situated at the apex, and another on the left side; in a second, one aneurism was at the apex, the other in the septum; in the third, one sac was situated partly in the septum and partly in the anterior wall, another was situated posteriorly in the septum, and a third occupied the middle of the external wall. Both these enumerations concur in showing that the most frequent situations for the aneurismal sacs are first the apex, then the base, and lastly the external wall and septum.

The greater liability to the occurrence of aneurisms at the apex of the ventricle is supposed by M. Breschet to be owing to the relative thinness of the parietes in that situation, exposing them to rupture during the active contraction of the heart. It is, however, more probably owing to the tissues being readily involved in inflammatory action, extending from the peri- or endo-cardium, when, as at the apex, those membranes are more nearly in contact, than when the layer of muscular structure is of greater width. The portions of the ventricle near the base are probably commonly affected, from the frequency of endocarditis of the aortic valves, leading to induration and thickening, and so to more or less obstruction to the flow of blood from the ventricle. Under these circumstances there is a tendency to excavation beneath the aortic valves, which may proceed to the extent of forming a distinct aneurismal sac. In some cases the disease is situated in what has been termed the "*undefended space*," the space which intervenes between the base of the ventricular septum and the convex sides of the left and posterior semilunar valves. This ordinarily is only closed by the endocardium of the left ventricle, and by a layer of fibrous tissue, a thin layer of muscle, and the endocardium of the right ventricle. Being thus imperfectly protected, the space is readily expanded under any unduly distending force, and a sac is formed which will protrude into the right cavities about the auriculo-ventricular aperture. When in Vienna a year ago Rokitansky showed me one or two cases of the kind; one was exhibited at the Pathological Society during the last session, by Dr. Hare, and I have found the condition myself. In some cases portions of the ventricular wall in this situation may be congenitally deficient, and a column of blood flowing from the left ventricle may distend and dilate the folds of the tricuspid valves, as shown in a specimen in the Museum of the Royal College of Surgeons. In other cases, the excavation may occupy some other portion of the base of the ventricle beneath the aortic valves, and a channel may be formed leading into a small aneurismal sac, situated external to the origin of the aorta; and such sac may be still further prolonged so as to open above into the aorta. Cases of this kind were first described and figured by Dr. Hope, though he supposed that the aneurisms originated in connection with the aorta and only opened into the ventricle. I have described two cases of the kind in the Pathological Transactions, and a similar one is also related by Dr. Bristowe. Aneurism at the base of the ventricle may also rupture into the right auricle or pericardium, Rokitansky mentions having seen a case in which both these results occurred. When the sacs form in the external wall of the ventricle, they may open into the left auricle or may burst into the left pleura, as in a case referred to by Sir A. Cooper. When seated in the septum they may press upon the right auricle and ventricle and open into one or other of those cavities, especially the right ventricle, as in the case related by Dr. Pereira, one existing in the Museum of St. Thomas's Hospital, and one referred to by Rokitansky. In cases of this kind a form of aneurism results,

which, as pointed out by Dr. Thurnam, is analogous to the "*spontaneous varicose aneurisms*" of authors.

Form and Size.—Aneurisms of the heart may be either circumscribed or diffused; or, in other words, the apertures by which they communicate with the ventricle may be more or less constricted; or the cavity of the aneurism may gradually extend from that of the ventricle without any obvious line of separation. The sacs, when situated at the apex, are more generally of the diffused form; those at the base, and in other parts of the ventricle, are more commonly circumscribed. In the first series of cases the sacs are inferred to have been circumscribed in twenty-five cases, and diffused in nineteen. As far as can be ascertained from the reports of the more recently published cases, it appears that of thirty-seven cases, twenty-five were circumscribed and twelve diffused.

The size of the sacs also varies according to the seat and duration of the disease. At the base and in the septum the sacs rarely attain any great size; on the contrary, when developed in the external wall or at the apex, they may form tumors of considerable magnitude or may even equal the dimensions of the heart itself. The acute forms of aneurism also appear, as might be expected, not to attain the dimensions of the more chronic cases. Dr. Thurnam states that in his cases the sacs might, in nine instances, be compared to nuts, in twenty to walnuts, in seven to fowls' eggs, in fourteen to oranges, and in nine their size almost or quite equalled that of the healthy heart itself. In thirty of the cases which I have myself collected, in four the aneurisms are simply stated to have been small; in five they are compared to hazel nuts or filberts; in two to walnuts; one is said to have been large enough to hold a plover's egg, one to hold a pigeon's egg, and six are compared to bantam's or smaller or larger fowl's eggs. One sac is said to have been as large as a nutmeg, another as a plum; one is reported to have been capable of holding the whole end of the thumb, another to have been as large as an apple, and a third as a small orange. Two are described as being large. In one case, in which there were two distinct cavities, both were the size of walnuts; in a second one was as large as a hen's egg, the other as a walnut. In a third there were three cavities, the largest the size of a nut. In several cases the cavities contained one or more loculi, and in one there were three large pouches projecting from the main cavity.

State of other parts of the Pericardium and Heart.—The frequency of alterations in the pericardium and endocardium and in the walls of the ventricle in the seat of the aneurismal swellings, has already been referred to. It must also be mentioned that the occurrence of thickening, opacity, and induration and ossification of the endocardium and pericardium, and the fibroid transformation of the muscular substance, are by no means confined to the immediate seat of disease. These changes often involve a considerable portion of the heart, and especially of the left auricle and ventricle. In addition to these morbid conditions, also, the effects of more recent inflammation are frequently found. Hemorrhagic pericarditis occurred in one of the first collection of cases; and in the recent series, pericarditis, with or without old adhesions and white patches, is recorded to have been found in four cases. In two also of the cases, blood was found in the pericardium, and in a large proportion of both series there was serous effusion in conjunction with general dropsy. In two cases also of the latter collection there were evidences of recent endocarditis, and in several instances fatty degeneration of the muscular structure had occurred in different parts of the heart.

In five of Dr. Thurnam's cases there is stated to have been disease of the mitral valves, in three of the aortic valves, and in one of both sets, and in only eight cases are the valves expressly stated to have been healthy. In my own cases, the valves are stated to have been healthy in only five cases. The aortic valves are reported to have been diseased in seven instances, the mitral in two, and both sets in three, and in two or three other instances the aneurismal sacs were so situated as to have interfered with the action of the auriculo-ventricular valves. It must necessarily follow that the state of the whole heart is affected to a greater or less extent in these conditions, which necessarily lead to alterations in the size of the cavities and in the thickness of the walls. From the first series of cases it was inferred that there was general dilatation of the organ in three cases, dilatation with hypertrophy in three, dilatation of the left ventricle only in two, hypertrophy in two, and dilatation with hypertrophy of both ventricles in nine. In only ten cases was the heart reported to have presented no other lesion than the aneurisms, and in three only was it stated to have been positively healthy. In the more recent collection the heart appears to have been greatly enlarged in seven cases; there was great enlargement, but especially hypertrophy and dilatation of the left ventricle in twenty cases; dilatation of the left ventricle in two; and dilatation of the right ventricle in one. In two cases the separation of the two sides of the heart was imperfect from the apertures having formed in the fold of the fora-

men ovale. In three cases the coronary arteries were diseased; and in six there existed more or less atheroma, calcification, dilatation, or aneurism of the ascending portion of the aorta. Of the whole number of cases, excluding from consideration six in which the reports are imperfect as to the general condition of the heart, there is not one in which there was not some alteration in the state of the heart or pericardium, in addition to the aneurism. In one case the heart is indeed said to have been of natural size, but in that instance there was recent pericarditis and an acute aneurism.

The shape of the heart is stated to have been frequently altered by the presence of the aneurismal swellings. In some it had an unusually wide or globular form; in others there was a bulging of the aneurismal sac, separated by a more or less distinct furrow from the other portion of the ventricular wall; and in yet other cases there were obvious tumors projecting from the surface of the organ. These were sometimes only of small size so as to be compared to a small nut or thimble; in others they were of considerable magnitude, and were separated from the walls of the heart by a distinct constriction or neck. In one specimen contained in the Museum of St. Thomas's Hospital, probably one of those referred to by Sir A. Cooper, there is a tumor with thin parietes as large as an ordinary heart projecting from the anterior surface of the organ, and separated from it by a neck which is not half the circumference of the tumor itself.

The existence, however, of an obvious tumor or irregularity on the surface of the heart depends upon the seat and size of the aneurismal sac. At the base the tumors are generally, if not always small, and do not form projections which can be detected till the parts around are dissected away. Aneurisms in the septum also can produce no marked alteration in the general form of the organ; but those on the anterior, outer, and posterior walls, if at all of large size, necessarily occasion either some general bulging or form a distinct tumor. Of fifty-four aneurisms it is inferred that only thirty-five were attended by tumor.

State of other Organs of the Body.—The condition of the other organs of the body is not recorded by Dr. Thurnam, probably from the histories of the cases which he collected being defective in these particulars. I regret also that I am not able to supply satisfactory information from the reports of the more recent cases. I find, however, that in a large proportion of them there was more or less general dropsy, and that serous effusion had occurred in one or both pleural cavities and in the peritoneal sac. In one of the cases the fluid in the pleura was bloody, the lungs being also engorged in the same case. In two cases there were signs of recent pleurisy, and in eight the visceral and parietal pleuræ were attached by old adhesions. In eight cases there was pulmonary apoplexy, emphysema, bronchitis, or pneumonia, and in one of the latter cases the lung was gangrenous. In one instance there were tubercles in the lungs, and in another old syphilitic disease of the larynx.

The liver is reported to have been small and pale in one case; fatty in one; and congested, enlarged, granular or indurated in eight cases. The spleen was large in two cases, small in one, and softened and containing fibrinous or purulent deposits in one. The kidneys were engorged in four cases; granular, atrophied, cystic, or otherwise diseased in six: and contained purulent deposits in one.

Symptoms and Cause of Death.—It is impossible to point out any symptoms which can, in the present state of our knowledge, be regarded as characteristic of the lateral or partial aneurisms of the heart; and, indeed, it is doubtful whether any such symptoms will hereafter be ascertained. This will readily be understood when the frequency with which the affection is associated with valvular diseases and with alterations in the size of the cavities and thickness of the walls of the heart is considered. On analyzing the reports which have been published, it appears that in several cases the condition was only detected on post-mortem examination, in the bodies of persons who were not known to be suffering from any form of cardiac disease, and were supposed to be previously in good health. In by far the largest proportion of cases, however, twenty-two out of twenty seven, there is a history of prolonged indisposition, not unfrequently commencing with acute rheumatism or in some inflammatory affection of the thoracic organs, and characterized by the usual symptoms of cardiac disease. Difficulty of breathing, and sense of suffocation and oppression at the chest; pain in the region of the heart, at the sternum, and at the epigastrium; palpitation and tumultuous action of the heart, and irregularity of the pulse; with cough, expectoration, and dropsical symptoms, are generally mentioned as having been present. Not unfrequently, also, the sounds of the heart are stated to have been replaced by morbid murmurs, but these appear to have been chiefly, if not wholly, referable to coincident valvular affections. The only symptoms, indeed, which can be regarded as at all of a specific character are pain and sense of weight in the region of the heart, which appear to be more constant attendants on these forms of diseases than on any other

kind of organic affection of the heart. It must, however, be concluded, that, at the present time, the diagnosis of these affections cannot be effected during life, and it is indeed doubtful whether it will be ever possible, with any exactitude, to diagnose them.

The cause of death is also often not clearly stated in the reports which have been published. It appears, however, that of the cases collected, in three the patients died suddenly, and probably from syncope, without any obvious reason being detected for the occurrence. In two, death resulted from cerebral congestion and convulsions. In one, from more acute disease supervening upon old laryngeal affection. In one from bronchitis, two from pneumonia, one from pleurisy, and in one from phthisis. In two cases the patients sank from coma and other symptoms connected with disease of the kidneys. In four instances death resulted from the rupture of the sac and the escape of blood into the cavity of the pericardium; in four from the rupture of an aneurism of the ascending aorta into the pulmonary artery. In two cases the patients died from extensive disease of the aortic valves connected with endocarditis; combined in one with the opening of an acute aneurism into the left auricle and very nearly externally, and in the other with purulent deposits in different organs. In the remaining seventeen, out of the twenty-five cases in which the particulars are given, it appears that death resulted from the progress of the general and dropsical symptoms and the affections of different organs superinduced by the cardiac defects. Of the cases previously analyzed, the cause of the death was assignable in twenty-four. In twelve of them death was sudden: in three from syncope, in six from rupture of the sac into the pericardium, in one into the left pleura, and in one from rupture of the heart itself. In four, the patients died of apoplexy or paralysis, and in one from epistaxis. In nine cases death ensued from the progress of the cardiac symptoms, and six from other coincident complications. Rokitansky mentions the case of a boy of twelve years of age, in whom a small aneurism at the base of the ventricle, after having first formed a connection with the right auricle, opened into the pericardial sac.

It thus appears that there are on record eleven cases in which the aneurismal cavities have terminated by rupture. In most of them the affection proved suddenly fatal. Such was the result in the instance of General Kidd, a gentleman of seventy-three years of age, whose case is related by Dr. Johnson, and who was found dead in his bed. Here the aneurism was of small size, and was situated near the base of the ventricle. In a case related by Dr. Wilks, a girl twelve years of age, died suddenly when playing, and an aneurism about the size of a walnut was found about the middle of the anterior wall of the left ventricle near the septum. In other instances, however, life has been prolonged for some short time after the occurrence of the rupture. Thus, in the case related by Galeatti, the symptoms indicating the rupture appeared about a week before the fatal termination; and in one which I have myself related, blood appears to have escaped into the cavity of the pericardium, not, however, by a distinct rupture, five days before death; more rapid extravasation having been prevented by adhesions between the layers of pericardium at the seat of disease.

In some cases the aneurism may be regarded as having undergone a partial natural cure. M. Cruveilhier has described cases in which the process of dilatation seems to have been arrested and the sac had been converted into bone, or more properly speaking, in which cretaceous matter had been deposited in its walls. In a case recorded by Dr. Wilks[1] the cure appears indeed to have been almost complete. A man, fifty-two years of age, of very intemperate habits, died of phthisis, and on examination after death, the heart and pericardium were found adherent to the diaphragm at the apex. In this situation there existed a hard calcified tumor, about the size of a pigeon's egg, which contained layers of decolorized coagulum. The cavity communicated with that of the ventricle by an aperture of about the same size as the sac itself. The edges of this aperture were smooth, and the membrane lining the sac was continuous with the endocardium of the ventricle. No history of the case could be obtained; but there is no doubt that the sac was aneurismal, and that the progress of the disease had been entirely arrested some time before the death of the patient.

Age and Sex of the Subjects of the Disease.—Dr. Thurnam found the sex assigned in forty of the cases which he collected, and of them thirty were males and ten females, and he points out the difference which this proportion displays to the frequency of aneurismal affections of the arteries in the two sexes. The facts which I have brought together show a still larger proportion of cases in females—the numbers being thirty-nine—twenty-five males and fourteen females. The ages of the patients in the first series of cases ranged from eighteen to eighty-one, and were pretty evenly distributed

[1] Path. Trans. vol. viii. p. 103.

throughout the middle and later periods of life, though somewhat more frequent between twenty and thirty, and in advanced life. The more recent cases display a tolerably equal distribution from early to advanced age, and are given in the following table :—

Age.	Males.
14 and 16	2
21 to 30	4
31 to 40	4
41 to 50	4
51 to 60	3
61 to 70	3
71 to 77	2
Between 60 and 70	1
Not stated	2
	25

Age.	Females.
12 and 15	2
21 to 30	4
31 to 40	0
41 to 50	0
51 to 60	0
61 to 70	4
71 to 77	2
Between 60 and 70	0
Not stated	2
	14

The most noticeable circumstance in this enumeration is the very early age at which the cardiac cases occur as compared with different forms of arterial aneurism; this being explained by the frequent origin of the disease in endocarditis, and the frequency of endocarditic affections, as complications of rheumatism, in early life. It would have been interesting to have given some more satisfactory information as to the influence which rheumatism exercises, either immediately or more remotely, in the production of the partial aneurisms of the heart. The reports of the cases are, however, very imperfect on this point; but they clearly indicate that the aneurisms are not unfrequently connected with rheumatism. They appear also to be very commonly predisposed to by habits of dissipation and intemperance, both causes which we know are very influential in the causation of other forms of cardiac disease.

Aneurism of the Left Auricle.—An instance of dilatation of the left auricle with deposition of coagula in the dilated part, the result of an injury, was related by Dionis in 1716.[1] With this exception, however, the condition does not appear to have been noticed till the beginning of the present century, when cases of the kind were related by Abernethy, Burns, and Hodgson, and, more recently, others have been placed on record by Sir A. Cooper, Elliotson, Hope, Chassaigniac, and Virchow, &c. Dr. Thurnam refers to eleven cases, including a further notice of one previously mentioned by Dr. Thomas Davies. Since the date of his memoir there have been four or five other cases published. Of these one is related by Dr. Fenwick,[1] another by Mr. Prescott Hewitt,[2] a third by Dr. Bristowe,[3] and one by myself.[4]

The so-called aneurisms of the auricle consist of dilatations containing coagula and fibrinous deposits of the sinus and auricular appendix, or both. They may either involve a considerable portion of the walls of the cavity and pass gradually from the undilated part without any obvious constriction or separation; or they may form distinct sacculated expansions. In the largest proportion of instances the sinus has been the seat of the disease, and the aneurism has been of the diffused form. In the cases, however, of M. Chassaigniac and Virchow, and in that of Dr. Fenwick, the cavity was distinctly circumscribed. Most generally, also, the disease has been found in connection with some, and often very marked, obstruction at the left auriculo-ventricular aperture; but in the instances named the valves were free from disease. The case of Dr. Fenwick was further interesting from there having existed during life a loud systolic sound audible at the apex, which was clearly due to the obstruction caused by the aneurismal swelling.

In two of the cases referred to, those of Mr. Prescott Hewitt and Dr. Bristowe, the right auricle was greatly dilated as well as the left, and the cavity contained coagula; in the former instance, apparently of similar character to those in the left auricle—in the latter, however, only the usual amorphous clots. Partial expansions of this kind should not, however, have the term aneurisms applied to them; but to maintain the analogy between the similar affections of the arteries and veins, the dilatations of the right side of the heart should be termed varicose.

Aneurisms of the Valves.—A dilatation of the mitral valve, to which the term aneurism may properly be applied, was described by Morand in 1729; another was mentioned by Laennec and Fizeau at the beginning of this century. Sir A. Cooper, also, in 1825, referred to a case then and still existing in the Museum of St. Thomas's Hospital, and two other instances of the kind have been more fully

[1] L'Anat. de l'Homme, p. 713.

[1] Lancet, Feb. 1846.
[2] Path. Trans. 1848–50, vol. ii. p. 194.
[3] Ibid. xi. 1859–60, p. 65.
[4] Ed. Med. and Surg. Journal, 1846.

related by Dr. Thurnam though previously noticed by others, of which one occurred in the practice of Sir Thomas Watson at the Middlesex Hospital. More recently specimens have been described by Cruveilhier, by Mr. Prescott Hewitt,[1] Dr. Habershon,[2] Dr. Ogle,[3] and myself;[4] and the affection has been noticed by Rokitansky in his Pathological Anatomy.

Aneurisms may occur both in the aortic and mitral valves. Of their mode of origin in the former situation a very interesting example is contained in the Museum of St. Thomas's Hospital. In one of the aortic valves there exists a small distinctly-marginated sac, which would have contained a small bean; in a second, there is one of somewhat less size, and in the third there is simply a deposit of fibrine in one part of the fold and a very slight dilatation in the same seat. It is evident that the last is the result of inflammatory action, and indicates the first stage in the production of the small aneurisms which exist in the other valves. Dr. Chevers has shown that in cases of contraction of the outlet of the ventricle and expansion of the inlet, whether relative or absolute, the aortic valves have a tendency to bulge at their most dependent parts. If this be unattended by any deposit of fibrine, the fold ultimately gives way in the weakened portion; if, however, the valve be strengthened by a deposit of fibrine, the bulging may increase till a distinct sac is produced. A very characteristic example of the kind was exhibited by myself at the Pathological Society.

In the mitral valve the disease is, I believe, always found in the free fold. The dilatation may occupy merely a small part of the valve, or may be of large size, so as to involve a large portion of the fold. In some cases the disease seems to originate in the protrusion of the endocardium of the left ventricle, through the fibrous structure of the valve, so as to come in contact with the lining membrane of the left auricle. In other cases all the coats are dilated. In both instances the sacs generally project into the cavity of the left auricle, and sometimes the base of the sac gives way, and an opening is produced in the valve as if a piece of the fold had been punched out. The sacs may vary in size from one which would lodge a pea or bean or filbert, to one capable of holding a pigeon's egg. Of the former size the cases of Mr. Prescott Hewitt and myself afford instances. Of the latter, the specimen in the Museum of St. Thomas's, referred to by Sir A. Cooper, is a most remarkable example. In several cases two or more sacs have been found in the same valve. These small aneurisms of the mitral valve not unfrequently occur in cases of aortic valvular obstruction, and I have described one which was found in a case of rupture of the aortic valves. The sacs may contain laminated coagula, and in one of the cases described by Mr. Keith, a portion of the valve was entirely wanting, and a small sac was produced by a fibrinous coagulum being attached on the auricular side.

These affections are not only interesting pathologically, but may be of practical importance, as both at the aortic and mitral valves they may give rise to the symptoms and signs of incompetency.

I have before referred to a specimen which exists in the Museum of the Royal College of Surgeons, in which the current of blood flowing through a congenital aperture existing at the base of the ventricular septum has expanded portions of the tricuspid valves, so as to form small sacs or aneurisms; and I have seen a similar condition of the tricuspid valve in a recent case of malformation of the same kind.

[1] Path. Trans. vol. iii. p. 78.
[2] Vol. ix. p. 117.
[3] Ibid. vol. vi. p. 156.
[4] Vol. iii. p. 71.

ADVENTITIOUS PRODUCTS IN THE HEART.

By Thomas Bevill Peacock, M.D., F.R.C.P.

TUBERCLE IN THE HEART AND TUBERCULAR PERICARDITIS.

Laennec[1] when alluding to accidental products says, that he had only three or four times met with tubercles in the substance of the heart; and when speaking of chronic pericarditis, he remarks, that a tuberculous eruption may sometimes be developed in the false membrane and may thereby convert the acute into chronic disease, as frequently happens in pleurisy and peritonitis, and he states that he had met with two cases of the kind. In this passage, Laennec indicates the forms in which tuberculous deposits are found in the heart; in one of these they take place in the substance of the organ; in the other on the surface, in connection with inflammation of the pericardium. The former is certainly a very rare condition. Louis[2] says that in 112 dissections of phthisical persons he did not meet with a single instance of the existence of tubercle in the substance of the heart. Rokitansky[3] also speaks of the extreme rarity of the affection; and in the records of 116 post-mortem examinations of persons who had died of phthisis which I have analyzed, I do not find more than two or three cases in which tubercle is said to have been found in the heart. The recorded instances of such deposits being at all of serious importance are also very few in number. The first writer who alludes to cases of the kind is, I believe, Dr. Baillie,[4] who in his "Morbid Anatomy" says that he "once saw two or three scrofulous tumors growing from the cavity of the pericardium, one of which was nearly as large as a walnut. They consisted of white soft matter, somewhat resembling new cheese," and he adds that "the pericardium is a very unusual part for any scrofulous affection;" and in his "Dissection,"[5] in alluding to the same case, he further says that both lungs were studded with tubercles, and the right in a state of suppuration in places. The subject of the dissection was a man twenty-one years of age. Dr. Macmichael,[1] in 1826, detailed the history of a man of thirty-five, who died at the Middlesex Hospital with dropsy and other symptoms of cardiac disease, and in whom the lungs and bronchial glands were found tuberculous, and the pericardium, especially at the base, studded with tuberculous deposits. In 1834, M. Sauzier, as quoted by Bouillaud,[2] found in a man thirty-four years of age, who died with abscess from caries of the sternum after accident, the lungs, pancreas, and pleura tuberculous, and in the substance of the auricles there were two tubercles, and around them the pericardium was adherent. The most remarkable case of the kind is, however, that related by Dr. Townsend in 1852.[3] In this instance a large mass described as tuberculous was connected with the left auricle, and had compressed that cavity and the entrances of the pulmonary veins, so as to give rise to extreme distension throughout their course; tubercles existed in the bronchial glands but not apparently in the lungs. The subject of the disease was a man sixty-two years of age, who died after an illness of twelve months. Since this time a case has been recorded by the late Dr. Baly in the Pathological Transactions.[4] It occurred in a prisoner at Millbank, sixteen years of age, who died with symptoms of subacute fever and head affection, after an illness of about ten days, and tubercular masses were found in the substance of the brain, and small tubercles in the lungs, bronchial glands and intestines. A yellow rounded mass, the size of a man's thumb, projected from the inter-auricular septum into the cavities of the right and left auricles, the two projections being parts of the same tuberculous mass which was situated in the septum. Dr. Quain also mentions that there were tubercular deposits in the pericardium in a Bosjesman girl, who died of tuberculosis.[5]

[1] Diseases of Chest, Forbes's trans. 4th edit. 1834, pp. 586 and 623.

[2] Sydenham Society's Trans. 48–50.

[3] Ibid. vol. iv. p. 210.

[4] Morbid Anatomy, and works by Wardrop, 1825, vol. ii. p. 9.

[5] Works by Wardrop, vol. i. p. 220.

[1] London Medical and Physical Journal, vol. lvi. (N.S. vol. i.) p. 119.

[2] Maladies du Cœur, 2me edit. tome ii. p. 442.

[3] Dublin Journal, vol. i. 1852, p. 176.

[4] Path. Trans. vol. iii. 1850–51, 1851–2, p. 34.

[5] Path. Trans. vol. ii. 1848–49, 1849–50, p. 182.

The second form of tuberculous deposit which occurs in connection with inflammation of the pericardium, is by no means so rare as that which has just been mentioned. The first instance of the kind that is recorded is probably that by Corvisart,[1] and another was figured by Cruveilhier, and is further alluded to in the General Pathology more recently published. The pericardium adhered intimately to the heart, and in these adhesions a thick and continuous layer of tuberculous matter was deposited, and this enveloped the vessels and had destroyed the muscular structure of the auricle. M. Fauvel, as quoted by Aran,[2] and by Rilliet and Barthez in their work on diseases of children, met with a case of tubercular pericarditis in a child six years and a half old, who died with dropsy and symptoms of disease of the heart. The pericardium was entirely adherent, the heart was considerably enlarged, and its surface was studded by whitish-yellow friable nodules, some of them the size of a nut, and as numerous behind as in front. The internal surface of the right ventricle displayed similar depositions everywhere except at the septum. Since this time the occurrence of tuberculous deposits in connection with pericarditis has been made the subject of a special memoir by Sir G. Burrows,[3] in which he details three cases which he supposes to be examples of the affection; and in two of them—one of which occurred in his own practice, the other under the care of the late Dr. Baly—the inference was confirmed by post-mortem examination. More recently, Dr. Bristowe has described three other cases in the Pathological Transactions,[4] and such instances cannot indeed be very uncommon. Cruveilhier says that he has many times met with tubercles, in connection with false membranes, in children with tuberculous lungs.[5] Louis also refers to such cases, and details the particulars of one in his memoir on pericarditis.[6] Otto[7] mentions having twice seen the condition in children, and Dr. Walshe[8] states that it is displayed in one of Dr. Carswell's drawings contained in the collection to illustrate morbid anatomy at University College. I have myself met with three cases of the kind, two while Pathologist of Edinburgh Infirmary and one at the Victoria Park Hospital.

Tubercular deposits in the pericardium bear a close resemblance to the similar disease of the arachnoid, pleura and peritoneum. They may be of very small size, mere specks, or may attain the dimensions of a cherry-stone, filbert, or walnut. In consistence they are generally soft, and they are usually of a grayish or yellowish color. In one of my own cases, the tubercles, which were thickly spread over the attached and reflected pericardium, varied in size from that of a pin's head to a cherry-stone. In another, while there were very small masses of yellowish tubercle thickly studied over the surface of the heart, there were also laminated false membranes, in some places a quarter of an inch, in other parts fully half an inch in thickness, and the middle layers of this deposit were of a yellowish color, soft and granular, and closely resembled what is commonly called tuberculous infiltration. In the third case the tuberculous deposit assumed the form of small granulations of a grayish color, the two layers of pericardium being entirely attached by cellular adhesions. The affection in two of Dr. Bristowe's cases consisted of small miliary granulations, in one with patches more closely set together in places; in the other there were both separate tuberculous masses and laminæ of considerable size.

In the cases which have fallen under my own notice the deposits were situated beneath the serous membrane, and in one of them there were masses which were more deeply imbedded in the substance of the ventricles and which were only exposed on section. One of these cases also, it will be observed, displayed tubercle in the centre of a thick layer of false membrane covering the heart, thus corresponding with the observations of Laennec and Cruveilhier. The different writers who have alluded to this subject have agreed in asserting that tuberculous affections of the heart are only met with in connection with similar deposits in other parts of the body, and the cases which have been recorded entirely confirm that view. The most frequent coexistence is with tubercle in the bronchial glands, or in the lymphatic glands of the mediastinum. In two of the cases which I have myself seen, though occurring in persons twenty-eight and sixty-seven years of age, there was tuberculous deposit only in the bronchial glands and heart; though the general rule is, as is well known, that, after early life, if tubercle be found in any part of the body it also exists in the lungs. In the third case, the subject of which was a girl thirteen years of age, no tubercle was found anywhere else. In this instance

[1] 3me édit. Paris, 1818, p. 26.
[2] Aran, Arch. Gén. de Méd. 4me série, 1846, tome xi. p. 181.
[3] Med.-Chir. Trans. vol. xxx. 1847, p. 77.
[4] Vol. xii. 1860–61, p. 63.
[5] Traité d'Anat. Path. tome iv. série 1862, p. 684.
[6] Revue Médicale, 1826.
[7] Path. Anat. by South, 1831, p. 258.
[8] Diseases of Heart, 1862, p. 357.

there was also slight mitral valvular disease. In Sir G. Burrows' case the lungs, pleura, bronchial glands, peritoneum, and spleen were tuberculous; and in Dr. Baly's there were tubercles and ulcers in the intestines and lungs. In one of Dr. Bristowe's patients there was tuberculous perforation of the intestines; in a second, there was tubercle in the mediastinum; and in the third, in the brain, lungs, pleura, spleen, and mesentery. The occasional occurrence of tuberculous deposits in the heart with similar affections of the bronchial glands and mediastinum, and in some cases when the lungs are entirely free, led Cruveilhier to suggest that possibly the affection of the glands might be secondary to that of the heart; but this supposition is scarcely in accordance with the advanced disease of the lungs which is reported to have existed in other instances. Laennec supposed that the tubercles in cases of this description were the result of the inflammation, and were situated in the false membrane; the latter is, however, certainly not usually the seat of the deposit, and Sir G. Burrows is much more probably correct in regarding the pericarditic affection as the effect of the irritation set up by the deposit under the membrane. Indeed, the first class of cases, in which the tubercles are situated deeply in the substance of the heart or under the endocardium and assume the form of separate tumors, cannot be regarded as essentially distinct from the second, in which the tubercles are more superficial. The absence of adhesions in some of the latter class of cases seems conclusively to show that the inflammatory exudation is at least generally secondary.

The tuberculous deposits in the heart occur under the same circumstances as those which attend similar affections in other parts of the body; they may be found in both sexes, and at all ages, but they are more common in comparatively early life.

The age and sex of the subjects of some of the cases referred to are as follows:—

Males	6½ years.	Females	14 years.
"	13 "	"	20 "
"	16 "	"	28 "
"	19 "		
"	21 "		
"	24 "		
"	34 "		
"	36 "		
"	62 "		
"	62 "		
"	67 "		

In several of the cases of tubercular pericarditis the evidences of effusion in the pericardium had been observed during life. When such signs arise in persons who are obviously tuberculous, and especially if they assume the subacute form and are not attended by any large amount of liquid effusion, they may be suspected to be connected with tubercular deposits. It must, however, be borne in mind that pericarditis, having no connection with tubercle, may occur during the progress of phthisis. The inference as to the tubercular origin of such cases is therefore by no means decisive.

CANCER.

Cancerous deposits in the heart are of more common occurrence than tubercle. Dr. Walshe,[1] writing in 1846, says that he had readily found twenty-five cases recorded; and more recently, in a paper in the Pathological Transactions,[2] I collected the particulars of forty-five, including in this number two which had fallen under my own notice. The earliest published examples of the disease were, I believe, those of Andral and Bayle in 1824.[3]

The cases of cancerous deposit in the heart may be classed into four series: First, Cases of primary cancer, in which the disease exists only in some part of the organ. These are of extremely rare occurrence; of the forty-five cases referred to, only two were expressly stated to have been instances of the kind,[4] though in the reports of seven others, no mention was made of the existence of cancer in any other part of the body.

Secondly, Cases in which the disease occurred coincidently and probably simultaneously, in the heart and in different parts of the body, and especially in parts adjacent to the heart. This form, though still rare, is more common than the other.

Thirdly, Instances in which the disease first appears in parts adjacent to the heart, — the bronchial or mediastinal glands, the lungs, or the glands around the larynx and in the neck,—and thence spreads so as to involve the pericardium and the large vessels at the base of the heart or the auricles. Cases of this kind are not uncommon, though less frequent than those of the next series.

Fourthly, By far the largest proportion of cases of cancerous diseases of the heart occur secondarily to the deposit of cancer in some distant organ. Of the forty-five cases, twenty were of this description; the primary disease being seated in different cases in the eye, the cheek and bones of the face, the lower lip, the breast and axillary glands, the ribs and pleura,

[1] Nature and Treatment of Cancer, p. 368.
[2] Vol. xvi. p. 99, 1864, 1865.
[3] Revue Médicale, 1824, tome 1re, p. 268.
[4] Ollivier, Traité de la Moëlle Epinière, 3me edit., 1837, tome ii. p. 164; Segalas, Rev. Méd. tome iv. 1825, p. 247.

the abdominal organs, the inguinal glands, the uterus, vagina, labia, the penis and testes, and the upper and lower extremities.

The heart may be affected by cancer in different forms. Thus, of the cases collected seven are reported to have been cases of scirrhus, four of melanosis, and twenty-five of encephaloid. The deposit also may assume either the form of distinct masses or tubera, or it may be infiltrated into the tissue, or occur on the surface.

The first form is the most common, especially when the deposits are secondary. The masses in different published cases are compared in size to peas or beans, to almonds or chestnuts, or to hen's eggs or oranges; and they may be only one, two, or three in number, or they may amount to a dozen or more,[1] and in one very remarkable case it is stated that they were so numerous that the examiner ceased counting them after enumerating six hundred.[2] The most frequent seat of the disease seems to be the right auricle and ventricle, though the tumors may also occur, either alone or otherwise, in other parts of the organ. Generally they are situated beneath the attached pericardium; more rarely beneath the endocardium; and still more rarely in the substance of the auricles and ventricles or in the septa. The deposits may only slightly project above the adjacent surface, or they may form distinct and nearly separate tumors, the mass being only attached to the part from which it projects by a narrow pedicle. In the Museum of St. Thomas's Hospital there is a specimen of medullary growth from the left auricle, which is almost entirely detached from the lining membrane. In some cases the masses are reported to have pressed upon the cardiac cavities or apertures, so as to interfere with the transmission of the blood or with the action of the valves.

More rarely the disease assumes the form of infiltration, and when this is the case, the structure of the heart may be only slightly affected, or it may be extensively and completely destroyed. In one instance it is stated that not more than a twelfth of the organ was free from the deposit.[3]

In the third form of disease the heart is found enveloped in a cancerous mass, which produces entire adhesion of all parts of the pericardium. This is, I believe, of very unfrequent occurrence. A case of the kind has, however, been described and figured by Dr. Bright in the Medico-Chirurgical Transactions.[1] A second is related by Dr. Kilgour,[2] in the "London and Edinburgh Journal of Medical Science;" and a third was described by myself in the paper in the Pathological Transactions before referred to.[3]

In only two or three of the recorded cases is the cancer stated to have been softened or ulcerated, and the nature of one of them may be doubted. In one instance, however, a cancerous mass situated near the origin of the anterior coronary artery had softened and caused perforation of the arterial coats and the escape of blood into the cavity of the pericardium.[4]

Cancerous deposits in the heart do not appear to be generally productive of any special symptoms by which their presence can be detected during life. In some cases, when there was disease of the adjacent organs, there were signs of pressure on the large vessels and of interference with the circulation of the blood; and in three or four other instances the formation of the deposits on the surface of the heart occasioned inflammation of the pericardium which was recognized by the usual signs during life. Of this I have myself seen two instances. Most usually, however, there are no symptoms by which the affection of the heart is indicated, and the condition is only detected on post-mortem examination. In the case under my own care which has been mentioned—notwithstanding that the existence of a tumor in the chest was ascertained a considerable time before the patient's death, and that the patient's father was said to have died of cancer of the heart, and thus attention was particularly directed to the state of the organ—no symptoms indicating the heart to have been involved were detected.

SIMPLE AND OTHER CYSTS.

Lancisi mentions having seen a cyst containing thick matter (meliceris) in the

[1] Exposition d'un cas remarkable de Maladie Cancereuse (Paris, 1825), quoted by Dr. Churchill in London Med. and Phys. Journal, vol. lvii. (N. S. vol. ii.), 1827, p. 280.

[2] Case of Dupuytren, quoted by Cruveilhier in Essai sur l'Anat. Path. Paris, 1816, vol. i. pp. 86–87.

[3] Rilliet; Bullet. de la Soc. de Méd. 1813, No. 5, tome iii. p. 357. A very marked case of cancerous infiltration with masses in the mediastinum, which occurred in a patient of Dr. Barker's, at St. Thomas's Hospital, is described by Dr. Bristowe in the Path. Reports, vol. vii. The specimen is preserved in the Museum, x. 67.

[1] Vol. xxii. 1839, p. 15.

[2] Vol. iv. 1844, p. 828.

[3] Vol. xvi. 1864–65, p. 100, Case 1. The specimen is preserved in the Victoria Park Hospital Museum.

[4] M. Broca, Bullet. de la Soc. Anat. 25me, année 1850, p. 253.

substance of the heart, and other writers describe the occasional occurrence of cysts of different kinds in the heart or pericardium. Thus Cruveilhier refers to hæmatoid cysts as occurring in the pericardium and other serous surfaces, but does not detail any instance of the kind; and I do not know any recorded case except that reported by Dr. Ogle in the Pathological Transactions for 1857 and 1858.[1] In this instance a large cyst was found beneath the pericardium covering the posterior surface of the right ventricle. It had firm and thick walls, and contained laminated coagulum with brownish granular material. The layers of pericardium were adherent, and there were old and thick adhesions of the right pleura, with some similar coagulum in its sac. No connection could anywhere be traced between any of the cavities of the heart and the cyst; and Dr. Ogle supposes that probably the blood had escaped from one of the branches of a coronary artery; and that having first lodged in the pericardium, it had subsequently ruptured into the pleura. The cavities of the heart were rather large, the lining membrane of the right auricle was thickened and opaque, and the coronary arteries were in various places rigid. The specimen was removed from a man fifty-five years of age, who died with symptoms of cardiac disease and dropsy, and who had been ill for two years; but no decided history of any attack to which the condition of the heart could be ascribed appears to have been obtained. The condition of the coronary arteries is in favor of Dr. Ogle's supposition, but it may be open to question whether the cysts might not have originated in acute hemorrhagic inflammation of the pericardium and right pleura.

Certainly in some cases the appearance of a cyst is produced by the remains of a pericarditic effusion; the two layers of serous membrane becoming adherent, except in one portion, where a cavity containing pus or serum still exists. A specimen of this kind was exhibited at one of the meetings of the Pathological Society.

ENTOZOA.

In the works of the earlier writers on morbid anatomy, cases are referred to in which the heart is stated to have contained worms. Such reports are, however, generally entitled to little credit, though of late years hydatid cysts have, in various cases, been found in different parts of the heart. Probably the earliest recorded instance of the kind is that mentioned by Morgagni,[1] of a man seventy-four years of age, who died in the hospital at Padua; but of whose previous state no further history was obtained than that he had not suffered from any of the usual symptoms of cardiac disease. A tumor about the size of a cherry was found at the posterior surface of the heart near the apex. It was half imbedded in the substance of the organ, and "on puncturing it a small quantity of clear fluid escaped, but a more turbid humor remained, and was only evacuated on laying it open. In so doing a small piece of membrane escaped. This displayed white, and, as it were, mucous particles, and a particle of tendinous hardness." The whole was included in a dense sheath. Dupuytren,[2] at the beginning of the present century, placed a similar case on record. It occurred in a female forty years of age, who died in one of the Paris hospices, whose body was dissected in the anatomical school. No history of the case during life was obtained. The right auricle was very greatly dilated, and on its inner surface, under a smooth membrane, were found numerous cysts which nearly filled the cavity. About the same time a third case was related by Dr. Trotter;[3] it occurred in a boy fourteen years of age, on board one of her Majesty's ships, who had been very livid and subject to dyspnœa and palpitation: a large cyst, containing several loose hydatids, was found in the right auricle, and two similar bodies were also contained in the ventricle. Two cases of the kind are contained in the Transactions of the Medical and Chirurgical Society; one of these, which was published in 1821, occurred in a boy of ten, who died suddenly without having been previously ill, and the case is imperfectly related by Mr. David Price.[4] The other was communicated by Mr. Evans[5] in 1832. The subject of the disease was a delicate female, forty years of age, who was suddenly seized with pain in the præcordia and difficulty of breathing, and died in a few days. The pericardium displayed an effusion of lymph and serum; and a considerable tumor was situated at the apex of the heart and projected into the right ventricle, filling a fourth of the cavity. The tumor proved to be a cyst containing numerous hydatids, varying in size from a pea to a pigeon's egg. A

[1] Vol. ix. p. 165.

[1] Alexander's Translations, vol. i. p. 583. Letter xxi. Art. 4. See also Letter iii. Art. 26, p. 60, where it is said a white membrane protruded like a hydatid.

[2] Journal de Corvisart et Leroux, tome v. année xi. p. 139.

[3] Medical and Chemical Essays, 1795, p. 123. Case of a Blue Boy.

[4] Vol. xi. p. 274.

[5] Vol. xvii. p. 507.

plate is given of the specimen, which is stated to be preserved in the Museum of St. Bartholomew's Hospital. In 1838, Mr. Smith of Bristol published a somewhat similar case,[1] which occurred in the practice of a surgeon at Warminster. The subject of the disease was a female, whose age is not stated, and who died after an illness of three hours. A large hydatid was found in the right ventricle, and must have obstructed the entrance of the blood into the pulmonary artery.

The more recent writers on cardiac diseases and on pathological anatomy very generally refer to cases of hydatid cysts found in some portion of the heart. Andral[2] says that he has seen three instances of the kind. In one a tumor, the size of a walnut, was imbedded in the substance of the left ventricle; in another a cyst, as large as a nut, was attached by a small pedicle to the lining membrane of the right ventricle; and in the third, three cysts, the size of nuts, were imbedded in the substance of the heart. The cysts were transparent except at one point which was white and could be made to protrude like a head from the centre, and he was thus led to regard them as cysticerci. Rokitansky[3] relates the case of a woman, twenty-three years of age, who died suddenly, and a tumor the size of a hen's egg was found at the upper part of the interventricular septum, and protruded into both ventricles. On the right side the cyst had burst, and the contained hydatid had become impacted in the conus arteriosus, so as to obstruct the entrance into the pulmonary artery. In another instance, in a soldier thirty-five years of age, who also died suddenly, a tumor of the size of a duck's egg was found in the upper part of the septum and corresponding portion of the left ventricle behind. The sac contained fibrinous coagula mixed with portions of acephalocyst. The surfaces of pericardium were adherent in the seat of the tumor. M. Aran,[4] in a paper on these and other forms of tumor of the auricles, published in 1846, relates a case which occurred to M. Dupaul, in a female twenty-three years of age, who died suddenly after her confinement, and on examination a hydatid cyst in the left auricle was found to have ruptured on both sides, so as to allow of the escape of blood from the auricle into the pericardiac cavity. It was evident that the tumor had been developed under the endocardium of the auricle. Mr. H. Coote, in 1854,[1] found a large cyst in the walls of the left ventricle of a subject under dissection at St. Bartholomew's Hospital, and he refers to a second specimen as existing in the museum, doubtless the case of Mr. Evans before referred to. In addition to the cases now mentioned several will be found reported in the Pathological Transactions by Dr. Budd,[2] Dr. Wilks,[3] Dr. Habershon,[4] &c., and one, which occurred in a patient of my own at St. Thomas's Hospital, is related by Dr. Hicks and myself.[5] I have also had the opportunity of examining a specimen exhibited by the late Mr. Ward, at one of the earlier meetings of the society.[6] In Mr. Ward's case the subject of the disease was a man, twenty-two years of age, who died shortly after having sustained an accident: the cyst was about the size of a French walnut, and was situated at the posterior and upper part of the left ventricle, beneath the superficial muscular fibres. My own patient was a boy of eighteen, who died after an illness of about thirteen months: the cyst, about the size of a walnut, was partially imbedded in the muscular substance of the right ventricle, but did not project into the cavity. In the sixth volume of the Transactions,[7] there is a description of a case in which a patient at the Colney Hatch Asylum died suddenly when under excitement, and after death two cysts, one of which had ruptured, were found beneath the attached pericardium.

The precise nature of the cyst in some of the above cases is not clear. The description given of that related by Morgagni is supposed by Laennec conclusively to indicate the hydatid to have been a cysticercus; and both Andral and Rokitansky speak of having met with cysticerci in the substance of the heart. Most generally, however, the cysts appear to be those of the echinococcus. Such is stated to have been the case in the instances related in the Pathological Transactions,

[1] Lancet, vol. ii. p. 628.
[2] Path. Anat. by Townsend and West, vol. ii. p. 348.
[3] Path. Anat., Sydenham Society's Trans. vol. iv. p. 208.
[4] Arch. Gén. de Méd. 4me série, tome xi. p. 187.

[1] Med. Times and Gazette, xxix. p. 156.
[2] Vol. x. p. 80.
[3] Vol. xi. p. 71.
[4] Vol. vi. p. 108.
[5] Vol. xv. p. 247.
[6] Vol. i. p. 225. Dr. Walshe mentions in his work on Diseases of the Heart, &c. (3d edit. 1862, p. 65), that a specimen is figured in one of Dr. Carswell's drawings, and that a hydatid, the size of a pigeon's egg, situated in the interventricular septum, is contained in University College Museum. In the Museum of St. Thomas's Hospital there is in addition to the specimen described by Dr. Hicks and myself (x. 68) another (x. 64) in which the cyst, as large as a duck's egg, is situated at the apex.
[7] P. 114. See Report on the case by Dr. Wilks.

though the bodies were not always met with. The Trichina, on the other hand, is usually considered not to be found in the heart. This is, however, denied by Dr. Cobbold,[1] who says that all the different forms of larvæ occur in the heart, but they do not stay there, the firmness of the muscular texture interfering apparently with the development of the worm in that situation. The same writer gives some calculations of the relative frequency with which the echinococcus is found in the heart and in other organs. Thus he states that Droaim, of 373 cases in which these cysts were found in some part of the body, met with them in the heart in ten cases; and Dr. Cobbold, of 136 cases, found echinococci in the heart or pericardium in nine instances. The most common situations for the cysts appear to be the right auricle and ventricle, but no part of the organ is free from them; cases being recorded in which the walls of the left ventricle were affected; and, it will be observed also, the interventricular septum. The cysts may be developed beneath the pericardium or endocardium, or in the substance of the muscle. According to the situation which they occupy is their tendency to grow, so as to protrude externally or internally; and they may ultimately rupture into the pericardium or into one of the cavities of the heart. In the former situation they may give rise to acute pericarditis, or to adhesion of the surfaces of the membrane covering the projecting portion. In the latter the loose hydatids may escape into the cavity and produce fatal obstruction to the circulation of the blood. In one case, it will be observed that a cyst ruptured both externally and internally, and so allowed of hemorrhage into the cavity of the pericardium.

The hydatids in the heart appear frequently to be solitary, not occurring in any other structure of the body. Such seems to have been the case in the instances related by Morgagni, Dupuytren, Dr. Trotter, Mr. Smith, and Mr. Coote, in one of those by Rokitansky, and in the cases described in the Pathological Transactions by Dr. Budd and Dr. Habershon, and probably also in that of Mr. Ward. On the other hand, in the second case of Rokitansky there were three separate cysts in the liver. In the case of Dr. Wilks, there was also a cyst in the liver; and in my own case, in addition to the cyst in the right ventricle, there were numerous hydatids in the liver, spleen, omentum, right kidney, and lungs; and portions of cysts were expectorated during life.

It will be seen that in the cases referred to the hydatids occurred in persons of both sexes and of all ages. It may also be observed that there are no certain signs by which their presence in the heart can be detected during life. In some cases they have been found without having been preceded by any indications of defect in the circulatory organs; in other instances they have occurred in persons who have died after longer or shorter illnesses, with symptoms clearly pointing to some cardiac disease. In cases of the latter description, if there were evidences of hydatids in some other part of the system the suspicion might be entertained that the cardiac symptoms were due to the development of hydatid cysts in some part of the heart. In my own case there was nothing observed during life which at all indicated that the heart was the seat of disease.

[1] Entozoa, 1864, p. 275.

FIBRINOUS DEPOSITS: SYPHILITIC AFFECTIONS OF THE HEART.

The substance of the heart is not unfrequently the seat of fibrinous deposits. These may occur either as the result of acute inflammation of the muscular structure, myocarditis, with or without peri- and endo-carditis; or they may be connected with an altered condition of the blood, leading to the effusion of fibrine into the muscular structure, in the same way as such effusions occur in other organs, the spleen or kidneys, or as the blood coagulates in the vessels themselves. When deposited the fibrinous material may soften and allow of the partial destruction of the walls of the heart, so as to constitute a false lateral or partial aneurism; or it may undergo an imperfect organization, being converted into fibroid tissue, and this, being less resistant than the natural muscle, may yield to the pressure of the blood, and a true partial aneurism be formed. Closely allied to these deposits are those which occur in the substance of the heart in connection with constitutional syphilis. Corvisart, struck with the remarkable resemblance sometimes presented by vegetations on the valves of the heart to syphilitic warty growths on the external organs of generation, suggested that in some such cases the vegetations might have a syphilitic origin; and he detailed several cases which he regarded as supporting this idea. His views have not, however, been generally adopted; and Laennec in particular, considering the frequency of venereal affections and the comparative rarity of such vegetations, expressed his decided dissent from the supposition. More recently, however, writers have attached more importance to the suggestions of Corvisart. Dr. Julia,

of Cazeres,[1] has published several cases in which vegetations on the endocardium were found in persons who were known to have recently had syphilis and presented other indications of the disease; and in two of these cases there were small patches of ulceration on the surface or in the substance of the heart. He also refers to a case published in 1778, which, though often quoted as an example of ulcerated cancer of the heart, is doubtless an instance of syphilitic ulceration. The case was reported by M. Carcassone to the Académie de Médecine, and occurred in a female of dissipated habits, twenty-two years of ago, who was an inmate of the House of Refuge at Perpignan. Her illness, which followed upon chancres, was characterized by weight and pain in the region of the heart, and rapidly proved fatal; after death a large ulcer with indurated base was found on the anterior surface of the heart. More recently cases have been recorded by Ricord, Lebert, and especially by Virchow.[2] The latter writer has indeed made the syphilitic affections of the heart the subject of a special memoir, of which a translation has been published as a separate work in French.[3] In this country several communications of a similar kind have recently appeared in the Pathological Transactions, chiefly by Dr. Wilks.

The syphilitic affections of the heart resemble the similar degeneration of muscular structure in general. They consist of fibrinous exudations into the connective tissue, which may either soften and suppurate, forming ulcers or small abscesses; or they may be converted into masses of hardened fibroid tissue, causing a puckered appearance resembling a cicatrix on the surface, and are generally combined with thickening and induration of the covering and lining membranes. In the first case described by Virchow, it is stated that a portion of the organ near the base of the posterior fold of the mitral valves, for the space of about an inch and a half, was occupied by a whitish-colored hard mass, and the intra-ventricular septum was also similarly degenerated to the depth of from a quarter to half an inch. The endocardium was nearly cartilaginous, and tendinous cords passed deeply into the substance of the heart; the muscular structure had undergone the fatty degeneration, and the surface of the ventricle was marked by callous tuberosities. Under the microscope in the points of a white color and tendinous structure, the muscular fibres had disappeared and were replaced by fibrous tissue. At the apex of the heart there was a slight dilatation, indicating the commencement of an aneurism.

[1] Gaz. Méd. de Paris, 1845, No. 52, p. 845.

[2] Archiv. für. Path. Anat. und Phys. etc., 1864, p. 468.

[3] Le Syphilis Constitutionelle, par M. Rudolphe Virchow, traduit de l'Allemand par le docteur Paul Picard, Paris, 1860.

FIBRO-CARTILAGINOUS AND OSSEOUS DEGENERATION.

Under these terms, authors have described changes which are not of uncommon occurrence. Corvisart has related a case in which he states that the walls of the left ventricle were at least an inch in width, and much hardened. "At the apex, up to a certain point and throughout its thickness, the muscular structure was cartilaginous. The fleshy bodies also had acquired a remarkable hardness, approaching that of cartilage."[1] This occurred in a man sixty-four years of age, who died after an illness of about two years' duration characterized by dyspnœa, dropsy, and other cardiac symptoms. The state of the pericardium is not mentioned, but the mitral valve was also cartilaginous. The condition here described was alluded to by Laennec, and has been more fully illustrated by Cruveilhier.[2]

The transformation may either be general or diffused, extending over a considerable portion of the heart; or it may be partial and limited to a small part. The diffused or more general change is chiefly seen in the parietes of the right ventricle, occurring in cases where the orifice of the pulmonary artery, the pulmonic circulation, or the left auriculo-ventricular aperture is obstructed, so as to subject the affected part to long-continued distension. This condition, which is well known to all pathologists, has recently been made the subject of a paper by Sir W. Jenner.[3]

The other or partial form is seen in the walls of the left ventricle, and especially at the apex or outer wall. When existing to a marked degree, it is generally combined with some dilatation of the cavity in the seat of the transformation, and not unfrequently with bulging of the walls; and it has been regarded by Cruveilhier as the first step towards the formation of the true lateral or partial aneurisms. A view somewhat similar is also maintained by Rokitansky.

In the slighter forms of the degeneration, such as occur in cases where the change is diffused, the structure of the heart is much coarser than usual, the altered parts have a yellowish color and a peculiarly hard leathery feeling, and resist when cut by the knife. The more

[1] 3me éd. 1818, p. 171, obs. 28.

[2] Traité d'Anat. Path. Gén. tome iii. 1856, p. 601.

[3] Med.-Chir. Trans. vol. xliii. 1860, p. 199.

advanced degrees of the transformation are only seen in cases in which the disease is limited in extent, and under such circumstances the muscular structure may be almost entirely replaced by dense white fibrous material. This, as before mentioned, is generally only found at the apex of the left ventricle, but it may occur over a large portion of the outer wall, or in the interventricular septum and fleshy bodies; and Cruveilhier says that he has seen the change affecting fully a third of the muscular substance of the organ.

The mode in which the transformation is effected probably varies in different cases. Cruveilhier supposed that it was a slow change, by which the cellular tissue in the muscular substance became thickened and indurated, and replaced the atrophied contractile tissue. Rokitansky refers the change to inflammation; and there can be no doubt that inflammatory action, affecting the peri- and endo-cardium or both these membranes, and involving to a greater or less extent the interjacent muscular substance, does in some cases give rise to the alteration. This is shown by the very general occurrence of thickening and induration of the investing membranes, or of adhesion of the visceral and reflected layers of the pericardium, in cases in which the muscular structure is transformed. The relative thinness of the muscular substance of the heart at the apex affords apparently the explanation of the greater frequency of the change in that situation; and the proneness to endocarditis on the left side accounts for the more marked changes being only found in the walls of the left ventricle.

In other cases the change is probably due, as pointed out by Sir W. Jenner, to long-continued congestion of the substance of the heart, causing slow hypertrophy and induration of the connective tissue and secondary atrophy of the muscular fibres. This seems the mode in which the diffused and general induration of the walls of the right ventricle is produced, though there does not appear to be any adequate reason why it should be so frequently confined to the right side. In yet other cases the transformation is probably the result of the imperfect organization of fibrinous material, which, in connection with an altered condition of the blood, is effused beneath the investing membranes or in the substance of the heart. These effusions are not of unfrequent occurrence and generally co-exist with similar depositions in the spleen, kidneys, &c. Whatever be the mode in which the disease commences, the subsequent changes correspond, the connective tissue becomes greater in quantity and more solid, and by its contraction compresses the muscular structure and so leads to its atrophy, and in some instances to its entire disappearance. It is not, properly speaking, a degeneration or transformation of the muscular substance, but the replacement of the muscle by fibrous tissue.

The older writers frequently speak of the conversion of portions of the heart into bone, or of bones being found in the substance of the heart, and most pathologists have met with cases of the kind. When such formations do not occur in connection with chronic pericarditis or in old false membranes, and are not traceable to the calcification of the fibrous structures around the orifices or in the valves, they take place in portions of the muscular substance which have undergone the changes now described. Such formations are not, however, to be regarded as truly bony, though they may be very hard, thick, and of large size. They consist indeed only of granules of calcareous matter, deposited in the altered tissue, without any of the elements of true bone structure.

POLYPOID GROWTHS.

Most writers on cardiac pathology mention polypoid growths as occurring in the different cavities of the heart. There can, however, be no doubt that many of the cases which have been described as of this[1] description were only instances in which decolorized coagula were adherent to the lining membrane. Such may be concluded to have been the nature of the bodies described by Dr. Ryan[2] and Mr. Stewart,[3] which have been frequently referred to by authors. In other instances, however, it may be inferred that the formations observed were new growths. Such apparently were the polypi described by Mr. Reeves, Mr. Mayo,[4] and Mr. Wilkinson King,[5] and by MM. Puisaye[6], Dubreuil,[7] Choisy,[8] and Bouillaud.[9] Most of these cases have been collected by M. Aran, in a memoir published in 1846.[10] Two other similar cases are described by Dr. Wilks[11] and Mr. Birkett,[12] in the Patho-

[1] Case of M. Renauldin; Corvisart, p. 175.
[2] Med. Gaz. vol. iii. 1829, p. 336.
[3] Ed. Med. and Surg. Jour. vol. xii. 1817, p. 182.
[4] Outlines of Human Pathology, 1836, p. 472.
[5] Lancet, 1842, vol. ii. p. 428.
[6] Gaz. Méd. de Paris, 1843, p. 270.
[7] Ibid. p. 512. Two cases, one of which is quoted by Bouillaud.
[8] Revue Médicale, 1833, tome ii. p. 425, quoted by Aran, p. 278.
[9] Vol. ii. p. 170, obs. 105.
[10] Arch. Gén. de Méd. 4me série, tome xi. 1846, p. 274.
[11] Vol. viii. p. 150. [12] Vol. i. p. 224.

logical Transactions, and one has fallen under my own notice.

The true polypoid growths appear generally to occur in the left auricle, and to be most usually attached to the fibrous zone of the auriculo-ventricular valves. Sometimes they are connected with some other part of the walls of the cavity, or are found in the right auricle or either ventricle. When in the former situation, they frequently project through the auriculo-ventricular aperture into the cavity of the left ventricle. They vary considerably in size in different instances. Some have been compared to partridge's or pigeon's eggs or to walnuts; others to hen's eggs; and yet others are stated to have filled the cavity from which they sprang. Most usually they assume a pyriform or cordate shape, and are attached to the walls of the cavity by a more or less constricted pedicle. The surface of the growths is sometimes smooth, sometimes nodulated or studded with vegetations; and most generally they are covered wholly or in part by the endocardium, this, especially at the root, being thickened and indurated. They may consist of a simple growth, or, on the contrary, may be composed of different portions. The precise nature of the bodies is not clear in the accounts of several of the published cases. Mr. Burns says, in reference to that which he has described, that it was dense, laminated, and fully organized, and closely resembled the polypi of the nose. Mr. Mayo is in doubt whether the specimen he mentions was to be regarded as a slowly growing polypus, or a medullary sarcomatous growth. In the case which occurred at the Middlesex Hospital,[1] the structure of the tumor is compared to the spleen; in that of M. Puisaye the growth is stated to have been fungous, and to have had the aspect and consistence of gelatine; and in those of M. Dubreuil, the tumors are called fibrous or albugineous. Dr. Wilks and the reporters on his case described the tumor as fibrous, and Mr. Birkett regards the specimen he exhibited as fibroid. The growths are included by Mr. Aran under the general term of "Tumeurs fongeuses sanguines." The specimen which fell under my own notice was about the size of a walnut; it was attached to the auricular surface of the mitral valve, was of a rounded form with a short and thin pedicle, and was studded on its upper surface with vegetations or granulations. It was apparently covered by endocardium throughout, and was of a pearly white color and obviously fibrous structure. The subject of the disease was a young woman who was insane and died of gangrene of the extremities, but had not during life presented any symptoms attracting attention to the heart.

The mode of origin of these growths probably varies in different cases. In some instances they may be simply adherent clots which have become organized; in others they probably originate in inflammatory exudations in the subserous cellular tissue. Indeed, this would appear to be the most usual mode of origin of the polypoid growths, for, as before stated, they generally spring from the fibrous tissue of the left auriculo-ventricular aperture and valves, and are usually covered by the endocardium. As might be expected, the cavities in which these bodies are developed are ordinarily considerably dilated; and similar effects are produced on other parts of the heart to those which would result from obstructions of any other kind in the same situation.

The polypoid growths have been met with at various ages and in both sexes, and generally in persons, who, for a longer or shorter time, have presented obvious symptoms of cardiac disease. When, as in most of the cases on record, the bodies obstruct the orifices of the heart or interfere with the action of the valves, they give rise to the ordinary effects of valvular disease, which manifest themselves by the usual signs. In one very interesting case, quoted by M. Aran from the "Annali Universali" for 1844,[1] a pulsating tumor was observed for a considerable period before the death of the patient, on the left side of the upper part of the sternum, between the cartilages of the second and third ribs; and this ultimately attained a considerable size. After death a tumor was found to occupy the upper and anterior part of the heart, and proved to be connected with the left auricle. The pericardium was inflamed and covered with recent exudation. The precise situation and character of the tumor is not clear from the description. M. Aran[2] also quotes from Schmidt, a case in which a hollow body was found filling the right auricle, passing through the auriculo-ventricular aperture, and communicating with the cavity of the aorta by an opening between the sigmoid valves.

[1] Lond. Med. Gaz. vol. xv. (1834–35), vol. i. p. 671.

[1] Supra, obs. x. p. 275.

[2] Obs. xviii. p. 287.

PNEUMO-PERICARDIUM.

By J. Warburton Begbie, M.D.

This is the term employed to designate the presence of air in the cavity of the pericardium, and may be applied to that condition, whether or not the signs of inflammatory action in the sac are present. There exist three different ways in which an accumulation of air in the pericardial sac may be determined: 1st. Such may be the direct product of the irritated membrane itself. It is admitted that, occasionally, air is produced in the cavities of the pleura and peritoneum, when these are the seat of inflammatory action, and if this be the case, there can be no reason why the same formation should not occur within the pericardium. Dr. Stokes has recorded an instance of this nature, in which, although recovery happily occurred, and the diagnosis must therefore be regarded as inferential rather than demonstrative, the opinion expressed by him seems alone tenable. "I could form," he says, "no conclusion but that the pericardium contained air in addition to an effusion of serum and coagulated lymph."[1] 2d. Air may result from the decomposition of fluid in the pericardium. Laennec and other observers have not only pointed out the physical signs which in their opinion indicate the existence of this lesion, but the former, more particularly, has in all probability greatly exaggerated the frequency of its occurrence. The effusion of fluid and air into the pericardial sac, in the opinion of Laennec, is a phenomenon likely to occur in the last stages of all diseases, and its existence he believed himself able to recognize both by percussion and auscultation. "L'épanchement liquide et aëriforme à la fois du Péricarde peut avoir lieu dans l'agonie de toutes les maladies. Il m'est arrivé quelquefois de l'annoncer à une résonnance plus claire du bas du sternum, survenue depuis peu de jours, ou à un bruit de fluctuation déterminé par les battements du cœur et par les inspirations fortes."[2] In a case recorded by M. Bricheteau, to which reference is made in Bouillaud's work, "Traité des Maladies du Cœur," as well as in a note by Andral to his edition of Laennec's Treatise, and which is also alluded to by Dr. Stokes and Dr. Walshe, the diagnosis of air as well as fluid existing in the sac of the pericardium was made during the life of the patient, and depended chiefly on the presence of a peculiar sound with the heart's action, a sound compared by Bricheteau to that produced by a water-wheel ("l'eau agitée par la roue d'un moulin"), while on examination after death the pericardium was found to be occupied by a peculiar fluid of very fetid character, air escaping with a whistling sound when the sac was opened. Acknowledging, however, the occasional occurrence during life of Pneumo-pericardium, as the result of decomposition in fluid occupying the sac, it is manifest that this source of the lesion is of much greater frequency as a post-mortem occurrence. Laennec, indeed, has acknowledged this, for after alluding to Pneumo-pericardium as of common existence in autopsies, he adds, "Et surtout de ceux (cadavres) qui ont été gardés pendant un certain temps." 3d. Air may reach the pericardium from a distance, through perforation, and the establishment of a communication between its cavity and that of any hollow organ normally containing air. Thus the sources of the air may be various, and the event may further be the result of direct injury or of disease. A very remarkable illustration is mentioned by Dr. Walshe, in which a communication was established between the œsophagus and pericardium, in an attempt to swallow a long blunt instrument, a juggler's knife—the case terminated fatally.[1] A case of traumatic Pneumo-pericardium, unattended by inflammation and resulting in complete recovery, is given by Dr. Austin Flint, to whom it was related by Dr. Knapp of Louisville. "The patient was stabbed with a knife, which penetrated the pleural cavity and perforated slightly the pericardium. A splashing sound with the heart's action was immediately heard, which continued for a few days and disappeared. The symptoms and signs, subsequently, did not denote pericarditis, but the patient had pleurisy, which was followed by considerable contraction of the

[1] Diseases of the Heart and Aorta, p. 21.

[2] Traité de l'Auscultation médiate: Des Maladies du Cœur—Du Pneumo-Péricarde, chap. xxiii.

[1] Diseases of the Heart. See pp. 46 and 271.

left side. The splashing sound in this case," continues Dr. Flint, "was fairly attributable to the presence of air and probably a little blood within the pericardium."[1] Whether the inference that no inflammation of the Pericardium succeeded the injury in this instance be correct or not, there can be no doubt that the ordinary result of a perforation of the sac, whether by wound or by communication established between it and any organ containing air, is pericarditis. Dr. Walshe observes in regard to the latter: "Now Pneumo-pericarditis must exist temporarily, be it for ever so few minutes, as the sole result of perforative communication between the pericardial sac and any hollow viscus containing gas; but in this isolated state *it has never been observed, pericarditis having supervened before clinical examination has been made.*"

After the operation of Paracentesis Pericardii and injections of iodine into the sac, physical signs have been discovered precisely similar in character to those met with in traumatic cases. Such resulted in the memorable instance recorded by the late M. Aran under the title, "Pericardite avec épanchement, traitée avec succès par la ponction et l'injection iodée."

Of communication established between the Pericardium and neighboring organs through the progress of disease, and permitting the entrance of air into the cavity of the former, several instances have been recorded by different writers. Dr. Graves has furnished a remarkable example of communication by fistulous opening between the stomach and an hepatic abscess on the one hand, and the pericardium on the other.[2] Dr. M'Dowel exhibited to the Pathological Society of Dublin the morbid appearances in a case of communication established between a cavity in the left lung and the pericardium.[3] The writer has placed on record the history of a very interesting case, in which disease of a cancerous nature primarily affecting the œsophagus, subsequently involved adjacent organs, giving rise to pericarditis with effusion, and ultimately by perforation led to Pneumo-pericardium. When the close anatomical relationship of œsophagus to the pericardium, the former lying in the posterior mediastinum in contiguity with the posterior portion of the pericardium for nearly two inches, is considered, it will be seen how, in their conditions of disease likewise, the one is very apt to influence the other. In the instance now specially referred to, a careful scrutiny had led to the opinion that rupture of the œsophagus where in contact with the pericardium, and affected by cancer, had taken place, and, as a result of the perforation, that the passage of air into the pericardial sac had occurred. Post-mortem examination confirmed the correctness of the diagnosis. On opening the chest, the pericardium, marked by the pressure of the ribs, bulged forwards, and on being punctured air escaped. Several ounces of dark-brown fetid fluid existed in the sac: lymph, recent in its deposition, and of yellowish color, coated the inner surface of the membrane. Cancerous ulceration, and destruction to a considerable extent of the wall of the œsophagus existed, corresponding to its usual point of contact with the pericardium.[1]

In the diagnosis of Pneumo-pericardium, of Pneumo-hydropericardium, and Pneumo-pericarditis, reliance may reasonably be placed on the physical signs as determined by percussion and auscultation. Laennec, who, as already observed, exaggerated the frequency of the occurrence of air in the pericardial sac before death, speaks of three signs to be expected when air and fluid exist in the pericardium. 1. Unusual resonance over the lower part of the sternum: 2. Fluctuation sound ("bruit de fluctuation") audible with the action of the heart, and on deep inspiration. 3. This specially relating to the diagnosis of simple Pneumo-pericardium, that is, without fluid effusion, or inflammatory product; the heart's sounds being heard at a distance from the chest. Upon this sign the distinguished inventor of auscultation placed very great reliance. Dr. Stokes, whose entire observations on the subject of Pneumo-pericarditis are most instructive, noticed the fact of the heart's sounds being heard at a distance in a case which he has recorded; he remarks, however, that this indication did not exist in the instances of Dr. Graves and Dr. M'Dowel, already alluded to. Auscultation over the region of the heart, when practised by the writer in the case which fell under his own observation, revealed the probable existence of air and fluid in the pericardial sac, by the extraordinary guggling sound which accompanied the heart's action—a sound which cannot, he thinks, be better described than as a churning splash. Dr. Stokes gives the following description of the sounds which he observed:—"They were not the rasping sounds of indurated lymph, or the leather creak of Collin, nor

[1] A Practical Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart. By Austin Flint, M.D.

[2] Clinical Lectures, edited by Dr. Neligan. Edition of 1864, page 616.

[3] See Dr. Stokes's work, p. 23, also p. 35; and Dr. Walshe's work, p. 271.

[1] Observations in Clinical Medicine, by J. Warburton Begbie, M.D. Edinburgh Medical Journal, 1862.

those proceeding from pericarditic with valvular murmurs, but a mixture of various attrition murmurs with a large crepitating and guggling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met so extraordinary a combination of sounds. The stomach was not distended by air, and the lung and pleura were unaffected, but the region of the heart gave a tympanitic *bruit de pot fêlé* on percussion, and I could form no other conclusion but that the pericardium contained air in addition to an effusion of serum and coagulable lymph." The phenomena on auscultation and percussion thus graphically described by Dr. Stokes, will receive a farther value as indicating the existence of Pneumo-pericarditis, if in addition there be noticed, as was done by Dr. Walshe in the singular case of traumatic communication between the œsophagus and pericardium, a dull or tympanitic sound over the precordial region, according to the position assumed by the patient. Even without this indication, and in default of a metallic character attaching itself to the cardiac sounds, as noticed by Dr. Stokes, the diagnosis of Pneumo-pericarditis, or, to be still more explicit, of Hydropneumo-pericarditis, may be made from observing a guggling or churning splash sound with the heart's action limited to the cardiac region, with which more or less of tympanitic precordial resonance on percussion is associated. These signs will be still more available, if the guggling sound has been noticed to succeed a distinct friction sound, and the tympanitic has replaced a dull percussion note.

It is satisfactory to note that the phenomena to which attention has now been called, and which serve to indicate the existence of a very serious lesion, are not necessarily of a fatal import. In Dr. Stokes's case, as already noticed, recovery resulted, and in the instance of Pneumo-pericardium, traumatic in origin, noticed by Dr. Knapp, and recorded by Dr. Flint, the termination was equally gratifying. We may indulge the hope that the records of medicine may yet contain other examples of a similar nature.

PERICARDITIS.

By Francis Sibson, M.D., F.R.S.

CLINICAL HISTORY OF PERICARDITIS AS IT OCCURRED IN THE AUTHOR'S PRACTICE IN ST. MARY'S HOSPITAL.

Inflammation of the surface of the heart and the lining of the pericardial sac occurs so very rarely by itself, and is so generally one of the attendant affections of a general disease, such as acute rheumatism, Bright's disease, and pyæmia or the secondary inflammations; or of a local affection, such as aneurism of the aorta or cancer; or of a local injury; that we cannot practically regard it as a distinct disease. Pericarditis is, indeed, with very rare exceptions, one of the inflammations attendant upon those diseases or injuries.

Pericarditis occurs so much more frequently in acute rheumatism than in any other disease, that I shall first consider the affection as it exists in connection with that disease; and in so doing shall examine the proportion of my cases of acute rheumatism that were affected with Pericarditis, and shall describe the progress of that affection in those cases.

RHEUMATIC PERICARDITIS.

I possess notes of 326 cases of acute rheumatism that were admitted under my care into St. Mary's Hospital during the fifteen years ending in the autumn of 1866. This number does not include fourteen patients in whom it was doubtful whether the affection was acute rheumatism or acute gout.

One-fifth of those cases[1] (63) were attacked with Pericarditis, which was ac-

[1] In two of those cases (59, 61) the evidence of pericarditis was slight and perhaps doubtful, but I am of opinion that in both of them the affection existed though in a slight and transient form. The numbers thus given here and elsewhere refer to the individual cases of Pericarditis as they occur in my records, so that the reader may trace for himself each of those cases as it appears from part to part of this analysis.

companied in all but nine instances (54) by endocarditis, and fully one-third of them with simple endocarditis (108), while in only one-fourth of them was there no evidence of either endocarditis or pericarditis (79). There was, however, an intermediate group, amounting nearly to one-fourth of the whole number (76), in which endocarditis, though not established, was either threatened or probable, the signs of that affection being either transient or imperfect. I think that we may class this intermediate group arbitrarily into two divisions, and consider that in one-half of them there was endocarditis, and that in the other half there was no endocarditis.

If we add the cases of pericarditis that were also affected with endocarditis (54) and half of those in which endocarditis was threatened or probable (38), to those in which simple endocarditis was present (108), we shall find that in my patients inflammation of the interior of the heart (200) was fully three times as frequent as inflammation of the exterior of the heart (63).

This summary, otherwise stated, stands thus :—

Cases of acute rheumatism with Pericarditis	63	
Cases in which the Pericarditis was accompanied by endocarditis		54
Cases of simple endocarditis .	108	
Cases of threatened or probable endocarditis	76	
Cases in which there was no sign of endocarditis	79	
Total number of cases of acute rheumatism	326	

I.—Sex, Age, and Occupation in Acute Rheumatism in especial relation to Pericarditis.

Sex.—Acute rheumatism affected the female sex somewhat more frequently than the male sex in the proportion of 168 to 158.

Pericarditis attacked 35 male and 28 female patients, so that nearly one in four of the former (35 in 154), and only one in six of the latter (28 in 166) were affected by it. Endocarditis was also present in 31 of the male and 23 of the female patients affected with pericarditis.

Simple endocarditis, on the other hand, attacked 47 male and 61 female patients, while, in addition, endocarditis was threatened or probable in 32 male and 41 female patients.

The cause of the greater proportional frequency of Pericarditis, usually accompanied by endocarditis, in the male sex, and of simple endocarditis in the female sex in these cases, will, I think, be in part explained by the influence of age and occupation on acute rheumatism and its complications.

Age.—One-half of the male (17 in 34)[1] and more than one-half of the female patients (17 in 27)[1] affected with Pericarditis, were below the age of 21 : while two-fifths of the male (13 in 34) and only one-seventh of the female patients (4 in 27) were above the age of 25.

If we group these two classes of cases separately in relation to age, and compare them with each other, we find that acute rheumatism attacked 70 male and 77 female patients *below the age of* 21, and that of these 17 of each sex were affected with Pericarditis, combined with endocarditis in all but one or two cases, and 25 of the males and 32 of the females with simple endocarditis ; that in 12 of the males and 20 of the females endocarditis was threatened or probable, and that in 15 of the males and in only 8 of the females there was no sign of inflammation of the heart, within or without.

On the other hand, we find that acute rheumatism affected 53 men and 53 women *above the age of* 25, and that of these 13 men (13 in 53 or one-fourth) and only 4 women (4 in 53 or one-thirteenth) were affected with Pericarditis which was usually accompanied by endocarditis, and 13 men and 17 women with simple endocarditis ; that in 11 men and 11 women endocarditis was threatened or probable ; and that the residue, or 16 men and 21 women, gave no sign of inflammation of the heart.

The accompanying Table shows the proportion in which endocarditis and Pericarditis were absent or present in the cases of acute rheumatism, and the influence of age and sex in the proportionate production of those affections of the heart in that disease.

[1] The age of one of the 35 male patients and that of one of the 28 female patients was not stated.

	No Endocarditis			Endocarditis threatened or probable.									Endocarditis			Pericarditis.			Total.		
				Threatened.			Probable.			Total.												
Male..... 42 / 37 } 79 Female				26 / 37 } 63			8 / 5 } 13			34 / 42 } 76			47 / 61 } 108			35 / 28 } 63			158 / 168 } 326		
Ages.	Male.	Female	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male	Female.	Total.
10 to 15	5	3	8	2	1	3	0	1	1	2	2	4	10	9	19	6	3	9	23	17	40
16 to 20	11	5	16	9	14	23	1	4	5	10	18	28	15	23	38	11	14	25	47	60	107
21 to 25	10	8	18	6	9	15	4	0	4	10	9	19	8	11	19	4	6	10	32	34	66
26 to 30	8	13	21	3	5	8	2	0	2	5	5	10	8	6	14	5	1	6	26	25	51
31 to 40	5	4	9	6	5	11	0	0	0	6	5	11	4	10	14	6	2	8	21	21	42
41 to 50	3	3	6	0	1	1	0	0	0	0	1	1	1	0	1	2	0	2	6	4	10
51 and —	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1	0	1	1	0	3	3
?.....				0	2	2	1	0	1	1	2	3	1	1	2	1	1	2	3	4	7
Total....	42	37	79	26	37	63	8	5	13	34	42	76	47	61	108	35	28	63	158	168	326

We thus see that in these cases of acute rheumatism, inflammation of the heart, grouping together those in which it attacked the interior and the exterior of the organ, affected the young below 21 (91 in 147) more frequently than the adult above 25 (47 in 106); that the heart was more frequently free from signs of inflammation in the adult above 25 (37 in 106), and especially in women (21 in 53), than in the young below 21 (24 in 147), and especially in girls (8 in 77); that endocarditis was threatened or probable as often in the young below 21 (32 in 147) as in the adult above 25 (22 in 106); and, this being the point to which I would especially call attention that Pericarditis—while it affected the two sexes in nearly equal proportions below the age of 21, the male patients (17 in 70) a little more frequently than the female patients (17 in 77)—attacked men above the age of 25 (13 in 53) three times more frequently than women above that age (5 in 53).

Occupation.—The study of the influence of occupation on the occurrence of acute rheumatism and on the production of inflammation of the heart, both outside and in, throws light in two directions, one on the influence of sex, the other on that of age in producing those affections.

The accompanying Tables show (I. pages 478–81) the influence of occupation in acute rheumatism in relation to age; the presence or absence of endocarditis and Pericardisis; the degree of the affection of the joints, and that of the heart: and (II. pages 482–5), for the sake of comparison, of ages (1) of 1000 patients, taken consecutively, with an occasional break, from my hospital books, affected with all other internal diseases besides acute rheumatism and acute gout, and (2) of 326 cases of acute rheumatism with its attendant Pericarditis and Endocarditis, in relation to occupation.

I take female domestic servants first, since they formed nearly one-third (101 in 326) of the whole number of those of both sexes, and nearly three-fifths of those of the female sex (101 in 168) who were affected with acute rheumatism. Among those patients affected with other diseases than acute rheumatism, female servants formed one-fifth of the whole number (204 in 1000) and two-fifths of the female patients (204 in 453). Nearly two-thirds of the female patients affected with acute rheumatism were below the age of 21 (57 in 100), while of those affected with other diseases, only one-third were below that age (64 in 195, or 33 per cent.).

The influence of that employment in causing Pericarditis and endocarditis in acute rheumatism, especially below the age of 21, is remarkable. Of the whole number of 101 servants only 13—one-eighth—presented no sign of inflammation of the heart, while one-fifth of them (19) were attacked with Pericarditis, accompanied in all but one instance with endocarditis also, and two-fifths of them (43) with simple endocarditis, while in the remaining fourth part (26) endocarditis was either threatened or probable. Servants formed fully two-thirds of the whole of the female patients affected with Pericarditis complicated usually with endocarditis (19 in 28), and with simple endocarditis (42 in 60); and three-fifths of those in whom endocarditis was threatened or probable (26 in 42): while they formed only one-third of those who gave no sign of affection of the heart (13 in 37).

The influence of age in inducing inflammation of the heart in servants affected with acute rheumatism is still more remarkable. Of the whole number of servants (101) attacked with that disease, 57 were below the age of 21. In only 3 of these was there no mark of affection of the heart, but one-fourth of them (14) were attacked with Pericarditis, all of whom had endocarditis also, and nearly one-half of them (25) with simple endocarditis, while endocarditis was either

threatened or probable in the remaining 15. Three-fourths of the servants attacked with Pericarditis and endocarditis (14 in 19), and three-fifths of those with simple endocarditis (26 in 42) were below the age of 21, while only one-fourth of those who were quite free from symptoms of heart affection were below that age (3 in 13).

Girls engaged in the hard labor of a servant, at work, at a tender age, from morning to night, when attacked with this disease to which they are so subject, are all but certain to have inflammation of the heart without or within. Servant-girls below the age of 21, keeping in view their time of life and constitution, are more exposed to the causes of acute rheumatism and its attendant inflammation of the heart than persons of any other class. They are growing, their frame is not yet knit, they are sensitive to cold and wet, and they are subject to palpitation. Before all, in these young women their joints are not yet perfected, the ends of the bones forming them being still united to their shafts by cartilage; their growth is active so that the blood circulates in them freely; their structures are sensitive; and while they are supple, and their play is free and lively, they are tender and do not bear undue pressure; they are liable to strains, are unequal to labor and fatigue,and are easily affected by draughts, and by exposure to wet and cold, especially after undue and prolonged exertion. Then the labor of these poor girls, especially in hard places of service, is great and constant; they carry weights up and down stairs, often in lofty houses; they are constantly on foot, standing rather than walking, so that full pressure is continuously made on the joints; or what is worse, they are kneeling sometimes on cold and even wet stone floors, hard at work, scrubbing and brushing.

The joint affection was, as a rule, more severe in servants suffering from acute rheumatism than in the rest of those so affected, the joints being attacked with severity in one-half of the servants (49 in 101), and a little over one-third of the rest (91 in 225). Among those servants who suffered from Pericarditis, the joint affection was severe in fully three-fourths (15 in 19), and in a large proportion of these (6) it was very severe. If we compare these cases with the rest of the servants affected with acute rheumatism, we find that the severity of the joint affection rose in the scale in exact proportion to the severity of the heart affection. The joint affection was severe in less than one-third (4 in 13) of those servants who presented no sign of inflammation of the heart, while it was so in a little over a third (9 in 26) of those in whom endocarditis was threatened or probable, and in one-half of those who were attacked with simple endocarditis (21 in 42); while, as I have just said, it was severe in three-fourths of the cases with Pericarditis (15 in 19).

In the servants who were attacked with Pericarditis, the severity of the joint affection bore a strict relation to the severity of the heart affection in the great majority of the cases.

In one-third of them (6 in 19) the joint affection was very severe; and in the whole of these the heart affection was very severe, while in one of them it was fatal.

In nearly one-half of these patients (9 in 19) the joint affection was severe in the second degree, and in two-thirds of these (6 in 9) the heart affection was severe; in two cases it was rather severe; and in one it was slight. In three patients the joint affection was rather severe, and of these the heart affection was severe in one, rather so in a second, and not so in a third.

The last case is a notable exception to this rule. The attack in the joints was slight, but the attack at the heart was very severe, and proved fatal.

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TABLE I.—ACUTE

OCCUPATION IN RELATION TO AGE, THE DEGREE

Male Patients.	Patients in whom					
	There was no indication of Endocarditis.			Endocarditis was threatened or probable.		
	Number.	Years.	Joint affection.	Number.	Years.	Joint affection.
Out-of-door Employments.						
Engaged in *laborious employments in the open air*, including laborers (17), gardeners (5), bricklayer, brickmaker, sawyer (in twice), mason, dustman, carter, plasterer, seaman, smiths (4), butchers (6), carpenters (4)	9	2 æt. 20 1 " 24 2 " 26 to 27 2 " 32 " 37 2 " 42 " 46	1 severe 6 rather sev. 2 not severe	11	3 æt. 20 4 " 22 to 25 1 " 26 " 29 3 " 31 " 38	6 severe 4 rather sev. 1 not severe
Engaged in *employments chiefly on foot, in the open air*, including porters (4), watchman, errand boys (4), milkmen (2), hawker, cowkeeper, out of work—"on tramp" (1)	7	2 æt. 10 to 12 3 " 16 " 20 1 " 28 1 " 38	1 severe 3 rather sev. 1 not severe 1 slight 1 doubtful	2	1 " 14 1 " 21	2 rather sev.
Employed *with horses and in stables*, including grooms (6), riding-master, horsekeeper, coachmen (9), cabmen (5).....	7	2 æt. 19 1 " 23 2 " 26 1 " 38 1 " 43	3 severe 4 rather sev.	7	4 " 16 " 19 2 " 21 " 24 1 " 27	4 severe 3 rather sev.
Total of those employed in the above laborious *out-of-door* employments	23	2 æt. 10 to 12 7 " 16 " 20 2 " 23 " 24 5 " 26 " 28 4 " 32 " 38 3 " 42 " 46	5 severe 13 rather sev. 3 not severe 1 slight 1	20	1 " 14 7 " 16 " 20 7 " 21 " 25 2 " 26 " 29 3 " 31 " 38	10 severe 9 rather sev. 1 not severe
Painters (3), plumber, gas-fitter...	2	1 æt. 17 1 " 26	1 rather sev. 1 not severe	2	1 " 16 1 " 21	1 severe 1 rather sev.
Commercial traveller.....	....		....	....		....
In-door Employments.						
Including servants (12), bakers (3), paperhanger, French-polishers, (4), boot and shoemakers (3), "shopman," greengrocer, drapers (4), cheesemonger, slop cutter, tailor, teacher, silversmith, chairmaker, bath-attendant, and two others.....	7	1 æt. 14 2 " 16 to 18 2 " 25 2 " 27 " 28	3 severe 2 rather sev. 1 not severe 1 slight	9	2 " 19 1 " 25 3 " 29 3 " 33 " 37	1 very severe 4 severe 4 rather sev.
Waiters (6), potmen and barmen (5)	8	1 æt. 19 6 " 21 to 23 1 " 40	2 severe 5 rather sev. 1 not severe	1	21	1 rather sev.
Schoolboys (5)	....		....	....		....
Had no employment, including one discharged from the navy (12) ..	2 æt. 14 to 15		1 rather sev. 1 slight	2	1 " 14 1 not stated	1 severe 1 slight
Occupation no stated.....	....		....	....		....

RHEUMATISM.

OF JOINT AFFECTION, AND THAT OF HEART AFFECTION.

Patients in whom there was								
Simple Endocarditis.			Pericarditis, usually with Endocarditis (54 in 63).			Total		
No.	Years.	Joint affec'n.	No.	Years.	Joint affec'n.	Heart affec'n.	No.	Years.	Joint affec'n.
15	6 æt. 16 to 20 4 " 21 " 24 3 " 26 " 29 2 " 35 " 38	10 severe 5 rather sev.	10	1 æt. 17 to 19 1 " 22 " 24 4 " 27 " 28 3 " 31 " 39 1 " 42	4 very sev. 4 severe 2 rather sev.	1 fatal 3 very sev. 2 severe 3 rather sev. 1 not severe	45	12 æt. 17 to 20 10 " 21 " 24 10 " 26 " 30 10 " 31 " 40 3 " 42 " 46	4 very sev. 21 severe 17 rather sev. 3 not severe
3	1 " 12 1 " 17 1 " 22	1 severe 1 not severe 1 slight	2	1 " 17 1 " 25	1 severe 1 not severe	2 severe	14	4 " 10 " 14 5 " 16 " 20 3 " 22 " 28 1 " 28 1 " 38	3 severe 5 rather sev. 3 not severe 2 slight 1 doubtful
8	3 " 18 " 20 2 " 21 1 " 26 1 " 33 " 38 1 " 42	2 severe 5 rather sev. 1 not severe	1	" 22	1 severe	1 severe	23	9 " 16 " 20 6 " 21 " 24 4 " 26 " 28 2 " 33 " 38 2 " 42 " 43	10 severe 12 rather sev. 1 not severe
26	1 " 12 10 " 16 " 20 7 " 21 " 24 4 " 26 " 29 3 " 33 " 38 1 " 42	13 severe 10 rather sev. 2 not severe 1 slight	13	2 " 17 " 19 3 " 22 " 25 4 " 27 " 28 3 " 31 " 39 1 " 42	4 very sev. 6 severe 2 rather sev. 1 not severe	1 fatal 3 very sev. 5 severe 3 rather sev. 1 not severe	82	4 " 10 " 15 26 " 16 " 20 19 " 21 " 25 15 " 26 " 30 13 " 31 " 40 5 " 41 " 46	4 very sev. 34 severe 34 rather sev. 7 not severe 2 slight 1 doubtful
....		....	1	" 35	1 very sev.	1 severe	5	2 " 16 " 18 1 " 21 1 " 26 1 " 35	1 very sev. 1 severe 2 rather sev. 1 not severe
....		....	1	" 18	1 severe	1 very sev.	1	" 18	1 severe
9	5 " 18 " 19 1 " 22 2 " 29 " 30 1 " 38	4 severe 3 rather sev. 2 not severe	13	2 " 14 8 " 17 " 20 1 " 23 1 " 38 1 " 50	6 severe 4 rather sev. 2 not severe 1 slight	1 fatal 2 very sev. 4 severe 4 rather sev. 2 not severe 1 slight	38	3 " 14 17 " 16 " 20 5 " 21 " 25 7 " 27 " 30 5 " 33 " 38 1 " 50	1 very sev. 17 severe 13 rather sev. 5 not severe 2 slight
2 " 27 " 30		1 rather sev. 1 not severe	1	" 39	1 severe	1 rather sev.	12	1 " 19 7 " 21 " 23 2 " 27 " 30 1 " 39 1 " 40	3 severe 7 rather sev. 2 not severe
3 " 9 " 15		2 rather sev. 1 not severe	2	" 11 " 15	1 rather sev. 1 not severe	1 severe 1 rather sev.	5	" 9 " 15	3 rather sev. 2 not severe
6 " 9 " 15		3 severe 3 rather sev.	2	" 14 " 15	2 rather sev.	1 rather sev. 1 slight	12	11 " 9 " 15 1 not stated	4 severe 6 rather sev. 2 slight
1 not stated		1 rather sev.	2	1 " 26 1 not stated	2 rather sev.	1 severe 1 rather sev.	3	1 " 26 2 not stated	3 rather sev.

TABLE I.—ACUTE

Female Patients.	Patients in whom					
	There was no indication of Endocarditis.			Endocarditis was threatened or probable.		
	Number.	Years.	Joint affection.	Number.	Years.	Joint affection.
Active In-door Employments.						
Servants	13	3 æt. 19 to 20 4 " 21 " 25 6 " 26 " 30	4 severe 5 rather sev. 4 not severe	26	1 æt. 15 14 " 16 to 20 4 " 22 " 25 3 " 26 " 30 3 " 31 " 40 1 not stated	9 severe 11 rather sev. 6 not severe
Cooks (5), charwomen (2), nurses (5), laundresses (9), washerwoman	6	2 æt. 26 to 28 1 " 36 2 " 42 " 50 1 " 52	2 severe 1 rather sev. 3 not severe	7	3 æt. 19 to 20 2 " 21 " 24 1 " 26 1 " 40	3 severe 4 rather sev.
Sedentary In-door Employments.						
Needlewomen (3), milliners, dressmakers (3), tailoress, shoebinder, shoemaker	4	1 æt. 20 2 " 26 1 " 49	1 severe 2 rather sev. 1 not severe		1 æt. 44	1 rather sev.
Kept a stall		....	....		1 æt. 21	1 severe
Married women, without special occupation, including 2 widows	8	3 æt. 25 2 " 28 to 30 3 " 36 " 40	4 severe 2 rather sev. 2 not severe	5	2 æt. 25 1 " 30 1 " 32 1 not stated	2 severe 3 rather sev.
Of no occupation	5	3 æt. 13 to 15 1 " 16 1 " 25	1 severe 1 rather sev. 2 not severe 1 slight	2	1 æt. 6 1 " 18	1 severe 1 rather sev.
Occupation not stated		æt. 29	1 rather sev.		....	....
Male Patients—total	42	5 æt. 10 to 15 11 " 16 " 20 10 " 21 " 25 8 " 26 " 30 5 " 31 " 40 3 " 41 " 46	10 severe 22 rather sev. 6 not severe 3 slight 1 doubtful	34	2 æt. 14 10 " 16 to 20 10 " 21 " 25 5 " 26 " 30 6 " 31 " 38 1 not stated	1 very severe 16 severe 15 rather sev. 1 not severe 1 slight
Female Patients—totals	37	3 æt. 13 to 15 5 " 16 " 20 8 " 21 " 25 13 " 26 " 30 4 " 31 " 40 3 " 42 " 50 1 " 52	12 severe 12 rather sev. 12 not severe 1 slight	42	2 æt. 6 to 15 18 " 16 " 20 9 " 22 " 25 5 " 26 " 30 5 " 31 " 40 1 " 44 2 not stated	16 severe 20 rather sev. 6 not severe
Grand total of the Male and Female Patients	79	8 æt. 10 to 15 16 " 16 " 20 18 " 21 " 25 21 " 26 " 30 9 " 31 " 40 6 " 41 " 50 1 " 52	22 severe 34 rather sev. 18 not severe 4 slight 1 doubtful	76	4 æt. 6 to 14 28 " 16 " 20 19 " 21 " 25 10 " 26 " 30 11 " 31 " 40 1 " 44 3 not stated	1 very severe 32 severe 35 rather sev. 7 not severe 1 slight

4 of these cases died

RHEUMATISM—(*Continued*).

Patients in whom there was						
Simple Endocarditis.			Pericarditis, usually with Endocarditis (54 in 68).			Total	
No.	Years.	Joint affec'n.	No.	Years.	Joint affec'n	Heart affec'n.	No.	Years.	Joint affec'n.
43	4 æt. 12 to 15 21 " 16 " 20 10 " 21 " 25 4 " 26 " 30 3 " 31 " 40 1 " 55	2 very sev. 19 severe 13 rather sev. 7 not severe 1 slight 1 doubtful	19	1 æt. 15 13 " 16 to 20 4 " 21 " 25 1 " 26	6 very sev. 9 severe 3 rather sev. 1 not severe	2 fatal 8 very sev. 6 severe 2 rather sev. 1 not severe	101	6 æt. 12 to 15 51 " 16 " 20 22 " 21 " 25 14 " 26 " 30 6 " 31 " 40 1 " 55 1 not stated	8 very sev. 41 severe 32 rather sev. 18 not severe 1 slight 1 doubtful
4	1 " 21 1 " 27 2 " 33 " 40	2 severe 2 slight	5	1 " 20 1 " 21 1 " 35 1 " 60 1 not stated	1 severe 2 rather sev. 1 not severe 1 doubtful	1 severe 2 rather sev. 1 not severe 1 slight	22	4 " 19 " 20 4 " 21 " 25 4 " 26 " 30 5 " 31 " 40 2 " 42 " 50 2 " 52 " 60 1 not stated	8 severe 9 rather sev. 4 not severe 1 doubtful
4	1 " 18 3 " 31 " 38	3 rather sev. 1 not severe	....	.....	.....		9	2 " 18 " 20 2 " 26 3 " 31 " 38 2 " 44 " 49	1 severe 6 rather sev. 2 not severe
	1 " 33	1 severe	....	.....	.....		2	1 " 21 1 " 33	2 severe
2	1 " 30 1 " 40	1 severe 1 rather sev.	2	1 " 24 1 " 34	1 very sev. 1 rather sev.	1 severe 1 slight	17	6 " 24 " 25 4 " 28 " 30 6 " 34 " 40 1 not stated	1 very sev. 7 severe 7 rather sev. 2 not severe
7	5 " 13 " 15 1 " 18 1 not stated	3 severe 3 rather sev. 1 not severe		2 " 13 " 14	1 severe 1 slight	1 very sev. 1 slight	16	11 " 6 " 15 3 " 16 " 20 1 " 25 1 not stated	6 severe 6 rather sev 3 not severe 1 slight
....	.....	....	.....	.....		1 " 29			1 rather sev.
47	10 æt. 9 to 15 15 " 16 " 20 8 " 21 " 25 8 " 26 " 30 4 " 33 " 38 1 " 42 1 not stated	20 severe 20 rather sev. 6 not severe 1 slight	35	6 æt. 11 to 15 11 " 16 " 20 4 " 22 " 25 5 " 27 " 30 6 " 31 " 39 2 " 41 " 50 1 not stated	5 very sev. 14 severe 11 rather sev. 4 not severe 1 slight	2 fatal 6 very sev. 12 severe 11 rather sev. 2 not severe 2 slight	158	23 æt. 9 to 15 47 " 16 " 20 32 " 21 " 25 26 " 26 " 30 21 " 31 " 40 6 " 41 " 50 3 not stated	6 very sev. 60 severe 68 rather sev. 17 not severe 6 slight 1 doubtful
61	9 æt. 12 to 15 23 " 16 " 20 11 " 21 " 25 6 " 26 " 30 10 " 31 " 48 1 " 55 1 not stated	2 very sev. 26 severe 22 rather sev. 9 not severe 1 slight 1 doubtful	28	3 " 13 " 14 6 " 16 " 20 6 " 21 " 25 1 " 26 2 " 34 " 35 1 " 60 1 not stated	7 very sev. 11 severe 7 rather sev. 2 not severe 1 doubtful	2 fatal 9 very sev. 9 severe 4 rather sev. 2 not severe 1 slight 1 doubtful	168	17 " 6 " 15 60 " 16 " 20 34 " 21 " 25 25 " 26 " 30 21 " 31 " 40 4 " 42 " 50 3 " 52 " 60 4 not stated	9 very sev. 65 severe 61 rather sev. 29 not severe 2 slight 2 doubtful
1081	19 æt. 9 to 15 38 " 16 " 20 19 " 21 " 25 14 " 26 " 30 14 " 31 " 41 1 " 42 1 " 55 2 not stated	2 very sev. 46 severe 42 rather sev. 15 not severe 2 slight 1 doubtful	632	9 æt. 11 to 15 25 " 16 " 20 10 " 21 " 25 6 " 26 " 30 8 " 31 " 40 2 " 41 " 50 1 " 60 2 not stated	12 very sev. 25 severe 18 rather sev. 6 not severe 1 slight 1 doubtful	4 fatal 15 very sev. 21 severe 15 rather sev. 4 not severe 3 slight 1 doubtful	326	40 æt. 6 to 15 107 " 16 " 20 66 " 21 " 25 51 " 26 " 30 42 " 31 " 40 10 " 41 " 50 3 " 55 " 60 7 not stat'd	15 very sev. 125 severe 129 rath. sev. 46 not severe 8 slight 3 doubtful

[1] 1 of these cases died.

[2] 5 of these cases died (1 from Bright's disease).

TABLE II.

AGES OF I.—1000 PATIENTS AFFECTED WITH ALL OTHER INTERNAL DISEASES EXCEPT ACUTE RHEUMATISM, WITH ITS ATTENDANT PERICARDITIS AND ENDOCARDITIS, AND

MALE PATIENTS.		BELOW THE AGE OF 21 YEARS.	FROM 21 TO 25 YEARS.
Workers out of doors. Laborious employments .	Other diseases except acute rheumatism and acute gout	21, or 10 per cent. of those whose ages are stated,	31, or 15 per cent.
	Acute rheumatism	12, or 26·6 per cent.	10, or 22·2 per cent.
	Ditto with pericarditis . .	1, or 8·3 per ct. at that age.[1] 1, or 10 per cent. of whole.[2]	1, or 10 per cent. at that age. 1, or 10 per cent. of whole.
	Ditto with simple endocard.	6, or 50 per cent of that age. 6, or 40 per cent. of whole.	4, or 40 per cent. at that age. 4, or 27 per cent. of whole.
	Acute gout	0	2
Workers on foot	Other diseases except acute rheumatism and acute gout	6, or 17·7 per cent.	4, or 11·7 per cent.
	Acute rheumatism	9, or 64 per cent.	3, or 21·4 per cent.
	Pericarditis	1, or 11 per cent. at that age. 1, or 50 per cent. of whole.	1, or 33·3 per ct. at that age. 1, or 50 per cent. of whole.
	Endocarditis	2, or 22 per cent. at that age. 2, or 66·7 of whole.	1, or 33·3 per ct. at that age. 1, or 33·3 per cent. of whole.
	Acute gout	0	0
Workers among horses . . .	Other diseases except acute rheumatism and acute gout	1, or 1·5 per cent.	4, or per cent.
	Acute rheumatism	9, or 39 per cent.	6, or 26 per cent.
	Pericarditis	0	1, or 16·6 per ct. at that age. 1, or 10 per cent. of whole.
	Endocarditis	3, or 33·3 per ct. at that age. 3, or 37·2 per cent. of whole.	2, or 30·9 per ct. at that age. 2, or 25 per cent. of whole.
	Acute gout	0	1
Painters, plumbers . . .	Other diseases besides acute rheumatism and acute gout	2, or 5 per cent.	9, or 22 per cent.
	Acute rheumatism	2, or 40 per cent.	1, or 20 per cent.
	Pericarditis	0	0
	Endocarditis	0	0
	Acute gout	0	0
In-door employments . . .	Other diseases besides acute rheumatism and acute gout	20, or 16·3 per cent.	20, or 16·3 per cent.
	Acute rheumatism	20, or 52·5 per cent.	5, or 13·5 per cent.
	Pericarditis	10, or 50·6 per ct. at that age. 10, or 77 per cent. of whole.	1, or 20 per cent. at that age. 1, or 7·7 per cent. of whole.
	Endocarditis	5, or 25 per cent. at that age. 5, or 55·2 per cent. of whole.	1, or 20 per cent. at that age. 1, or 11 per cent of whole.
	Acute gout	0	2
Waiters, barmen, and one commercial traveller . .	Other diseases besides acute rheumatism and acute gout	2, or 16·6 per cent.	2, or 16·6 per cent.
	Acute rheumatism	2, or 15·3 per cent.	7, or 54·1 per cent.
	Pericarditis	1, or 50 per cent. at that age. 1, or 50 per cent. of whole.	0
	Endocarditis	0	0
	Acute gout	0	0
Of no occupation and at school . . .	Other diseases except acute rheumatism and acute gout	37, or 100 per cent.	0
	Acute rheumatism	16, or 100 per cent.	0
	Pericarditis	4, or 25 per cent. at that age.	0
	Endocarditis	9, or 56·6 per ct. at that age.	0
TOTAL OF MALE PATIENTS .	Other diseases except acute rheumatism and acute gout	89, or 17 per cent.	70, or 13·4 per cent.
	Acute rheumatism	70, or 45 per cent.	32, or 20·8 per cent.
	Pericarditis	17, or 21·3 per ct. at that age. 17, or 51·5 per cent. of whole.	4, or 12 per cent. at that age. 4, or 12 per cent of whole.
	Endocarditis	25, or 35·7 per ct. at that age. 25, or 54·3 per cent. of whole.	8, or 24·2 per ct. at that age. 8, or 17·4 per cent. of whole.
	Acute gout	0	5

[1] Here and elsewhere in these columns add after "age" of those with acute rheumatism who were so affected and who were engaged in the class of employments indicated in the column headed "Male Patients."

[2] Here and elsewhere in these columns "whole" applies to the whole number of all ages of those with acute

TABLE II.

ACUTE RHEUMATISM AND ACUTE GOUT, AND II.—326 PATIENTS AFFECTED WITH III.—58 PATIENTS AFFECTED WITH ACUTE GOUT, IN RELATION TO OCCUPATION.

ABOVE 25 YEARS.	AGE AND OCCUPATION NOT STATED.	TOTAL.	
55, or 75 per cent.	Age not stated 14	221	Or { 40·4 per cent. of the males. 22·1 per cent. of the whole.[1]
23, or 51 per cent.	.. ..	45	Or { 29 per cent. of the males. 14 per cent. of the whole.
8, or 35 per cent. at that age. 8, or 80 per cent. of whole.	.. ..	10	{ Or 22 per cent. of those with acute rheumatism.
5, or 21 per cent. at that age. 5, or 33 per cent. of whole.	.. ..	15	{ Or 33·3 per cent. of those with acute rheumatism.
13	.. ..	15	
24, or 70·6 per cent.	 4	38	Or { 6·9 per cent. of the males. 3·8 per cent. of the whole.
2, or 14·3 per cent.	.. ..	14	Or { 9 per cent. of the males. 4·3 per cent. of the whole.
0 0	.. ..	2	{ Or 14 per cent. of those with acute rheumatism.
0 2	.. ..	3	{ Or 21·4 per cent. of those with acute rheumatism.
61, or 93 per cent.	 3	69	Or { 12·6 per cent. of the males. 6·9 per cent. of the whole.
8, or 35 per cent.	.. ..	23	Or { 14·5 per cent. of the males. 7·1 per cent. of the whole.
0	.. ..	1	{ Or 4·3 per cent. of those with acute rheumatism.
3, or 37 per cent. at that age. 3, or 37·5 per cent. of whole.	.. ..	8	{ Or 35 per cent. of those with acute rheumatism.
14	.. ..	15	
30, or 73 per cent.	.. ..	41	Or { 9·5 per cent. of the males. 4·1 per cent. of the whole.
2, or 40 per cent.	.. ..	5	Or { 3·2 per cent. of the males. 1·5 per cent. of the whole.
1, or 50 per cent. at that age. 0 4	.. ..	1 0 4	Or 20 per cent. of those with ac.rh.
83, or 67·4 per cent.	 6	129	Or { 23·6 per cent. of the males. 12·9 per cent. of the whole.
13, or 34 per cent.	.. ..	38	Or { 24·5 per cent. of the males. 11·4 per cent. of the whole.
2, or 15·3 per cent. at that age. 2, or 15·3 per cent. of whole.	.. ..	13	{ Or 35 per cent. of those with acute rheumatism.
3, or 23 per cent. at that age. 3, or 33·3 per cent. of whole.	.. ..	9	{ Or 24·3 per cent. of those with acute rheumatism.
11	.. ..	13	
8, or 66·6 per cent.	.. ..	12	Or { 2·2 per cent. of the males. 1·2 per cent. of the whole.
4, or 30·6 per cent.	.. ..	13	Or { 8·4 per cent. of the males. 4 per cent. of the whole.
1, or 25 per cent. at that age. 1, or 50 per cent. of whole. 2, or 50 per cent. at that age. 2, or 100 per cent. of whole.	.. ..	2 2	{ Or 8·3 per cent. of those with acute rheumatism. { Or 16·6 per cent. of those with rheumatism.
3	.. ..	3	
0	.. ..	38	Or { 6·9 per cent. of the males. 3·8 per cent. of the whole.
0	 1	17	Or { 11 per cent. of the males. 5·3 per cent. of the whole.
0	.. ..	4	Or 24 per cent. of those with ac.rh.
0	.. ..	9	Or 53 per cent. of those with ac.rh.
361, or 69·4 per cent.	Age not stated 27	547	
{ 52, or 33·7 per cent. + 1 occupation not stated.	{ Age (?) 1+2 occ. not stated	{ 155 + 3 occ. not stated	
12, or 25 per cent. at that age. 12, or 36·3 per cent. of whole.	{ Age (?) 1+1 occ. not stated	{ 33 + 2 occ. not stated	
13, or 25 per cent. at that age. 13, or 28·2 per cent. of whole.	{ Age (?) and occ. not stated 1	{ 46 + 1 occ. not stated	
47, + 1 occupation not stated.	{ Age (?) — 3 occ. not stated	52	

rheumatism whose ages were stated, and who were so affected, who were engaged in the class of occupations indicated in the column headed "Male Patients."

[1] Here and elsewhere in this column "whole" applies to the whole number of patients of both sexes.

TABLE II.—*Continued.*

Female Patients.		Below the Age of 21 Years.	From 21 to 25 Years.
Servants . . .	Other diseases except acute rheumatism	64, or 32·8 per cent. of those whose ages are stated.	60, or 30·8 per cent.
	Acute rheumatism	57, or 57 per cent.	22, or 22 per cent.
	Pericarditis	14, or 24·5 per ct, at that age.[1] 14, or 73·7 per ct. of the whole.[2]	4, or 18·2 per ct. at that age. 4, or 22·2 per ct. of the whole.
	Endocarditis	25, or 44 per cent. at that age. 25, or 58·5 per cent. of whole.	10, or 45·5 per ct. at that age 10, or 23·2 per ct. of the whole.
Other in-door active employments. .	Other diseases except acute rheumatism and acute gout	1, or 1·4 per cent.	9, or 12·5 per cent.
	Acute rheumatism	4, or 18·2 per cent.	4, or 18·2 per cent.
	Pericarditis	0	1, or 25 per ct. at that age. 1, or 25 per ct. of the whole.
	Endocarditis	0	1, or 25 per cent.
	Acute gout	0	0
Sedentary in-door employments . . .	Other diseases except acute rheumatism	1, or 2·7 per cent.	5, or 13·8 per cent.
	Acute rheumatism	2, or 22·3 per cent.	0
	Pericarditis	0	0
	Endocarditis	1, or 50 per cent. at that age. 1, or 25 per cent. of whole.	0
Married women without special employment. . . .	Other diseases except acute rheumatism	3, or 4·3 per cent.	9, or 12·6 per cent.
	Acute rheumatism	0	6, or 37·5 per cent.
	Pericarditis	0	1, or 16·6 per ct. at that age. 1, or 50 per ct. of the whole.
	Endocarditis	0	
Out-of-door employment. Kept a stall . .	Other diseases except acute rheumatism	0	0
	Acute rheumatism	0	1, or 50 per cent.
	Pericarditis	0	0
	Endocarditis	0	0
Of no occupation, including girls at school . . .	Other diseases except acute rheumatism	57, or 100 per cent.	0
	Acute rheumatism	14, or 93 per cent.	1, or 7 per cent.
	Pericarditis	2, or 14·4 per ct. at that age.	0
	Endocarditis	6, or 43 per cent. at that age.	0
Total of female patients . .	Other diseases except acute rheumatism and acute gout	126, or 29 per cent.	83, or 19 per cent.
	Acute rheumatism	77, or 47·2 per cent.	34
	Pericarditis	17, or 22 per cent. at that age. 17, or 63·3 per ct. of the whole.	6, or 17·6 per ct. at that age. 6, or 22·2 per ct. of the whole.
	Endocarditis	32, or 41·5 per ct. at that age. 32, or 53·3 per ct. of the whole.	11, or 32·3 per ct. at that age. 11, or 18·3 per ct. of the whole.
	Acute gout	0	0
Grand Total of Male and Female Patients . . .	Other diseases except acute rheumatism and acute gout	215, or 22·5 per cent. of the whole with ages stated.	153, or 16 per cent. of whole, with ages stated.
	Acute rheumatism	147, or 46·7 per cent.	66, or 20·8 per cent.
	Pericarditis	34, or 33 per cent. at that age. 34, or 56·6 per ct. of the whole.	10, or 15 per ct. at that age. 10, or 16·6 per ct. of the whole.
	Endocarditis	57, or 38·8 per ct. at that age. 57, or 53 per ct. of the whole.	19, or 28·8 per ct. at that age. 19, or 18 per ct. of the whole.
	Acute gout	0	5

[1] Here and elsewhere in these columns add after "age" of those with acute rheumatism who were so affected and who were engaged in the class of employments indicated in the column headed "Male Patients."

[2] Here and elsewhere in these columns "whole" applies to the whole number of all ages of those with acute

TABLE II.—*Continued.*

Above 25 Years.	Age and Occupation not stated.	Total.	
71, or 36·4 per cent.	Age not stated 9	204	Or { 42 per cent. of the females. 20·4 per cent. of the whole.
21, or 21 per cent.	 1	101	Or { 60·5 per cent. of the females. 31·3 per cent. of the whole.
1, or 4·7 per cent. at that age. } 1, or 5·2 per cent. of whole. {	.. ..	19	{ Or 18·8 per cent. of those with acute rheumatism.
8, or 38 per cent. at that age. } 8, or 18 per cent. of whole. {	.. ..	43	{ Or 42·5 per cent. of those with acute rheumatism.
62, or 86·1 per cent.	 5	77	Or { 15 per cent. of the females. 7·7 per cent. of the whole.
13, or 62 per cent.	 1	22	Or { 13·7 per cent. of the females. 6·8 per cent. of the whole.
3, or 37·7 per cent. at that age. } 3, or 75 per cent. of whole. {	 1	5	{ Or 22·7 per cent. of those with acute rheumatism.
3, or 75 per cent.	.. ..	4	{ Or 18 per cent. of those with acute rheumatism.
3	.. ..	3	
31, or 84·5 per cent.	 1	38	Or { 8·4 per cent. of the females. 8·8 per cent. of the whole.
7, or 77·7 per cent.	.. ..	9	Or { 5·4 per cent. of the females. 2·8 per cent. of the whole.
0	.. ..	0	
3, or 43 per cent. at that age. } 3, or 75 per cent. of whole. {	.. ..	4	{ Or 44·4 per cent. of those with acute rheumatism.
59, or 83 per cent.	 4	75	Or { 16·3 per cent. of the females. 7·5 per cent. of the whole.
10, or 62·5 per cent.	 1	17	Or { 10·2 per cent. of the females. 5·3 per cent. of the whole.
1, or 10 per cent. at that age. } 1, or 50 per cent. of whole. {	.. ..	2	{ Or 11·8 per cent. of those with acute rheumatism.
{ 2, or 20 per cent. at that age. } 2, or 100 per cent. of whole. {	.. ..	2	{ Or 11·8 per cent. of those with acute rheumatism.
2, or 100 per cent.	.. ..	2	Or { 0·4 per cent. of the females. 0·2 per cent. of the whole.
1, or 50 per cent.	.. ..	2	Or { 1·2 per cent. of the females. 0·6 per cent. of the whole.
0	.. ..	0	
1, or 100 per cent. at that age.	.. ..	1	Or 50 per cent. of those with ac. rh.
0	.. ..	57	Or { 12·5 per cent. of the females. 5·7 per cent. of the whole.
0	 1	16	Or { 9·6 per cent. of the females. 5 per cent. of the whole.
0	.. ..	2	Or 12·5 per ct. of those with ac. rh.
0	 1	7	Or 44 per cent. of those with ac. rh
225, or 51·8 per cent.	 19	453	
53 + 1 occupation not stated.	 3	{ 167 + 1 occ. not stated }	
4, or 7·5 per cent. at that age. } 4, or 15 per cent. of whole. {	 1	28	
17, or 32·7 per cent. at that age. } 17, or 28·3 per cent. of whole. {	 1	61	
3	.. ..	3	
{ 586, or 61·4 per cent. of those with ages stated. {	 46	1000	
{ 104 + 2 occupation not stated, or 32·8 per cent.	{ Age (?) 5+2 occ. not stated. }	{ 322 + 4 occ. not stated }	
{ 16 + 1 occupation not stated, or 15·4 per cent. at that age, or 26·6 per cent. of the whole.	{ Age (?) 1+2 occ. not stated. }	{ 61 + 2 occ. not stated }	
{ 30, or 29·4 per cent. at that age. { 30, or 28·3 per cent. of whole.	{ Age (?) 1+1 occ. not stated. }	{ 107 + 1 occ. not stated }	
50	{ Age and occ. not stated 3 }	{ 55 + 3 occ. not stated }	

rheumatism whose ages were stated, and who were so affected, who were engaged in the class of occupations indicated in the column headed "Male Patients."

[1] Here and elsewhere in this column "whole" applies to the whole number of patients of both sexes.

The accompanying Tables show (1) the proportion in which female domestic servants affected with acute rheumatism were attacked by endocarditis and Pericarditis, and the influence of age in the proportionate production of those affections of the heart in that disease; and (2) the relation of the degree of the joint affection to the degree of the heart affection in those cases.

1. Degree of the Joint Affection in Servants affected with Acute Rheumatism, in relation to Age and Heart Affection.

Joint Affection.	No Endocarditis.				Endocarditis threatened or probable.					Endocarditis.				Pericarditis.				Total.				Grand Total.
	Below 21.	21 to 25.	Above 25.	Total.	Below 21.	21 to 25.	Above 25.	? age.	Total.	Below 21.	21 to 25.	Above 25.	Total.	Below 21.	21 to 25.	Above 25.	Total.	Below 21.	21 to 25.	Above 25.	? age.	
Very severe..	0	0	0	0	0	0	0	0	0	2	0	0	2	5	1	0	6	7	1	0	0	8
Severe	1	0	3	4	5	0	4	0	9	10	5	4	19	7	1	1	9	23	6	12	0	41
Rather severe	1	3	1	5	7	3	1	1	12	7	4	1	12	2	1	0	3	17	11	3	1	32
Not severe...	1	1	2	4	3	2	1	0	6	5	0	2	7	0	1	0	1	9	4	5	0	18
Slight.....	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	1	0	0	0	1
Doubtful	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	1	0	1
Total....	3	4	6	13	15	5	6	1	27	25	9	8	42	14	4	1	19	57	22	21	1	101

2. Degree of the Joint Affection in Servants in relation to the degree of the Heart Affection in the cases of Rheumatic Pericarditis.

Degree of the Joint Affection.	Degree of the Heart Affection.						Total Degree of the Joint Affection.
	Fatal.	Very Severe.	Severe.	Rather Severe.	Not Severe.	Slight.	
Very severe	1	5	0	0	0	0	6
Severe.....	0	1	5	2	0	1	9
Rather severe	0	0	1	1	1	0	3
Not severe.....	1	0	0	0	0	0	1
Heart affection ..	2	6	6	3	1	1	19

I will now briefly consider the occupations of the remaining *female patients* who were attacked with acute rheumatism. I have thrown into one group the cooks, charwomen, nurses, and laundresses, who numbered altogether 22. Of these 5 had Pericarditis, 4 of whom had endocarditis also, and 4 had simple endocarditis; in 7 endocarditis was threatened or probable; and in 6 the heart gave no evidence of being affected. Of the whole number less than a fifth were younger than 21 (4 in 21[1]). Of the five cases with Pericarditis, in one the attack was severe but transient, and in that patient the joint affection was severe. In two others the heart affection was rather severe, and in the remaining two it was slight, while in none of these was the joint affection severe.

Nine of the women followed sedentary employments, using chiefly the needle; and in none of these was there Pericarditis; four of them, however, had endocarditis.

The married women numbered 17, and of these only two had Pericarditis and endocarditis, one severely, the other slightly. In both the joint affection was rather severe. Of the remainder, 2 had simple endocarditis, and 5 were threatened with it, while one-half (8) gave no sign of heart affection. These patients were all older than 23.

Sixteen of the female patients had no occupation, only one of whom was above the age of 20. Only two of them had Pericarditis, one of whom had endocarditis also; in one of these the heart affection was fatal, in the other it was severe; and in one of them the joint affection was severe, while in the other that ended fatally it was so only to a moderate degree. Seven of these cases had simple endocarditis and 2 were threatened with it; while 5 of them presented no indication of endocarditis.

These cases, taken as a whole, show that those women who followed at a mature age occupations as laborious as the young servants, were affected in but a moderate proportion with Pericarditis, and that in a comparatively mild form. They also show that those of tender age who followed no occupation were not attacked with inflammation of the heart with anything like the same frequency as young female servants. We thus see, in

[1] In one of the 22 cases belonging to this group the age of the patient is not stated.

brief, that in acute rheumatism affecting the female sex, youth with labor is nearly always attacked or threatened with endocarditis or Pericarditis, or both; that youth without labor is thus attacked with comparative infrequency; and that mature age with labor is attacked less frequently and much less severely with inflammation of the heart than youth with labor.

The *male patients* give us two great illustrations. One of these is supplied by those working *indoors*, and they naturally run in the same grooves as the female patients, who were, all but two, occupied indoors. The other is supplied by those following *out of door* occupations; and they stand completely apart in kind of labor, age, and character of disease, as well as in sex, from the female patients, whose cases have just been considered.

I have brought the male patients working *indoors* including ten servants, into one group, numbering 37. In several features this group presents a remarkable agreement as regards age and the frequency of heart affection, and especially of Pericarditis, with the important and large analogous group of female servants. Thus in each group more than half of the patients were below the age of 21 (of the male patients 19 in 37, of the female servants 57 in 100);[1] in each, the proportion of cases with Pericarditis was great, amounting among the males to one-third (13 in 37), among the female servants to one-fifth (19 in 101); in each three-fourths of those thus affected with Pericarditis were below the age of 21 (10 of the 13 male patients, and 14 of the 19 female servants); in each the proportion of those in whom the heart presented no sign of inflammation was small, amounting to one-sixth of those male patients (6 in 37), and one-eighth of the female servants (12 in 101); and in each few of the patients whose hearts were thus unaffected were below the age of 21, amounting to fully one-third of those male patients (3 in 7), and to one-fourth of the female servants (3 in 13). Here, however, this close parallel ends, since among the patients affected with acute rheumatism above the age of 25, Pericarditis attacked the men working indoors more frequently (2 in 13) than the female servants (1 in 22), and among those with Pericarditis, less than one-half of the males (6 in 13), and almost as many as three-fourths of the females (15 in 19) were attacked with severity; while the proportion of cases affected or threatened with simple endocarditis was much smaller among the male patients (9 and 9 respectively in 37) than the female servants (42 and 26 in 101).

[1] In one of the 99 female servants affected with acute rheumatism, the age of the patient is not stated.

Looking at these two sections of the patients in their larger and more vital relations, it is evident that in both sexes the same causes produce, under like conditions, the same effects; and that a very large proportion of the young persons who work on foot indoors during many hours daily, are attacked with inflammation of the heart when affected with acute rheumatism, while a very small proportion are thus attacked of the men and women of mature age who are engaged in the same manner.

If we looked solely to the kind of employments just considered it would be natural to infer that overwork indoors in young people of both sexes was the main cause of acute rheumatism and of its attendant Pericarditis and endocarditis. While, however, as we have just seen, the whole of the female patients with occupations were engaged indoors, save two poor women who each kept a stall, only about one-fourth of the male patients worked indoors.

The larger proportion of the male patients affected with acute rheumatism, amounting nearly to three-fifths (82 in 154), excluding those working with lead, worked *out of doors*. More than one-half of these (45 in 84) were engaged in hard labor. Pericarditis attacked nearly one-fourth of these patients (10 in 45). We here find, what is at first sight an unexpected result, that of these laborious workers out of doors thus attacked with Pericarditis only one in ten was below the age of 21; whereas of the male indoor workers thus affected, fully three-fourths (10 in 13) were below that age. If we look at those of older age, we find the scale exactly reversed; since of those laboring out of doors four-fifths (8 in 10) were above the age of 25; while of those working indoors only one-sixth (2 in 13) were above that age. We here, I consider, find the explanation, that I promised when considering age, of the twofold fact, that the male cases of Pericarditis usually combined with endocarditis outnumber the female cases by one-fifth (35 to 28); and that the number of the men so affected above the age of 25 is three times as great as that of the women so affected (men with Pericarditis 13 in 53, women 4 in 53). I think we may infer from these facts that excessive labor in the open air in men of mature age is a frequent cause of acute rheumatism having a strong tendency to Pericarditis.

Male patients with acute rheumatism, whose occupation was chiefly on foot, such as watchmen and porters; and those employed with horses and in stables, whose habits make them liable to gout, including coachmen, cabmen, and grooms; did not suffer from Pericarditis so frequently as those who were engaged in hard labor:

since of those working on foot only one seventh (2 in 14) and of those employed with horses only one-twenty-third (1 in 23), while of those whose work was laborious, nearly one-fourth (10 in 45) were thus attacked.

These facts support the view that Pericarditis tends to attack men of mature age affected with acute rheumatism when their work is hard, but not when it is comparatively easy.

It remains to me to speak of two other classes of employments, painters and plumbers on the one hand, and waiters and barmen on the other, who tend to have gout much more frequently than acute rheumatism. I find, however, that 11 waiters and barmen and 5 of those working with lead were attacked with acute rheumatism. One of each of those classes was attacked with Pericarditis, both of whom were above 30 years of age. Seven of the waiters and barmen and two of the workers in lead presented no sign of heart affection. These were all but one below the age of 24, and in none of them was the great toe affected.

It would thus appear that when barmen, painters, or workers among horses, whose employments tend to induce gout, are attacked with acute rheumatism, especially when young, they do not tend to have Pericarditis or endocarditis.

II.—The Affection of the Joints in Rheumatic Pericarditis.

The inflammation of the joints and the inflammation of the heart in acute rheumatism form one disease. We know that in a certain proportion of the cases the heart shows no sign of being touched by the disease, and here and there perhaps in a very rare instance the heart is attacked with inflammation when the joints are free from it. The unity of the two phases of the disease, the external phase, in the joints, and the internal in the fibrous structures of the exterior and the interior of the heart being established, we have to inquire what was the relative intensity of the inflammation of the joints and the inflammation of the heart in my cases of acute rheumatism, and especially in those affected with Pericarditis.

We have just seen that in servants attacked with acute rheumatism, the joint was, as a rule, only of moderate severity when the heart gave no signs of being affected; that the joint affection was more severe when the heart was threatened or probably attacked with endocarditis; and that the severity of the joint affection increased in a direct ratio with the increased certainty and severity of the heart affection; the joint affection being greater when simple endocarditis was actually present than when it was threatened or probable, and much greater when the heart was attacked with both endocarditis and Pericarditis.

I find that the same rule applies to the whole body of the cases of acute rheumatism; as may be seen in the accompanying Table, showing the degree of intensity of the joint affection in relation to the absence or presence of endocarditis and Pericarditis in cases of acute rheumatism.

Degree of intensity of the Joint Affection in relation to the absence or presence of Endocarditis and Pericarditis in cases of Acute Rheumatism.

Joint Affection.	No Endocarditis.	Endocarditis threatened or probable.	Endocarditis	Pericarditis.	Total.	Female servants.	Other employments.
Very severe	0	1	2	12	15	— 8	= 7
Severe	22	32	46	25	125	— 41	= 84
Rather severe	34	35	42	18	129	— 32	= 97
Not severe	18	7	15	6	46	— 18	= 28
Slight	4	1	2	1	8	— 1	= 7
Doubtful	1	0	1	1	3	— 1	= 2
Total	79	76	108	63	326	—101	=225

Thus the joint affection was severe in one-fourth (22 in 78)[1] of those patients in whom the heart gave no sign of inflammation; in two-fifths (32 in 76) of those in whom endocarditis was threatened or probable; in more than two-fifths (48 in 107)[1] of those affected with simple endocarditis, and in three-fifths (37 in 62)[2] of those who were attacked with Pericardi-

[1] The degree of the joint affection was not stated in one of the 79 cases belonging to this group.

[1] The degree of the joint affection was not stated in one of the 108 cases belonging to this group.

[2] The degree of the joint affection was not stated in one of the 63 cases belonging to this group.

tis, all but 9 of whom (54) had endocarditis also.

The inflammation of the joints was very intense in 12 of the 37 patients with Pericarditis, usually coupled with endocarditis, in whom the inflammation of the joints was severe, whereas in only 3 of the 184 patients in whom simple endocarditis was present or threatened, and in none of the 79 in whom the heart gave no evidence of being affected, was the joint affection of this great degree of intensity.

Table giving the actual number of the Joint Affection in relation to the degree of Heart Affection in 62[1] cases of Rheumatic Pericarditis.

Joint Affection.	Heart Affection.					
	Fatal.	Very severe.	Severe.	Rather severe.	Not severe, or slight.	Total.
The joint affection was very severe in 12 cases	....	....	....	....	....	12
Of these the heart affection was fatal in	1					
" " very severe in	....	6				
" " severe in	....	....	3			
" " rather severe in	....	....	....	2[2]		
The joint affection was severe in 25 cases	....	....	....	....	....	25
Of these the heart affection was fatal in	2					
" " very severe in	....	7				
" " severe in	....	....	11[3]			
" " rather severe in	....	....	....	3[4]		
" " not severe in	....	....	....	....	2[4]	
The joint affection was rather severe in 18 cases	....	....	....	....	....	18
Of these the heart affection was fatal in	0					
" " very severe in	....	2				
" " severe in	....	....	5			
" " rather severe in	....	....	....	6[4]		
" " not severe 2, or slight 3 in	....	....	....	....	5 {2 3}	
The joint affection was not severe 6, or slight 1 = 7	....	....	....	....	....	7
Of these the heart affection was fatal in	1[5]					
" " very severe in	....	0				
" " severe in	....	....	2			
" " rather severe in	....	....	....	4		
" " not severe or slight in	....	....	....	....		
The joint affection was not described (?) in 1	....	....	....	....	....	1
In this case there were no signs of Endocarditis.						
Of the total of the above 63 cases of Pericarditis, the heart affection was fatal in	4					
The heart affection was very severe in	....	15				
" " severe in	....	....	21			
" " rather severe	....	....	....	15		
" " not severe or slight in	....	....	....	....	7 {3 4}	
Total number of cases	....	....	....	....	....	64

In the cases of Pericarditis, there was a close correspondence in severity between the inflammation of the joints and the inflammation of the heart. The above Table shows in detail the degree of the joint affection in relation to the degree of the heart affection in sixty-two cases of Rheumatic Pericarditis.[1] The joint affection was very severe in 12 cases, and in three-fifths of those cases (7) the heart affection was very severe, being fatal in one; in one-fourth of them (3) it was severe; and in only one-sixth of them (2) was it of moderate severity. The joint affection was severe in 25 cases, and in one-third of those cases (9) the heart affection was very severe; in less than one-half of them (11) it was severe, and in one-fifth of them (5) it was of moderate severity, or slight. If we combine these two groups of cases, amounting to 37, that were

[1] In 1 of the 63 cases of Rheumatic Pericarditis the condition of the joints is not described.

[2] In these 2 cases the signs of Endocarditis were absent or doubtful.

[3] In 2 of these cases the signs of Endocarditis were absent or doubtful.

[4] In 1 of these cases the signs of Endocarditis were absent or doubtful.

[5] In this case Endocarditis was absent or doubtful.

[1] In one of the 63 cases of Pericarditis the joint affection was not described.

marked by the severity of the joint affection, we find that in four-fifths of them (30) the affection of the heart was severe, while in one-fifth of them (7) it was not severe or only moderately so. Endocarditis was present in all but two of the 30 cases in which the affection both of the joints and the heart was severe; while the signs of endocarditis were either absent or doubtful in 4 of the 7 cases in which the affection of the joints was severe, while that of the heart was either of moderate severity or slight.

If we examine those cases, amounting to 26, or two-fifths of the whole number, in which the degree of the joint affection was below the line of severity, we find that in 18 of them the affection of the joints was only of moderate severity, while in 7 of them it was slight; and that in two-fifths of these (10) the heart affection was severe, while in three-fifths of them (15) it was either slight or of moderate severity. We find, then, that in the 37 cases of Pericarditis in which the joint affection was more severe, the heart affection was more severe in four-fifths (30) and less severe in one-fifth (7); while in the 25 cases of Pericarditis in which the joint affection was less severe, the heart affection was more severe in two-fifths (10) and less severe in three-fifths (15).

III.—The Degree of the Joint Affection during the Acme of the Effusion into the Pericardium.

When the exterior of the heart is attacked by inflammation in cases of acute rheumatism, the distress and oppression in the region of the heart and in the chest is often so great as to call the patient's attention away from the seat of suffering in the joints. At the same time the physician or the clinical clerk is so much interested in the state of the central organ that he readily overlooks that of the joints. I find that in 12 of the 45 cases given in the accompanying plans (see pages 478, 479), the condition of the joints was not reported during the acme of the pericardial effusion, and in one other case the joint affection was not noted until the attack of Pericarditis had declared itself.

The state of the joints during the period of the acme of the inflammation of the exterior of the heart, marked by the extent of fluid in the pericardium being then at its height, is shown in 32 of the 45 patients under examination. These cases divide themselves naturally into two groups; in one of these, amounting to 12, the Pericarditis was at its acme at the time of admission, or on the following day; while in the remaining 20 cases the effusion into the pericardium reached its acme after the admission of the patient. In the latter set of cases, the intensity of the joint affection had been, as a rule, modified and lessened by rest and soothing treatment, and, especially in four-fifths of the cases, by opium given at repeated intervals; while in the former set of cases in which the pericarditis was at its height at the time of admission, the joint affection had been, as a rule, somewhat aggravated by the removal of the patient from home to hospital. The set of cases, therefore, that were admitted with pericarditis at its height show the natural relation of the degree of the joint affection to that of the heart affection during the period of the acme of the disease, in a manner less affected by other influences than the set in which the pericarditis came on and reached its height after admission.

The inflammation of the joints was severe at the time of admission in more than one-half of the patients (7 in 12) who came in with the Pericarditis at its height, and in six of these seven cases the joint affection was of about equal severity before admission and at the time of the acme of the effusion into the pericardium; while in one of them the joints were less severely affected before than during the period of the height of the Pericarditis.

In two-fifths of this group of cases (5 in 12) the joint affection was not severe when the Pericarditis was at its height, at the time of admission or on the next day, and in three and perhaps in four of these the inflammation of the joints was more severe before admission than after it and during the period of the acme of the effusion into the pericardium. The remaining case stands alone, since in it, although the affection of the heart proved fatal, that of the joints was but slight, both before and after admission.

The second group consists of twenty cases in which the effusion into the pericardium reached its acme after admission; and it will be seen that the relation of the joint affection to the heart affection was very different in this group from what it was in the former one in which the patients came in when the Pericarditis was at its height.

The inflammation of the joints was more severe at the period of the acme of the pericardial effusion than before that period in one-fifth of these cases (4 in 20), and it was of equal severity during the two periods in one other case.

The affection of the joints became less severe during the period of the acme of Pericarditis than before that period in three-fourths of these cases (15 in 20). Four-fifths of these patients (12 in 15)

took repeated doses of opium, with lessening joint affection during the acme of Pericarditis, while only one of the four patients with increasing joint affection during the acme was placed under the influence of opium.

It is evident that if we look only to the first group, or only to the second group of these cases, we should arrive at opposite conclusions with regard to the relation of the degree of the joint affection to that of the heart affection during the acme of Pericarditis. Thus the joint affection lessened during the acme of the disease in one-third of the first group (4 in 13) and in three-fourths of the second group (15 in 20). The influence of repeated doses of opium evidently told on the second group of cases, and the movement of the patients from their homes to the hospital, on the first group of cases, to modify the relation of the joint affection to the heart affection.

I think that we may safely draw an inference midway between these two extreme illustrations, and consider that in about one-half of the cases of Pericarditis the joint affection was of equal severity during the period of the acme of the disease, and before that period; and that in about one-half of them the joint affection became less severe when the Pericarditis was at its height. The general conclusion may be drawn from this inference, that the joint affection tends to lessen in severity when Pericarditis is at its height in about one-half of the cases.

IV.—Time in the Hospital.

The accompanying Table shows the average time that the patients remained in the hospital in relation to the absence or presence of endocarditis or pericarditis in acute rheumatism :—

Time in the Hospital in relation to the absence or presence of Endocarditis and Pericarditis in cases of Acute Rheumatism.

In the Hospital.	No Endocarditis	Endocarditis threatened or probable.	Endocarditis	Pericarditis.	Total.
From 6 to 20 days	33	22	14	7	76
" 21 " 30 "	23	21	31	8	83
" 31 " 50 "	15	21	37	16	89
Over 50 days	3	8	21	28	60
An uncertain number of days . .	2	2	3	4	11
Total	76	74*	106*	63	319*

* Since this table was drawn up, seven cases have been added, making the total number 326.

The time that the patient remained in the wards measures the duration and severity of the disease. Two-fifths of the patients in whom the heart gave no sign of being affected, left the hospital before the end of the third week (33 in 76), three-fourths of them during the first month (56 in 76), and one-fourth of them after the first month (20 in 76). Those who had Pericarditis usually accompanied by endocarditis remained in the wards for a much longer period, since only one-ninth of them (7 in 63) left the hospital before the end of the third week, and one-fourth of them (15 in 63) during the first month, while three-fourths of them remained in the hospital longer than a month (48 in 63), and one-half of them more than fifty days. Those with simple endocarditis remained in the house much longer than those whose hearts were healthy, but not nearly so long as those with Pericarditis usually combined with endocarditis.

V.—Occurrence or Non-Occurrence of one or more Previous Attacks of Acute Rheumatism.

The following Table shows the proportion in which the patients affected with acute rheumatism had been previously attacked by that disease in fully three-fourths of the patients (243 in 319 cases). Less than one-third of those who gave no sign of endocarditis (23 in 76) and nearly one-half of those who were affected with endocarditis (48 in 106), had suffered from one or more previous attacks of acute rheumatism; so that in my cases the occurrence of a previous attack evidently favored the presence of endocarditis. This did not, however, appear to be the case with pericarditis, for only one-third of the cases with that affection had been previously attacked by acute rheumatism. The previous occurrence of acute rheumatism implies in a certain proportion of the cases the pres-

ence of valvular disease of the heart, a condition that promotes the occurrence of endocarditis in acute rheumatism. It is open to inquiry why valvular disease should have more frequently influenced the production of endocarditis than of pericarditis in my cases.

Occurrence or Non-occurrence of Previous Attacks of Acute Rheumatism in relation to the Absence or Presence of Endocarditis and Pericarditis.

Joint Affection.	No Endocarditis.	Endocarditis threatened or probable.	Endocarditis.	Pericarditis.	Total.
No previous attack	37	23	31	26	117
No note of previous attack . .	16	17	27	16	76
One previous attack	17	24	35	15	91
More previous attacks than one .	6	10	13	6	35
Total	76	74*	106*	63	319

* Since this table was drawn up, seven cases have been added, making the total number 326.

VI.—The Time of the First Observation of Friction Sound and of the beginning of Rheumatic Pericarditis in Relation to the beginning or relapse of the affection of the joints.

In a large proportion of the cases of acute rheumatism affected with Pericarditis, friction was heard over the heart either at the time of admission or very soon after it. Thus in more than one-third of the total number of the cases, the rubbing noise was noticed on the day that they entered the hospital (22 in 63); in all but one-half of them (29 in 63) it was heard on that or the following day; and in fully two-thirds of them (41 in 63) it was observed either at the time of admission or during the three days following it. In nine-tenths of the whole number of cases affected with Pericarditis (55 in 63) the frottement was distinguished during the first nine days of the patient's residence in the hospital.

These facts do not, however, point out how soon Pericarditis occurred after the commencement of the attack of acute rheumatism. To ascertain this we must add the number of days from the commencement of the attack to the time of admission, to the number of days from that time to the period at which the to-and-fro sound was heard. This plan answers with those cases in which the friction sound was observed on or after the third day from the date of admission, since in all but four of them the heart had been previously examined. It does not, however, apply to those patients in whom the frottement was detected during the day of admission or on the next day, since in those cases we do not know how long the rubbing sound may have been in existence before the patient came in. This applies to one-half of the patients affected with rheumatic Pericarditis, since they had suffered from acute rheumatism for a period varying from two days to three weeks before entering the wards. These cases are, however, of use in showing how early in the disease, and how late, Pericarditis may declare itself by friction sound in full play. Thus out of the twenty-nine cases in which frottement was heard during the first two days, more than one-fourth (8 in 29) had been affected with acute rheumatism for a period of from two to four days; while on the other hand one-fifth of them (6 in 29) had been ill for from two to three weeks before admission.

If we bring together the whole of the 63 cases of Pericarditis, we find that in one-sixth of them (10 in 63) the rubbing sound was audible as early as from the third to the sixth day after the commencement of the disease; while in one-half of them (30 in 63) that sound was audible on or before the eleventh day of the illness.

In only seven of the cases did the heart affection show itself so late as the twenty-fifth day, and from that to the sixty-third after the onset of the acute rheumatism.

These facts point, I think, to the conclusion that in a certain small proportion of the cases, amounting perhaps to one-eighth (8 in 63) the onset of the inflammation both of the exterior and the interior of the heart took place at the very commencement of the disease, and at the same time with the onset of the inflammation of the joints.

It is scarcely needful to say that the first appearance of the rubbing sound is later than the beginning of the inflammation of the surface of the heart. In this respect, the inflammation of the outside of that organ corresponds with the inflammation of the joints, since, as in inflammation of the joints, pain and tenderness precede exudation and swelling, so in Pericarditis, in at least some instances to which I shall now refer, pain

and exquisite sensitiveness over the heart preceded the notable increase of effusion into the pericardium and the existence of a rubbing sound.

In five of the cases in which friction sound was heard on the day of admission (13, 15, 44*a*, 53, 61), pain had existed over the region of the heart, or in the left side, or in the chest, for one or more days before the patient entered the hospital. In one of these cases (44*a*) pain was present over the heart from about the beginning of the illness, the precise time of which is not stated.

In nearly one-half of the patients in whom the frottement was heard for the first time from one to fifty-three days after admission (16 in 39), there was pain over the region of the heart or in the chest from one to seven days before the rubbing noise was observed. In seven (51, 8, 26, 28, 50, 29, 5) of them the pain was noticed one day ; in three (57, 56, 23), two days ; in one, three days (14) ; in two, four days (55, 36) ; in two, six days (123, 30) ; and in one, seven days (20) before the first observation of the friction sound.

The patient (24), in whom friction sound was heard on the fifty-third day after admission, presented a chain of symptoms interesting in two points of view, one, that the attack of Pericarditis was immediately preceded by a relapse of the joint affection ; the other, that pain over the heart preceded the frottement. The patient was a laborer, aged 27, and had almost passed through a severe attack of acute rheumatism with endocarditis, resulting in permanent injury to the mitral and aortic valves. On the 36th day, he, being stronger and of better color, was allowed to get up. On the 42d his general health was good, his pains were diminished, and he walked about. On the 45th he felt stiffness in the right hip-joint on walking, that joint having been affected for eight months previously ; and on the 48th the pain in the hip was worse, though he was otherwise free from complaint, and his appetite was good. On the 50th day, however, his neck was stiff, and he had flying pains about the knees ; and on the next day his face was flushed. he perspired copiously, and complained of great pain over the region of the heart and palpitation. On the 52d he suffered from a terrible pain in the neck and head, the wrists were swollen and painful, and the heart's action was so loud that the mitral and aortic murmurs were inaudible ; and on the following day a loud and harsh double friction sound was heard over the heart. Here the attack of Pericarditis immediately followed the relapse in the joint affection, and the pain over the heart preceded the rubbing sound by two days.

In four other cases in which the friction sound appeared some time after admission, the Pericarditis followed closely upon a *relapse* of the joint affection. In one of these (36), a woman, aged 20, who was motionless on admission from the affection of the joints, the pain was worse on the 6th day, she was still powerless on the 7th from the pain in the joints, and on the 8th a harsh grating frottement, chiefly systolic, was heard over the apex of the heart. In another patient (3), a man, aged 26, who was re-admitted with a severe relapse of the affection of the joints six days after leaving the hospital, the hands and hips were better on the 5th day after his readmission, but on the 8th there was again pain in the hip, and on the 9th there was excessive pain and tenderness in the fascia of the thigh. On the next day (the 10th) there was pain, and increased dulness on percussion over the heart, and a double friction brush was audible at the apex. In a third case (30), a man, aged 31, all the joints were swollen and painful when he came in, but were so much better on the 8th day that they only pained him when he moved. The pain in the joints returned, however, on the 9th, being better next day, when a harsh double friction sound was audible over the heart.

In the last case of this group (17), a female servant, aged 20, the joints were painful and swollen on admission, they were less so on the 4th day, and on the 7th they were almost of the natural size. On the 9th a little pain returned in the joints and there was oppression over the heart. On the 13th the pain had increased and she suffered much in the chest, the first sound being rough and prolonged. On the 16th there was a murmur all over the heart, which was the seat of pain ; and on the 17th a soft double friction sound was established over the region of the pericardium.

To these cases must be added one of a series that were treated by rest during the years 1866–68. In this patient, a man, aged 20, the pain in the joints, which was considerable on admission and which lessened on the 4th day, again increased in the arms and neck on the 5th, when a pain, beginning at the lower portion of the breast bone, shot through the region of the heart to the back. This symptom and pain in the region of the apex were relieved by leeches. The joints also improved, but on the 10th, after he had been using his hand, pain returned in the finger, and on the 14th, the next report, Pericarditis had fully declared itself.

VII.—The Presence or Absence of Endocarditis in Rheumatic Pericarditis.

I. *Cases where Endocarditis was present.*—There was evidence of inflammation in the interior of the heart in all the cases excepting nine (54 in 63).

The heart was healthy at the time of the attack in 46 of the cases with endocarditis, and the mitral, or mitral and aortic valves were crippled by previous disease in the remaining 8 cases, including one just alluded to (24) in which Pericarditis followed a relapse of the affection of the joints, the aortic and mitral valves having become affected during the earlier part of the attack of acute rheumatism.

A tricuspid murmur was alone present in 3 of the 46 cases of endocarditis: in two of these cases that murmur was persistent, and in one of them it disappeared. These cases were comparatively free from serious symptoms, the heart affection being severe in only one instance, and the inflammation of the joints being very severe in another. The proportion of cases of this class with simple tricuspid murmur, was much smaller in these cases of combined endocarditis and Pericarditis than in those of simple endocarditis; 1 in 18 of the former, as we have just seen (3 in 54), and 1 in 8 of the latter (13 in 108) being thus affected.

The mitral valve was affected in 42 of the 46 patients with Pericarditis in whom endocarditis attacked the heart when previously healthy, in 6 of whom the aortic valve was affected as well as the mitral. The aortic valve was attacked in one other case in which the mitral valve was not involved.

I have divided these 43 cases with mitral (36), aortic (1), and mitral and aortic (6) incompetence into three groups; in the first group, containing 16 cases (11 mitral, 5 mitral and aortic incompetence), valvular disease was finally established, or, in two instances, the disease proved fatal when the murmur was in full play; in the second group, which numbered 8 cases with mitral regurgitation, the murmur was lessening when the patients were discharged; while in the third group, amounting to 19 (17 mitral, 1 aortic, and 1 mitral and aortic incompetence), the murmurs disappeared on the recovery of the patients from acute rheumatism, and the heart was restored to a healthy condition.

The accompanying Table shows the relation of the degree of the affection of the joints and that of the affection of the heart to the occurrence and degree of endocarditis in cases of acute rheumatism affected with Pericarditis.

If we compare the cases of endocarditis thus combined with Pericarditis, with the cases of uncomplicated or simple endocarditis, we find that valvular disease was finally established, that the murmur lessened in intensity, and that the murmur finally disappeared in nearly the same proportion in the two sets of cases. Thus in 70 cases of simple endocarditis, either mitral '53), aortic (10), or mitral and aortic (7) incompetence was present. If we divide these cases, like those with Pericarditis and endocarditis, into three groups, we find that in the first group containing 28 cases (16 mitral, 5 aortic, and 5 mitral and aortic incompetence) valvular disease was finally established, or, in two instances, the disease proved fatal; in the second group, which numbered 11 cases (11 mitral incompetence), the murmur was lessening when the patients were examined for the last time; while in the third group, amounting to 31 cases (24 mitral, 5 aortic, and 2 mitral and aortic incompetence), the murmur had disappeared on the recovery of the patients from acute rheumatism, and the heart became again healthy. A tricuspid murmur was alone audible in 13 additional cases of simple endocarditis: in 7 of these the murmur disappeared, but in 6 of them it was still audible when the heart was listened to for the last time.

I am of opinion, notwithstanding the remarkable correspondence in the effects of the inflammation of the valves in the three parallel groups of each of these two sets of cases, that when inflammation attacks the interior of the heart alone, it is less likely to induce permanent valvular disease, that when the heart is inflamed without and within. This, I think, is *à priori* self-evident, and it is supported by two pieces of clinical evidence that I shall now adduce. (1) Disease of both the mitral and aortic valves, which is the most extensive form of valvular disease, was established in 5 of the 43 cases affected with both endocarditis and Pericarditis, and in 5 only of the 70 cases affected with simple endocarditis. (2) Simple endocarditis was present in 28 out of 74 cases of acute rheumatism that were treated by me in St. Mary's Hospital on a careful and rigid system of *rest*. Valvular disease of old standing existed in 7 of those patients, and a recent mitral murmur, accompanied in one instance by aortic incompetence, affected the remaining 21 cases. The heart regained its healthy condition in 14 of these patients, the murmur was lessening or doubtful in 4 of them on their recovery from acute rheumatism, and valvular disease was established in 3 only of the whole series of 21 cases.

The inflammation both of the joints and the heart was more often severe in those cases in which the valves became permanently diseased, than in those in which the recovery of their function was complete. The heart affection was severe in 12 of the 16 cases in which the valves were permanently disabled, being fatal in two and very severe in six of them; while it was severe in 13 of the 19 in which the valves were restored to health, being very severe in four of them. The relative in-

tensity of the joint affection was even greater than that of the heart affection; since, in the former class of cases, it was severe in 12 of the 16 in which the organ became diseased, and in only 10 of the 19 in which its recovery was perfect.

PERICARDITIS WITH AND WITHOUT ENDOCARDITIS.

Relation of the degree of the Heart Affection and the Joint Affection to the occurrence and degree of Endocarditis.

			Fatal.	Very severe.	Severe.	Rather severe.	Not severe.	Slight.	Doubtful.	Total.
With Endocarditis.										
Tricuspid murmur . . . . . permanently established	2	Heart affection	..	..	1	1	..	..	..	2
		Joint affection	..	..	..	2	..	..	..	2
Tricuspid murmur . . . . . disappearing on recovery	1	Heart affection	..	..	..	..	1	..	..	1
		Joint affection	..	..	1	..	..	..	..	1
Tricuspid murmur . . . . . Total number	3	Heart affection	..	..	1	1	1	..	..	3
		Joint affection	..	.	1	2	..	..	..	3
Mitral regurgitation, ending . . . . in mitral valve disease	11	Heart affection	1	5	1	2	..	2	..	11
		Joint affection	..	4	4	3	..	..	..	11
Aortic and Mitral regurgitation, ending . . in established disease of both valves .	5	Heart affection	1	1	3	..	..	..	..	5
		Joint affection	..	1	3	1	..	..	..	5
Mitral and Mitral-aortic regurgitation . . . ending in established valve disease .	16	Heart affection	2	6	4	2	..	2	..	16
		Joint affection	..	5	7	4	..	..	..	16
Mitral regurgitation . . . . . lessening on recovery	8	Heart affection	..	1	3	2	1	1	..	8
		Joint affection	..	..	3	2	2	1	..	8
Mitral regurgitation . . . . . disappearing on recovery	17	Heart affection	..	4	8	4	1	..	..	17
		Joint affection	..	2	7	6	2	..	..	17
Aortic regurgitation . . . . . disappearing on recovery	1	Heart affection	..	..	1	..	..	..	..	1
		Joint affection	..	1	..	..	..	..	..	1
Mitral-aortic regurgitation . . . . . disappearing on recovery	1	Heart affection	..	..	..	1	..	..	..	1
		Joint affection	..	..	..	1	..	..	..	1
Mitral, Aortic, and Mitral-aortic regurgitation . disappearing on recovery	19	Heart affection	..	4	9	5	1	..	..	19
		Joint affection	..	3	7	7	2	..	..	19
Mitral valve-disease of old standing	5	Heart affection	1	1		1	..	..	..	5
		Joint affection	..	1	2	1	1	..	..	5
Mitral-aortic valve-disease of old standing (1 recent)	3	Heart affection	..	3	..	..	..	..	..	3
		Joint affection	..	1	1	1	..	..	..	3
Mitral and Mitral-aortic valve disease of old standing	8	Heart affection	1	4	2	1	..	..	..	8
		Joint affection	..	2	3	8	1	..	..	8
TOTAL number of cases of Pericarditis accompanied by Endocarditis	54	.....	3	15	19	11	3	3	..	54
		.....	..	10	21	17	5	1	..	54
Endocarditis absent or doubtful										
Cases without signs of Endocarditis . . .		Heart affection	1	..	2	1	1	1	..	6
		Joint affection	..	1	3	..	1	..	..	5
Cases in which the signs of Endocarditis were doubtful		Heart affection	..	..	..	3	..	..	..	3
		Joint affection	..	1	1	1	..	..	..	3
TOTAL number of cases in which Endocarditis was absent or doubtful		Heart affection	1	..	2	4	1	1	..	9
		Joint affection	.	2	4	1	1	..	1?	9
GRAND TOTAL of cases of Pericarditis . .		Heart affection	4	15	21	15	4	4	..	63
		Joint affection	..	12	25	18	6	1	1?	63

There was mitral regurgitation in the whole of the group of cases, amounting to 8, in which there was previous valvular disease, in three of which the aortic valves were also incompetent. The heart affection was severe in the whole of these cases save one, and the joint affection was so in five of them. The all but universal presence of inflammation within the heart in patients of this class, supports the inference that in acute rheumatism, old standing valvular disease, by throwing additional labor on the organ, tends to produce endocarditis and pericarditis, and to increase the severity of the inflammation of the heart, both within and without.

II. *Cases in which Endocarditis was absent or doubtful.*—The signs of endocarditis were absent or uncertain in only 9 of the 63 cases of Pericarditis. In five of these patients no murmur was audible; in one there is no note that a murmur could be heard, and in the remaining three the existence of a murmur was doubtful. One of these cases proved fatal, and the affection of the heart was severe in two and of moderate severity or slight in the remain-

ing six patients. The joint affection was severe in six of these cases.

Classification of the cases of Pericarditis.—I have classified the cases according to the presence or absence of endocarditis, and subdivided those with endocarditis into the groups which have just been described and which are specified in the following scheme :—

I. Cases of Pericarditis in which Endocarditis was present			54
A.—Cases with Endocarditis attacking the healthy heart		46	
1.—Cases with tricuspid regurgitation		3	
a.—Cases in which the regurgitation became permanent after recovery from acute rheumatism	2		
c.—Cases in which the regurgitation disappeared on recovery	1		
2.—Cases with mitral (36), aortic (1), and mitral-aortic (6) regurgitation	43		
a.—Cases in which the regurgitation became permanent after recovery from acute rheumatism (mitral 11, mitral-aortic 5)	16		
b.—Cases in which the regurgitation lessened after recovery (mitral 8)	8		
c.—Cases in which the regurgitation disappeared after recovery (mitral 17, aortic 1, mitral-aortic 1)	19		
B.—Cases with Endocarditis attacking a heart already affected with mitral (5), or mitral-aortic (3) valve-disease		8	
II. Cases of Pericarditis in which Endocarditis was absent (6) or doubtful (3)			9
Total number of cases of Pericarditis			63

VIII.—Sketch of the Progressive Changes that take place in the Heart and Pericardium during the progress of Pericarditis.

We cannot rightly understand the symptoms and signs of Pericarditis unless we keep in the mind's eye the changes that are going on in the heart and pericardium, and the surrounding organs during the periods of the beginning, increase, and *acme*, the decline and ending of the disease. I shall, therefore, before discussing the symptoms and signs of the disease that were present in my cases, give here a slight sketch of the more important morbid changes, in so far as they make themselves appreciated during life, and shall afterwards describe some of those changes more fully when the consideration of the symptoms and signs of the affection seems to call for it.

When the surface of the heart becomes inflamed, a blush of fine vessels, consisting of a velvety network, appears on the surface of the organ, and especially over the larger coronary vessels at the base and septum of the ventricles. The inner surface of the pericardial sac, wherever it rests upon the inflamed heart, kindles also into a blush of fine vessels. The inflammation caught from the heart on the inner lining of the sac, spreads rapidly to the fibrous structure of the pericardium, and through it may even often extend to the surface of the pleura covering the sac. The inflammation of those parts tells upon the nerves distributed to them. The surfaces of the heart and sac, instead of being smooth and glistening, become dull and velvety ; and fluid is poured out and lymph exudes from the inflamed surfaces.

The liquid in the pericardium increases rapidly. At first it falls into the back part of the sac, but as it increases in quantity it makes a space for itself between the floor of the pericardium, which it depresses, and the lower surface of the heart, which it elevates, and it gradually distends the pouch in every direction, displacing the lungs to each side in front, pushing the central tendon of the diaphragm, the stomach, and the liver downwards, and pressing backwards, when the distension from the fluid becomes great, upon the bifurcation of the trachea, the left bronchus, the œsophagus, and the aorta. The fluid at the same time reacts upon the heart so as to compress the auricles, the venæ cavæ, the pulmonary veins, and the ascending aorta ; and to displace the apex and body of the organ and its great arteries upwards and forwards, owing to the extensive interposition of the fluid between the lower surface of the heart and the floor of the pericardium.

The lymph is poured out upon the surfaces of the heart and the sac. Where those two surfaces touch each other, the soft lymph is drawn into threads and little pointed ridges and prominences, and wrought into a network, so that when

ridges or prominences are present on the heart, ridges or prominences are present on the inner surface of the pouch lying upon it, and when a network of lymph covers the heart, a network of lymph lines the corresponding sac. The constant play of expansion and contraction of the heart alternately stretches and relaxes its coating of lymph, so that its surface resembles a honeycomb in structure.

The heart, elevated by the fluid between the under surface of the ventricles and the base of the pericardium to a degree proportioned to the amount of the fluid, leaves the broader part of the chest below, and ascends into the narrower part of the chest above. The lungs, and especially the left lung, are consequently displaced from before the swollen sac and the heart, and the front of the right and left ventricles, including the apex and the great arteries, beat with some force against the higher costal cartilages and intercostal spaces, and the adjoining portion of the sternum, with which they come into close contact. Owing to the narrowing compass of the portion of the chest in which the heart is then situated, and the withdrawal of the lung from before the organ, its impulse is both elevated and widened outwards, so that it is felt beating strongly in the second and third, or third and fourth left spaces, according to the amount of the effusion, the apex-beat being felt above, and beyond the nipple; instead of the impulse, as in health, being felt gently in the fourth and fifth spaces, the apex-beat within the nipple-line. When the pericardium is distended to the utmost, its sac becomes pyramidal or pear-shaped, the apex or narrowest part of the pyramid pointing upwards, behind the lower portion of the manubrium and to the left of it, the base of the pyramid bearing downwards and extending across the ensiform cartilage from the sixth right costal cartilage to the lower border of the sixth left cartilage at its attachment to the rib. The fluid rapidly fills the sac, and often reaches its acme in two, three, or four days; but it soon begins to lessen, and in from four to six additional days it usually returns to its healthy amount. At the same time the heart descends and comes again in contact with the lower end of the sternum and the top of the ensiform cartilage, the fifth space, the sixth costal cartilage, and the diaphragm. In most instances slight threads of adhesion form between the sac and portions of the right auricle, and often also between the sac and the apex and interventricular septum, that being the portion of the front of the heart that presents the least movement during the action of the ventricles. These soft threads of adhesion are generally drawn out, by the oscillating movements of the heart, until they at length yield, and break away, but sometimes permanent adhesions form, which may be partial or universal.

IX.—Over-Action of the Heart in Acute Rheumatism as a Cause of Endocarditis and Pericarditis; and (in illustration), Over-Action of the Limbs, Local Injury, and other Influences, as Causes of Acute Rheumatism with Affection of the Heart.

In a small number of my cases of rheumatic Pericarditis, the inflammation of the heart commenced soon after laborious, or violent action of the organ.

A woman (12), aged 26, a servant, was attacked, seven days before admission, with great pain in the soles of her feet. On the following day the pain continued, and proceeded up the legs to the knees and hips, so as to confine her to bed. On the third day she was seized with violent palpitation of the heart, and pain below the lower part of the sternum. On admission her countenance was flushed and anxious, the pulse was 160, and there was pain on pressure over the region of the heart, which was beating with great force. A friction sound was perceptible at the apex with each beat, but indistinctly, owing to the violent action of the organ. The breathing was hurried. Eight leeches were applied over the region of pain, and next day her aspect was better, the action of the heart was natural, the area of dulness on percussion over the region of the pericardium was greatly enlarged, reaching as high as the second cartilage, and friction sound was audible over the whole front of the heart, where the pain was only slight. After this the heart's action became feeble, irregular, and intermittent, but it regained its regularity in eighteen days. The friction sound lasted for about three weeks, and a mitral murmur became permanently established.

Another patient (24), already referred to, a laborer, aged 27, came in with acute rheumatism and endocarditis, presenting first mitral and then aortic regurgitation, both of which became established. He was allowed to get up on the 36th day. On the 48th he looked well, but pain in the hip, a trouble of old standing, had increased in severity. On the 50th the right side of his face was swollen and flushed, and he complained much of stiffness in the muscles of the neck, and next day of great præcordial pain and palpitation, the heart acting strongly and rapidly. On the 52d he was seized with terrible pain in the neck and head, and the heart's action was so loud that the endocardial murmurs were rendered inaudible, and on

the 53d he suffered from acute pain about the præcordia, the left cartilages were arched, præcordial dulness extended up to the third space, and a loud and harsh double friction-sound was heard over the front of the heart. His attack was of unusual severity, but the rubbing sound had disappeared on the 68th day after his admission, and on the 83d he was walking about.

A third case (17), a servant girl, aged 20, who was affected with permanent mitral disease owing to a previous attack, was admitted on the fifth day of her illness with severe joint affection, the heart being rapid and its sounds loud. Next day its action was very tumultuous, its impulse was strong, and its sounds were ill-defined, loud, and harsh. Leeches were applied to the chest, and the bleeding from one of the bites could not be restrained. On the 3d the sounds of the heart were softer; on the 13th the first sound was more rough, on the 16th the impulse was very much diffused, and a murmur was audible over the front of the heart, and next day friction sound was heard over that region and Pericarditis in a severe form was fully established. After this the heart's action became irregular and intermittent, and she looked and felt anxious and depressed. A long, severe and varying illness followed. On the 55th day she seemed to be sinking, though she thought herself better. On the 58th day she kept nothing on her stomach, but on the 59th she felt better and looked much brighter. Smallpox, however, then in the wards, declared itself on the 62d day, and on the 63d she died.

In the first and second of these cases the heart continued to act with increased force during the period of the onset of the Pericarditis; but in the first of them this condition gave way after the application of leeches to irregular action of the heart, which lasted for eighteen days. In seven or eight other cases the impulse of the heart was strong during the early period of the inflammation of the exterior of the heart. As a rule, however, the impulse of the heart was feeble when first observed during the attack of Pericarditis. The condition of the impulse of the heart during Pericarditis will, however, be considered under its proper heading.

If we look at these cases, and especially the first and second of them; combine with them the six others already given in which Pericarditis followed closely upon a relapse in the joint affection, brought on often by getting up too soon; and add to these the relation that existed in my cases of acute rheumatism, between the severity of the joint affection and the presence, character, and severity of the heart affection, the joint affection being slight in the majority of cases without signs of endocarditis, severe in the majority of cases with simple endocarditis, and still more severe in the great majority of cases with Pericarditis and endocarditis; the severity of the heart affection corresponding, as a rule, with the severity of the joint affection; we must, I consider, conclude that we may have here not a mere lifeless chain of passive links, but a living succession of active events, one giving birth to the other. Exposure to cold and wet, combined with undue labor or exertion, give the first impulse,—the start, to the affection of the joints. When the joint affection is severe, it may call forth excessive labor or even tumultuous action of the heart. In acute rheumatism, inflammation attacks the fibrous structures, especially if those structures are unduly strained, and the increased action of the heart may therefore, I consider, induce inflammation of the fibrous tissues of that organ, such inflammation being proportioned in severity to the augmented action of the heart.

This interesting subject derives larger illustration from the influence, already considered, of *sex*, *age*, *and occupation* in the production of acute rheumatism, accompanied, in proportion to the severity of the affection of the joints, by inflammation of the heart within and without. I need only here again refer to the large number of young female servants, in whom the ends and shafts of the bone are as yet only united by cartilage, who are attacked by acute rheumatism in a severe form; and the very large proportion in which those cases have endocarditis or Pericarditis, or both, the heart being subject, in those overworked young women, to undue action and palpitation.

In illustration of the influence of over-action of the heart in producing inflammation of the interior and the exterior of that organ, I shall give here a brief summary of the influence of local injury, scarlet fever, chorea, abscess, and general illness in the production of acute rheumatism with endocarditis and Pericarditis, including those cases of acute rheumatism in which a relapse of the joint affection, followed by Pericarditis, was induced by the too early use of the limbs, when the recovery was almost but not quite perfect.

Two influences usually combine to produce acute rheumatism; one, exposure to wet and cold; the other, the *over-use* of certain limbs and joints. The part immediately in use is usually the part first attacked, while the joints that take the greatest share in the permanent labor of the patient are generally those visited by the disease with the greatest severity and duration. Thus, among the coachmen admitted under my care, one was first

attacked in the right thumb, the knees being afterwards affected; another was seized badly in the right arm, and then in the left; a third in the wrist and hands; and a fourth in the hands, and especially the middle finger, the arms, and then the knees, the affection of the fingers being obstinate; in a fifth the back and hips were the seat of pain; and in the sixth the ankles, knees, hands, and hips were all involved. If we take the carpenters, we find that one of them was attacked in the arms, wrists, and elbows; another, who was in search of work, in the arms, back, ankles, and knees; and a third was seized, when walking, with pain in the knees, the ankles, shoulders and arms being afterwards affected. Young female servants, for to them I must here again refer, who usually work too hard, whose joints are not yet perfect, being still in a state of active growth, are for the most part first attacked in the feet and ankles, that is to say, the parts that more immediately tread the ground. The knees usually then suffer, or perhaps earlier, at the same time as the feet and ankles; and afterwards the wrists, hands, arms and shoulders, in succession, share in the affection. The knees, which generally bear not only the internal pressure of standing, but also the external pressure of kneeling when at work, are as a rule, more constantly and deeply affected, and for a longer period, than any other joint. The effect of past labor is, so to speak, stored up in the knees, which are therefore in these cases more affected in acute rheumatism than any other joint.

Under the combined influence, then, of exposure and overwork, rheumatic inflammation is set up in the joints, and under the combined influence of the disease thus established, and overwork of the heart, rheumatic inflammation is established in that organ.

In a small but important group of my cases, acute rheumatism followed *local injury*. The first of these, a stonemason, fell from a scaffold on his back. He had pain in his back and legs, and could not stand. On the 5th day he had a profuse sour perspiration, and his finger and elbow-joints were red, swollen, and painful. The hips, knees, and shoulders were afterwards attacked, and he probably had endocarditis, the first sound being prolonged, while the second was followed by a soft murmur. The second patient was admitted under Mr. Lane's care for a slight injury, and was attacked on the fourth day with pain in the chest and inflammation of the wrist and ankles. On the 7th he was transferred to my charge with acute rheumatism, mitral murmur, and Pericarditis. A third patient, a dustman, hurt his back by carrying a sack of flour. The pain in the back was increased by his getting wet; and this was followed by acute rheumatism. A fourth patient was attacked with the disease in the wrists 32 days after breaking his leg. A fifth came in with acute rheumatism five days after leaving the surgical ward; and a sixth, who was admitted with endocarditis and transient Pericarditis, had received a kick in the groin five weeks previously, and since then had been subject to pain in the loins. In some of these cases the disease appeared to be directly, and in others to be indirectly, caused by local injury.

These cases and others given below are allied to those previously given, in which the *too early use of a limb*, during the period of convalescence from acute rheumatism, produced inflammation in the used joint, a *relapse* of the affection in the other joints, endocarditis and Pericarditis, ending in permanent crippling of the valves of the heart. The whole of these results, the latter of them so permanently injurious, started from the renewed focus of the disease in the single joint thus affected for the second time.

Through what means is this diffusion and transmission of the disease effected? Is it by a blood poison? Is it by a change in the fibrous structures of the limbs and the heart? Or is it by reflex influences, transmitted through the afferent nerves, locally acted upon in the inflamed joint or injured part, and sent back through the vaso-motor or other nerves distributed to the fibrous structures of the joints and the heart? The local character of the injury inducing this general effect, and the quickness with which the effect is induced, would appear to forbid the material agency of either blood poison or change in the tissues; and would tend to throw us upon the transmission of influences through the nerves for an explanation of these remarkable effects,—effects not less remarkable, but rather more so, that they are open to daily observation; or must we look for some other explanation than any of these here suggested?

We cannot, however, limit ourselves to the points of view just sketched in our inquiry into that many-sided disease, acute rheumatism, with its attendant inflammation of the heart; and I would here briefly state the other influences that have been apparently at work in the origin of the disease, besides overwork and exposure on the one hand, and local injury on the other.

In three of my patients the disease was associated with *scarlet fever*, one of whom had Pericarditis in the hospital, one out of it. The latter was the son of a medical friend, who detected symptoms of acute rheumatism just as the scarlet fever was declaring itself, and by which the acute rheumatism was suspended. When,

however, the eruption had ceased and desquamation was going on, endocarditis and Pericarditis, the offspring of the original rheumatism, declared themselves. This case did well, and though a mitral murmur existed for some time, it at length disappeared. In the two other cases, acute rheumatism followed a chill caught by too early exposure after the scarlet fever had disappeared.

In several of my cases, *chorea* has given place to acute rheumatism or the reverse. In one patient, a girl, acute rheumatism passed into chorea, for which she was admitted. After a time the choreal movements were for a period suspended by the renewal of acute rheumatism. I do not here speak of that terrible complication, the occurrence of serious local choreal and tetaniform symptoms in connection with rheumatic endocarditis and Pericarditis, complications to which I shall soon refer.

In three patients the acute rheumatism was preceded by recent *abscess*, in one of them in the axilla, in another in the perineum, and in a third in the tonsil; and in a fourth case, abscess in the neck existed some time before the supervention of the rheumatism.

Sore throat appeared for from one day to three weeks before the occurrence of acute rheumatism in thirteen cases, including the case of abscess in the tonsil just quoted. Two of these patients had Pericarditis; three had simple endocarditis; in three endocarditis was threatened; and five gave no sign of heart affection.

In eleven patients, *pain in the chest*, sometimes accompanied by cough, existed for from one day to two or even three weeks before the development of acute rheumatism.

I refrain from pursuing this important collateral subject farther in this place.

X.—Pain.

I.—Pain over the Region of the Heart and Pericardium.

Pain over the region of the heart and pericardium showed itself in six different ways: 1. Over the front of the organ; 2. On pressure at the same place; 3. In the epigastrium, chiefly on pressure; 4. Over the back of the heart, when it was excited by swallowing and by eructation; 5. After eating; and, 6. Pain shooting through the heart, evidently anginal in character.

1. The pain over the front of the heart extended usually from the right of the sternum at its lower two-thirds to the left nipple; it was more or less continuous, and was complained of in three-fourths of the cases (48 in 63). This pain came on in one-fourth of the patients affected with it (9) before the friction sound was heard, and in a greater number (16, including 5 in which the pain and the friction sound were both present on the day of admission) at the time that the sound was first audible. In a few instances (7) it was felt soon after the appearance of the rubbing sound. It was either relieved, suspended, or removed by the application of leeches. It was complained of in about one-fourth of the cases (8) at the time the effusion was at its height, but usually relief, which was permanent, came at that time. In two instances (15, 51) of relapse, the second, and in one (44*a*) even a third, wave of increase of pericardial effusion was preceded by a second, and in one even a third attack of pain over the heart; but in three cases the pain came late in the period of the relapse, and when the effusion was declining. In scarcely any instance did the pain over the heart continue during the whole period of the duration of the friction sound, and in only two or three of the cases did it last over the first half of that period. When the pain comes on with the first blush of the inflammation on the surface of the heart, before it has spread to the inner surface of the pericardial sac, and before friction sound is audible, it may be inferred that it is seated in the sentient nerves distributed to the surface of the heart. When, however, the pain strikes over the heart at the same time as the appearance of the friction sound, and still more when it comes on at a later period, it is generally, I believe, seated in the pericardial sac, and especially in the pleura covering the sac.

The accompanying table gives a *résumé* of the period of the occurrence of pain over the region of the heart in relation to the time of the appearance of friction sound in the cases of Pericarditis (see page 501).

2. If the pain over the heart is increased or excited by pressure over the region of the organ, it may, with an approach to certainty, be attributed to inflammation of the pleura, especially if the pain on pressure is complained of, not before, but at the time of or after the first presence of friction sound.

Pain on pressure over the heart occurred in one fourth (14 in 63) of the whole number of cases affected with acute rheumatism, and in one-third of those who suffered from continuous pain in the region of the heart (11 in 38). In two only of these cases was the pain excited by pressure before the friction sound was audible, and in these the pain was probably excited over the surface of the inflamed heart. In one-half of the patients the pain on pressure and the rubbing sound appeared on the same day, and in the rest

the pain was preceded by the friction sound. In most or all of these cases, the pleura covering the pericardiac sac, or the fibrous structure of the sac itself, was the probable seat of the suffering.

In one-half of those patients (7 in 14) the skin over the region of the pericardium was tender and sensitive, so much so indeed, in some instances, as to forbid the slightest manipulation over the chest, and to make a proper examination of the heart impossible until this exquisite sensibility was subdued by the application of leeches or of belladonna liniment with chloroform.

In the majority of the cases the pain was deeper than the skin, and was not excited unless actual pressure was made. In three of the patients the pain was only felt when pressure was made over the region of the heart; but in all the others continuous pain already existed over that region, and was intensified by the pressure. In one or two instances the suffering and distress of the heart were so great as to drown all other complaints; but in three others, as I have just said, the pain was only brought into play when pressure was exerted. Between these two opposite extremes, there was every shade in the extent, variety, and constancy of the pain.

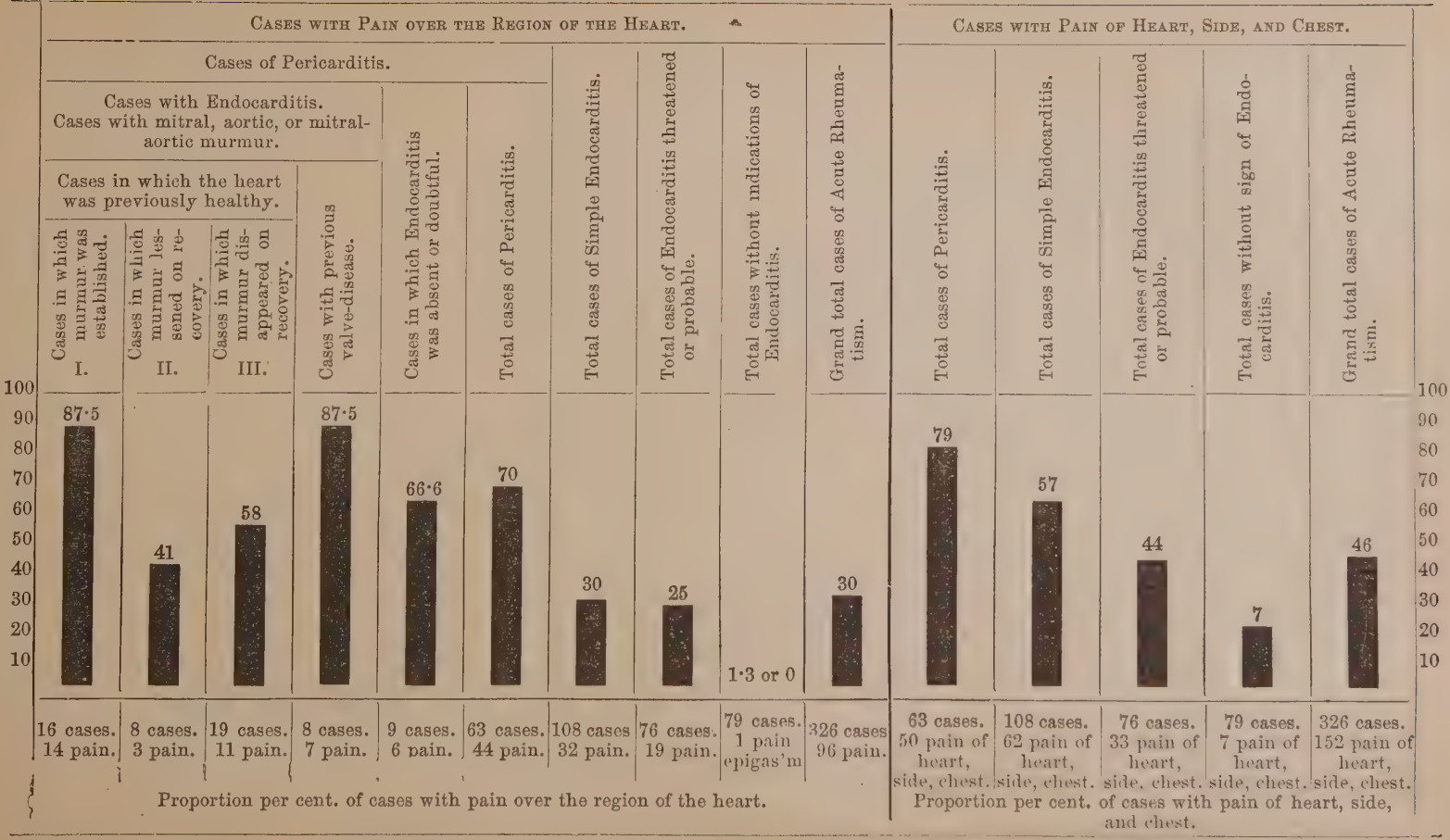
Period of the occurrence of Pain over the region of the Heart and Pericardium in relation to the time of the appearance of Friction Sound in cases of Rheumatic Pericarditis.

Pain over the region of heart and pericardium, including pain over the epigastrium.	Cases.
Pain over heart and friction sound on day of admission	5
Pain over epigastrium and friction sound on day of (in one day after) admission, included above	4
Pain over heart before admission, friction sound on admission	4
Pain over heart before appearance of friction sound	9
Pain over epigastrium before appearance of friction sound, included above	2
Pain over heart and friction sound occurring on same day	11
Pain over epigastrium, and friction sound occurring on same day, *not* included above	3
Pain over heart coming on after friction sound had been observed	7
Pain over epigastrium coming on after friction sound had been observed, *not* included above	2
Ditto, included above	8
Pain over heart appearing shortly before relapse (renewed increase of fluid in the pericardium) *not* included above	1
Ditto, included above	1
Pain over epigastrium before relapse	1
Pain over heart late in period of relapse, *not* included above	1
Ditto, included above	2
Pain over heart at the time of acme of Pericarditis	8
Pain over heart shortly before time of acme of Pericarditis	2
Pain of epigastrium at time of acme of Pericarditis	6
Ditto before acme of Pericarditis	4
Ditto after acme of Pericarditis	4

Pain on pressure over the region of the heart.	*	*	* or †	No endocarditis.	Total.
Appearing before friction sound	1	..	1	..	2
Appearing same time as friction sound	4	2	..	1	7
Appearing after first indication of friction sound	2	1	1	..	4
Appearing after friction sound had ceased	1	..	..	..	1
	8	3	2	1	14

Explanation of Symbols.

* mitral disease.
† aortic disease.


Cases with Pain over the Region of the Heart.
Cases of Pericarditis.
Cases with Endocarditis. Cases with mitral, aortic, or mitral-aortic murmur.
Cases in which the heart was previously healthy.
Cases in which murmur was established. I.
Cases in which murmur lessened on recovery. II.
Cases in which murmur disappeared on recovery. III.
Cases with previous valve-disease.
Cases in which Endocarditis was absent or doubtful.
Total cases of Pericarditis.
Total cases of Simple Endocarditis.
Total cases of Endocarditis threatened or probable.
Total cases without indications of Endocarditis.
Grand total cases of Acute Rheumatism.
100 90 80 70 60 50 40 30 20 10
87·5
41
58
87·5
66·6
70
30
25
1·3 or 0
30
16 cases. 14 pain.
8 cases. 3 pain.
19 cases. 11 pain.
8 cases. 7 pain.
9 cases. 6 pain.
63 cases. 44 pain.
108 cases 32 pain.
76 cases. 19 pain.
79 cases. 1 pain epigas'm
326 cases 96 pain.
Proportion per cent. of cases with pain over the region of the heart.
Cases with Pain of Heart, Side, and Chest.
Total cases of Pericarditis.
Total cases of Simple Endocarditis.
Total cases of Endocarditis threatened or probable.
Total cases without sign of Endocarditis.
Grand total cases of Acute Rheumatism.
100 90 80 70 60 50 40 30 20 10
79
57
44
7
46
63 cases. 50 pain of heart, side, chest.
108 cases. 62 pain of heart, side, chest.
76 cases. 33 pain of heart, side, chest.
79 cases. 7 pain of heart, side, chest.
326 cases. 152 pain of heart, side, chest.
Proportion per cent. of cases with pain of heart, side, and chest.

3. Pain was present over the epigastric region, frequently increased and sometimes induced by pressure, in one-fourth of the patients with rheumatic pericarditis (16 in 63), and in nearly two-fifths of those who suffered from pain over the region of the heart (14 in 38). It would appear curious, at first sight, that pain over the pit of the stomach should be a marked feature in so many cases of Pericarditis. When, however, we consider that in health the lower boundary of the heart is situated behind the upper third of the ensiform cartilage, and that the pericardial sac, when distended with fluid in Pericarditis, dips downwards so that its lower boundary may be on a level with the point of that cartilage, or perhaps even below it, we see how natural it is that pain should be excited by pressure over the epigastric region.

This epigastric pain appeared in only two cases before the supervention of friction sound. Those two patients, however, suffered from a renewal of the pain after the commencement of the rubbing sound, consequently in every case the suffering over the pit of the stomach was complained of either at the time of the first observation of the friction sound (7 in 16, including 4 in which the pain and the friction sound were both present on the day of admission), or from one to several days later (9 in 16).

In one-third of the cases (6) the epigastric pain appeared at the time when the effusion into the pericardium was at its height, and when the sac bulged downwards into the epigastric space; and in four of them it was complained of before, and in four of them after, the effusion had reached its acme.

In all these cases the disease had reached a stage in which the heart was separated by the intervention of fluid from the floor of the pericardial sac, which is formed by the central tendon of the diaphragm. The pain in the epigastric region in these cases, especially when it is increased or excited by pressure, is therefore seated not in the surface of the heart, but in the lower portion of the pericardial sac. It is natural to suppose that the branches of the phrenic nerve must be the immediate seat of the pain, but the exact anatomical distribution of the phrenic nerve has not yet been ascertained. These questions suggest themselves: is this pain seated in the fibrous tissue, the pericardial surface, or the peritoneal surface of the affected diaphragmatic portion of the sac?

Peritonitis affecting the central tendon of the diaphragm has been noticed in few or no fatal cases of Pericarditis, but indirect evidence of its existence has been supplied in rare instances by the discovery of partial adhesions of the spleen and liver to the diaphragm in cases with adherent pericardium. We may, however, I think, fairly infer that the pain on pressure below or at the side of the ensiform cartilage is in these cases due, not to peritonitis, but to inflammation of the fibrous structure and pericardial or inner surface of the central tendon of the diaphragm, where it form the floor of the pericardial sac, and the lower and anterior portion of that sac.

The distribution of the nerves to the pericardium, like that of the phrenic nerve, has not yet been ascertained, and this interesting clinical question therefore invites the attention of the physiologist.

4. In three of the patients affected with rheumatic Pericarditis deep pain was felt between the shoulder-blades, and in one of them this pain was increased by the act of swallowing. Pain in the chest was excited in three cases of swallowing, and in two others it was complained of there after eating. Another patient complained that the ascent of wind from the stomach gave much pain over the posterior region of the heart. In all these instances, amounting to nine, the suffering must have been seated in the back of the inflamed pericardium, being either constant or induced by local pressure, due to swallowing or eructation. In several other cases it is stated that pain was seated in the back, but it is impossible to say, from this description, whether the pain was situated in or near the pericardium or lower down.

5. Pain and fulness after eating was complained of by one patient, and I think it likely that the suffering in this instance was excited by the pressure made by the distended stomach over the lower and back part of the pericardium.

We thus see that in a large proportion of my cases affected with rheumatic Pericarditis, pain was felt over the heart, frequently in front of the pericardial sac, and occasionally behind and below it, the pain being usually fixed, sometimes increased by pressure, and less often excited by it.

6. The heart was attacked with a shooting pain, more or less violent, associated either with faintness or failure in the action of the organ, and evidently anginal in character, in four of my patients affected with rheumatic Pericarditis.

In two of these cases the heart, already crippled by valvular disease, was attacked with inflammation within and without, but in the others the Pericarditis and endocarditis seized upon the virgin heart, the valves being previously healthy; one of these two cases proved fatal, and in the other valvular disease became established.

In the fatal case (4), a man, aged 27, a carpenter, a darting pain passed now and

then from the heart to the right side on the day of his admission. This pain was relieved by leeches, the application of which was followed by faintness. On the 3d his limbs started when he fell asleep; on the 6th he was seized with delirium and trembling; and on the 7th, the day of his death, he was noisy and restless, and was continually moving his lower jaw.

Another patient (15), a servant girl, suddenly became very faint on the evening of the 10th day, when she was suffering from a relapse of Pericarditis, and was attacked with great pain over the heart. This pain returned on the evening of the 12th, when it was also felt between the shoulders.

One (3) of the two remaining patients had old standing aortic and mitral disease, and suffered from pain over the region of the heart on the 10th day, when friction-sound appeared. On the 16th day, when the Pericarditis was at its height, when I was examining him, he cried out as if from pain, beginning over the stomach, and begged to be raised up, the dyspnœa becoming extreme, the face being flushed, the perspiration pouring off it, the lips somewhat livid, and his countenance being expressive of extreme anxiety. He was immediately raised up, and having a towel placed behind him, was as it were slung in it, when he took a little port wine and fell asleep.

The other patient (17), a young woman, affected with mitral disease, was attacked on the 17th day, when the Pericarditis was at its acme, with great pain over the sternum and the whole front of the chest, the pain passing through to the back. She ultimately died on the 63d day, with smallpox, which attacked her when in a state of extreme exhaustion.

If we add to the cases in which there was continuous pain over the region of the heart (38) those others not so affected in which *a*, there was pain on pressure over the heart (3); *b*, pain over the epigastric region (2); and *c*, pain at the back of the pericardium on eructation (1); we find that in 44 of the 63 cases of Pericarditis, or in 70 per cent., there was pain over the heart or pericardium.

II.—Pleuritic Pain in the Side.

Pain in the side was complained of in one-half of the cases of rheumatic Pericarditis (31 in 63). Pain was present over the region of the heart and pericardium also in all but 4 of these patients. The pain was limited to the left side in 19 cases, and to the right in only 5, while it attacked both sides in 6 instances. There were, besides the pain, other symptoms or physical signs of pleurisy in all but seven of the patients thus affected.

Pleuritic friction sound was heard in nearly one-half of those cases (15 in 31) and in five others there was tenderness on percussion over the seat of pain. In a large proportion of the cases the pain was increased or excited by a deep breath (18 in 31), and in four of these it was catching. The pain was induced by coughing or laughing, stooping or moving in fourteen instances, and in three it was "pleuritic" or cutting.

The first complaint of pain in the side was made after the appearance of the friction sound in 19 of the 31 cases that suffered in this manner; the pain and the friction sound appeared together in seven patients; and the pain occurred before the friction sound in five. In one, of the five, and three of the seven patients just spoken of, the pain affected both sides, having appeared at a late period in one side, and at a period actually or comparatively early in the other.

The pleurisy that induced the pain in the side which came into play either with or after the friction sound, was due to two causes; one the extension of the inflammation through the fibrous structure of the pericardium to the pleura covering it; the other, the occurrence of pulmonary apoplexy with its attendant pleurisy.

The more frequent appearance of the pain, and the greater spread of the pleurisy on the left side of the chest than the right, is, I conceive, due in many instances to the greater extent to which the inflamed pericardium occupies the left side of the chest than the right, and the great displacement backward of the left lung, and especially its lower lobe, by the distension of the pericardial sac. Perhaps the pressure of the distended pericardium on the left bronchus increases the tendency of the left lung to inflammation.

In one of the five patients that were seized with pain in the side before the supervention of the friction sound, the pain came on at the first onset of the disease, and at the same time as the affection of the joints three days before admission. I think it likely that this case was attacked with pleurisy and acute rheumatism affecting the joints at the same time, the pleurisy being, however, rheumatic in its nature, like the joint affection in this instance, and like the Pericarditis in the other cases. We may have, in short, in these cases, rheumatic pleurisy, just as we may have rheumatic Pericarditis.

In another of these cases (20), the patient, a married woman, aged 24, was attacked with pain in the joints the day after being wet through, and a week be-

fore admission. She came in with very severe pain in the left side, which had existed for some days, and which was somewhat reduced by leeching. On the 6th day after admission she suffered much in the left side, and a pleuritic friction sound was audible just below the seat of pain. Friction sound from Pericarditis was heard over the region of the heart for the first time on the same day. In this case the pleurisy preceded the Pericarditis by ten days.

Pain in the side was, in proportion, twice as frequent as in Pericarditis usually accompanied with endocarditis as in simple endocarditis; one-fourth of the latter (26 in 108), and, as we have just seen, one-half of the former (31 in 63) being thus affected. A similar proportion of such cases existed among the patients who were threatened with endocarditis, of whom rather more than one-fourth were affected with pain in the side (17 in 63). None of the thirteen cases classed under the heading of "probable endocarditis" suffered from pain in the side, and only three of those who were attacked with acute rheumatism and had no endocarditis, complained of pain in that region (3 in 71). The pain more frequently attacked the left side than the right in the cases of endocarditis in the proportion of 14 to 6; but among those threatened with endocarditis, the two sides were affected in nearly equal numbers, the right side being rather more often attacked than the left in the proportion of 7 to 6.

III.—"Pain in the Chest."

"Pain in the chest" was present in 30 of the 63 cases of rheumatic Pericarditis. The pain thus described is so indefinite in situation—that it may be seated either at the centre of the chest or at its sides, either over the pericardium or the pleura. Fortunately, to guide us to the actual seat of suffering, the "pain in the chest" was attended in all but two instances with other pain, either over the heart, or in the side, or in both regions. Thus in all but four of the thirty cases, pain was present over the region of the heart or the epigastrium; in all but nine, in the side; and in one-half of them (16 in 30) it was situated both over the heart and in the side.

In fully one-half of the cases (17 in 30) the pain in the chest was itself associated with symptoms of pleurisy, in the way of being increased or caused by deep breathing, or coughing, or it was accompanied, in two instances only, by pleuritic friction sound. There were symptoms of pleurisy in eight of the nine cases in which pain of the chest was not associated with pain of the side, and I think those eight cases may be added to the 31 in which pain in the side was actually present, thus bringing their number up to 39 in 63 cases of rheumatic Pericarditis. On the other hand, there were four cases with pain in the chest in which there was no notice of pain in the heart, and I think that these four cases may probably be added to those in which the presence of cardiac pain is stated; thus bringing the total number so affected up from 44 to 48 in 63.

Eleven patients suffered from pain in the chest, either previously to admission or before friction sound was audible. In the greater number of these I think that the pain was seated over the region of the heart, and was not due to pleurisy. And I find, giving strength to this view, that in all of these but two, pain was described as being present over the heart.

It would be futile to compare the relative frequency of pain in the chest ih Pericarditis, and in the other various groups of cases in acute rheumatism, since to do so would be to compare unlike conditions under the same name. But it will be instructive to compare the proportion of cases attacked with pain over the heart, in the side, and in the chest, combined together, with those in which there was no such pain, in cases of acute rheumatism with Pericarditis and endocarditis, and with and without simple endocarditis. The accompanying table, and graphic scheme, will show this comparison, the one by study, the other at a glance (see pages 501–502).

In those affected with Pericarditis, most of whom had endocarditis also, four-fifths had pain in the heart, chest, or side, and one-fifth had no such pain; in those with endocarditis nearly six-tenths had such pain and over four-tenths had none; in those threatened with endocarditis, less than one-half had pain, and more than one-half had none; and in those who gave no sign of endocarditis only one-tenth suffered from this kind of pain, and nine-tenths had no internal pain, thus nearly reversing the proportion that we find in cases affected with Pericarditis.

XI.—Irregularity and Failure of the Action of the Heart. Faintness.

We have already seen that in two of the patients the action of the heart, which was powerful and tumultuous before the occurrence of Pericarditis, became at a later period feeble, irregular, and intermittent, this state being accompanied by a look of great anxiety and depression. We have also seen that the four patients who were attacked with pain shooting through the heart, experienced faintness or failure in the action of the organ (p. 503).

In the following case death took place from syncope. A female servant, aged 25, came in on the 7th day of her illness, with difficult, hurried breathing, which was relieved when she was raised, great pain in her chest, cough, which had continued from the 2d day of the attack, mucous rattle, slightly rusty phlegm, a sensation of choking, difficulty in swallowing, and great anxiety. The joint affection was slight, and apparently limited to the shoulder. Pericarditis, with friction sound and great effusion, was at its height. She was very ill throughout, perspiration being profuse, the voice husky, the face flushed and anxious, and breathing laborious. Her face was brighter, and she breathed with ease from the 7th day to the 13th, when her appetite was improving; but at two hours after midnight, in the early morning of the 14th, when attempting to turn on her side, she became quite pulseless, her face turned livid, and she frothed at the mouth. After taking some wine she gradually recovered. An hour later the sounds of the heart were muffled, and the rubbing noise, which had been harsh, loud and dry on the previous day, could not be detected. In another hour she had a similar attack, in which she died. There were 18 ounces of fluid in the pericardium, the heart was covered with honeycomb lymph, and there were patches of pulmonary apoplexy in the left upper lobe.

Faintness occurred as a symptom in several of the cases, but in none, besides those alluded to and that just given, did it appear in a threatening form.

Although, as we have already seen, in a few cases the action of the heart was unusually strong during the early period of Pericarditis, yet even then, or rather when the attack was first observed, the impulse of the heart was more frequently feeble than strong, and this was especially the case during the remaining course of the affection.

Feebleness, irregularity, and even failure of the heart's action, may evidently be induced in these cases by several influences working separately or together, and by the exhaustion of the nervous and general forces induced by the accumulated effect of those influences, all tending to lower and exhaust the power of the heart, and even, as in the case just told, to arrest its action. Among such influences are, the pain and inflammation of the joints when severe, extensive, and prolonged; the pain in the heart and pericardium, the side, and the chest; the existence of endocarditis with its immediate and remote consequences; the presence of previous valvular disease; the grave influences exerted by great distension of the pericardium, which,—by compressing the venæ cavæ, the pulmonary veins, both auricles, and the aorta, impedes the supply of blood through the venæ cavæ and pulmonary veins to both sides of the heart, and through the aorta to the system, and causes the accumulation of blood in the lungs,—by pressing upon the bifurcation of the trachea and the left bronchus, and by lessening the size of the lungs, seriously embarrasses respiration—and by compressing the œsophagus, renders deglutition difficult; and the existence of congestion of the lungs, of pulmonary apoplexy and pleurisy, due to one or more of the causes just named.

Besides these, there are two important influences that may induce feebleness, irregularity, and perhaps even failure of the action of the heart; one, the inflammation of the superficial muscular fibres of the heart; the other, the inflammation of the nerves situated at the surface of the heart and great vessels. Inflammation of the superficial muscular fibres of the heart, which sometimes occurs in pericarditis, paralyzes the affected fibres. This paralysis of the inflamed fibres must in itself embarrass the action of the heart, especially when we consider that those superficial fibres turn inwards by a double entrance at the apex, to become the innermost fibres of the left ventricle, where they end in the papillary muscles of the mitral valve. But this influence cannot be limited to those fibres, but must extend in a varying degree to the other muscular structures of the organ so as to interfere with the exercise of their power; just as inflammation of certain limited fibres of a voluntary muscle, say the biceps, while it paralyzes those fibres, interferes with the exercise of the whole muscle.

The many and important nerves situated at the surface of the heart and great vessels may be more or less involved in the inflammation affecting those parts in Pericarditis. That accurate physiologist, Dr. Burdon Sanderson, remarks, "that nothing is known either as to the anatomical distribution of nervous elements in the hearts of mammalia, or as to the functions which they perform."[1] When, however, we consider that electrical or other excitation of the vagus retards the contractions of the heart, and if it is strong enough, arrests the organ in diastole, and in the dog, lessens arterial pressure, while division of the vagi produces acceleration of the contractions of the heart, and in the dog, increased arterial pressure; that the lower cervical ganglion of the sympathetic exercises an accelerating influence, not always in action, on the contractions of the heart; and that in the frog, the ganglion cells contained in the heart are

[1] Handbook for the Physiological Laboratory, p. 263.

the springs of its automatic movement; and that the surface of the heart is rich in nerves connected with the vagi, the sympathetic and the intrinsic ganglia of the heart, and that those nerves are therefore locally affected by the inflammation in Pericarditis; we must, I consider, conclude that this affection exercises in such cases an important influence, either to stimulate or to injure those nerves and so to accelerate or retard the contractions of the heart, to excite or, more frequently, depress the powers of the organ, and to increase or diminish arterial pressure. It is for the pathologist to ascertain, by direct experiment, the effect of the inflammation or irritation of the nerves on the functions of the heart.

It is right that I should mention another depressing influence on the action of the heart in Pericarditis, accidentally due, in the case about to be referred to, to treatment. In one case (17) already given at page 498, the loss of blood due to irrepressible hemorrhage from a leech-bite seemed to produce serious irregularity of the action of the heart.

XII.—Difficult and Quickened Respiration.

Respiration was disturbed to a marked degree in 49 of the 63 patients affected with rheumatic Pericarditis; it was slightly or not at all affected in 3, and in 11 its character was not recorded. The Pericarditis was severe in 2 only of the 11 cases in which the state of the respiration was not noticed, and in none of the 3 in which the breathing was but slightly affected; but the attack was severe in 37 of the 49 patients in whom the respiration was markedly disturbed.

The respiration was rendered difficult and quick by three or four local causes: first, in order of time, the inflammation of the heart, without and within, and of the pericardial sac, including the central tendon of the diaphragm, and the accompanying pain in the heart, the sac, and the diaphragm, with the consequent restraint imposed upon the movements of the latter; after this, the distension of the pericardial sac with fluid, which greatly enhanced the severity of the symptoms; and, at a later period, the supervention of pleurisy with its attendant permanent pain and stitch in the side, or of pulmonary apoplexy, often accompanied by pleurisy. The breathing is hurried, and rendered laborious by distension of the pericardium, often so as to demand a raised posture, owing to two causes, one, the encroachment of the swollen sac upon both lungs, and especially upon the lower lobe of the left one; the other, the direct pressure, backwards and upwards, exerted by the fluid in the tense pericardium on the trachea at its bifurcation, and on the left bronchus, a pressure that is materially relieved by the erect posture, and still more by the forward attitude which throws the volume of the liquid forwards and downwards towards the diaphragm and away from the trachea.

There was great distress, difficulty, and rapidity of respiration in 24 of the cases of rheumatic Pericarditis, and in one-half of them it is recorded that the patient was raised or propped up. The attack was fatal in 4 of those patients, and severe in 18, being very severe in 11.

One of those cases, a sawyer, aged 26, who had aortic and mitral valve-disease of old standing, came in feeling low and anxious, and was delirious at night. On the 5th day he was better, the respirations being 20 in the minute; but on the 10th he had pain and friction sound over the heart, and the respirations rose to 30 in the minute. The dulness over the pericardium increased, and reached its acme on the 19th. On the 16th he was seized with extreme and urgent dyspnœa, which was relieved when he was raised. The respirations were 70 during the attack, and fell after it to 35; on the 18th they varied from 36 to 44, and on the 21st, when the pericardial dulness had greatly lessened, they had fallen to 28 in the minute.

A man whose case I have already given, had Pericarditis with rubbing sound, on the 53d day, the pericardial effusion being at its height on the 57th. On the 55th the respirations were 44 in the minute, and he had extreme difficulty in breathing, which was relieved by the upright posture. On the 58th the pericardial effusion had lessened, the respirations had fallen to 24, and he breathed easily in the recumbent posture.

Another patient, a servant girl, breathed 32 times in a minute on admission, as well as on the 7th day when leeches were applied over the region of the heart. On the 8th friction sound appeared, and the effusion was at its height next day, when the respirations were 52, and on the 10th her head and shoulders were propped up. On the 11th the effusion had lessened, and her breathings numbered 40. On the 14th there was pleuritic pain, followed by friction sound, and the respirations rose to 48; but on the 20th, when there was no pain in the chest, they had fallen to 24.

In the following case, a female servant, the breathing rose in frequency a second time during a second wave of increased pericardial effusion. On the 6th the respirations were 28 in the minute; on the 7th they were 40; on the 9th friction sound was heard over the heart, and on the 10th the pericardial dulness was at

its height. On the 12th the effusion had lessened; she was in a raised position breathing more freely, 40 times in a minute; but on the 17th the fluid in the pericardium had again attained to the full; she had pulmonary apoplexy and pleurisy, and the respirations mounted up to 66; but next day, with a renewed diminution of the fluid, there was a renewed lowering of the respirations to 44.

I would gladly illustrate this point by additional cases, but shall limit myself to one more instance that shows the effect on the breathing of pulmonary apoplexy and pleurisy in cases of rheumatic Pericarditis. A young man was admitted with pain in the chest and shortness of breath. On the second day friction sound was heard, and pericardial effusion had already reached its acme; leeches gave relief, and the breathing was more free; but on the 6th he had a stitch in the side, and the respirations numbered 60 in the minute; on the 8th, when he was easier, they were 46; but on the 13th pulmonary apoplexy was established, and they had risen to 72. On the 17th he had diphtheria, the respirations being 50; on the 28th this was nearly well, and he raised little phlegm, the respirations being 36, and on the 35th they were 28.

We thus see that with pain over the heart and pericardium, the breathing is hurried and distressed, while it is slackened and relieved with the relief of the suffering; that with the rise and fall of Pericarditis, with the increase, the acme, and the decline of pericardial effusion, we have an increase, an acme, and a decline in the number of the respirations; that a second wave of increase in the amount of pericardial effusion, leads to a second wave of increase in the number of the respirations; and that the respirations are also again accelerated, if, in the later progress of the case, pleurisy should spring up from the spreading of the pericardial inflammation; or if pulmonary apoplexy should declare itself, especially if combined, as it usually is, with notable pleurisy.

XIII.—Difficulty in Swallowing.

There was difficulty or pain in swallowing in 13 of my cases of rheumatic Pericarditis.

I have already spoken of cases in which the act of deglutition caused pain over the back of the inflamed pericardium, generally complained of, however, in the chest, by the pressure of the morsel of food upon the inflamed structures during its descent along the œsophagus, where it passes behind the affected region.

The difficulty in swallowing, of which I now speak, occurs when the pericardial sac is distended to the full with fluid, and is caused by the compression of the œsophagus between the swollen sac and the spinal column. When the effused fluid lessens, the pressure diminishes, and swallowing becomes easy; but it becomes again difficult when a relapse takes place and the effusion again increases.

When the patient lies flat, the weight of the fluid in the pericardium falls backwards with full pressure upon the œsophagus, and deglutition becomes more difficult; when, however, he is raised into the sitting posture, and especially if he leans forwards, the volume of the liquid tends forwards and downwards, and swallowing is more easy.

A servant girl, aged 16, who had been ill about three weeks, came in suffering much both in the joints and the chest. Her breathing was laborious and very rapid; she looked anxious; dulness was increased over the pericardial region, and a soft friction sound was audible over the heart on pressure. On the 3d day the amount of effusion in the pericardium had reached to its acme; swallowing was difficult, breathing was accelerated, her face was livid and anxious, she had pain in the epigastrium increased by pressure, and the veins of the neck were full. On the 5th she still had much difficulty in deglutition, but on the 8th the pericardial dulness had lessened all round, and she swallowed much more easily. On the 9th she was more bright and lively, the pericardial dulness had lessened much, but pain came in catches over the heart. On the evening of the 10th she had a relapse, she became suddenly faint, her lips turned blue and dusky, and she had great pain over the heart, which was soon relieved, but difficulty in swallowing returned. Next day the dulness over the pericardium had again increased, and the difficulty in swallowing was very great. On the 12th she was still very ill, but she could swallow more easily, and on the 15th the effusion into the pericardium had again lessened, and she was better. The friction sound was audible until the 17th day. She improved daily and gained strength.

The poor female servant, who died from sudden failure in the action of the heart, whose case I have just related, on the day of her admission, when the amount of effusion into the pericardium was great, swallowed more easily when the shoulders were raised than when she was lying flat.

One patient, a female servant, had a fourfold attack of difficulty of swallowing; on the second day after admission, from great distension of the pericardium, the effect being heightened by shortness of breath; on the 4th from diphtheria; on the 7th from a renewed increase in

the effusion owing to a relapse, there being great distress in the chest; and on the 11th to a slighter degree from a second relapse with increase of the pericardial effusion. This case recovered perfectly without valvular mischief, after passing through an attack of pneumonia or rather pulmonary apoplexy and pleurisy.

Each patient presents some peculiarity in the way in which deglutition is affected; but I shall only allude here specially to two more cases; one of them, a youth, could not swallow solids readily, but could drink freely; the other, a coachman, aged 22, sometimes when drinking had a spasm which stopped his breath before he could swallow.

The possibility that diphtheria may be the cause of the difficulty of swallowing must not be overlooked. It was, as we have seen, the intervening cause, in my case (44*a*), with double relapse, and it was the cause of dysphagia in another patient (55), a young man of 18, a commercial traveller, who had diphtheria on the 6th day after the cessation of friction sound, and the 16th after admission.

XIV.—Loss of Voice.

In the case fatal from syncope, a female servant, to whom I have several times alluded, on the 5th day after admission the voice was husky, and she spoke in a whisper, but she could, with a great effort, speak aloud. She was less husky on the 5th, and on the 8th her voice was more natural. This case tends to support the view that the left laryngeal recurrent nerve may become so affected by the contiguous inflammation as to paralyze the larynx.

XV.—Effects on the Pulse of Rheumatic Pericarditis.

The pulse obeys the same law as the respiration under the influence of the disease; it rises in number, like the respirations, as the disease rises in intensity, is at its greatest rapidity when the disease is at its acme, and falls in number as the disease declines. The increase in the rate of the pulse is not as a rule in proportion to the increase in the number of the respirations. During the early stage of the inflammation of the heart, when pain is generally felt and friction sound is audible over the organ, the pulse usually mounts up to 90, 100, or even 120, while the respirations increase to from 30 to 40 in the minute, so that at this early period the ratio of the pulse to the breathing is in number as about three to one, instead of maintaining the healthy standard of about four to one.

When the amount of the effusion into the pericardium reaches its height, the pulse is usually quicker than it is during the early stages, and on rare occasions it becomes very much quickened, reaching even to 160. More often, however, the pulse is not more rapid at this the stage of the acme of the disease than it is during its early period. The breathing, as we have just seen, is almost always more quickened and laborious at the time the fluid in the pericardium has reached to its height than at any previous period, so that then the ratio in number of the pulse to the respiration is often two or two and a half to one, instead of maintaining the healthy ratio of four to one.

At a later period, when the effusion is lessening, and the inflammation of the pericardium is coming to an end, the pulse, like the respiration, falls in number. At this stage, however, in severe cases, one or other, or even both of the two secondary affections, pleurisy and pulmonary apoplexy, that quicken the respirations quicken also the pulse, when the numbers of both, and the proportion that they bear to each other, are as a rule nearly the same that they were during the early period of the attack, the ratio of pulse to respiration being usually three to one.

In considering the effects of rheumatic pericarditis on the pulse and respiration, I have separated from each other the advance, the acme, and the decline of the disease, and the two secondary influences, pleurisy and pulmonary apoplexy. In nature, however, those stages melt into each other, and those various causes combine and operate together to produce the hurry and distress of breathing and the quickening of the pulse of which I have just spoken.

XVI.—Fulness of the Veins of the Neck from Distension of the Pericardial Sac.

In several of the cases of rheumatic pericarditis there was fulness of the veins of the neck, sometimes with pulsation, during the period that the effusion into the pericardium was at its height, and the sac was distended to the utmost.

The fulness of the veins of the neck present at this period must, I consider, be mainly due to the resistance offered to the return of the blood through the venæ cavæ into the right auricle, owing to the yielding inwards of the thin walls of that cavity before the pressure of the fluid contained in the swollen pericardium. The fluid exerts also direct pressure upon the thin walls of the descending vena cava, which carries on the latter part of its course for the extent of an inch within the pericardial sac. The ascending cava, on

the other hand, sustains this pressure to a considerable extent by being short and very large, and by possessing walls thickened by fibrous structure derived from the central tendon of the diaphragm. We may, indeed, measure the degree of the distension of the pericardial sac by the degree of the distension of the veins of the neck. This compression inwards of the right auricle must be looked upon as one of the most serious consequences of pericardial distension, for it materially lessens, or in extreme cases may almost tend even to cut off the supply of blood to the right side of the heart, the lungs, the left side of the heart and the system. The walls of the left auricle, being thicker, do not yield so readily as those of the right, but the compression of the left auricle and of the pulmonary veins by the fluid in the distended pericardium produces its own special mischief by impeding the flow of blood from the lung, thus often inducing pulmonary apoplexy. From this joint compression of the sister auricles flows a succession of consequences to which I need not here allude in detail, but which in their turn tend to produce weakening and intermission of the heart, a feeble irregular pulse, and even death from syncope. I shall have occasion by-and-by to speak of the support that the thin walls of the auricles and veins derive from the coating of lymph with which they are covered, and which enables them to bear much of the pressure to which they are then subjected.

One patient, a servant girl, after being ill for a week and affected severely in the joints for two days, came in breathing hurriedly, suffering from pain over the region of the heart, and in great distress. There was dulness over the pericardium from the second space to the sixth, and a loud, harsh friction sound was heard over all that region. The left jugular vein was distended and did not empty during inspiration; next day the amount of effusion had lessened, she improved rapidly, and the friction sound ceased on the ninth day, when a mitral murmur declared itself.

In another servant, whose case, already referred to, proved fatal, the veins on the right side of the neck pulsated strongly, while those on the left side did so to a less extent, as they did not fill or empty themselves so completely. She died in a fit of syncope on the 14th day. Eighteen ounces of fluid were found in the pericardium, and several patches of pulmonary apoplexy were diffused through the upper lobe of the left lung.

Another fatal case, a carpenter, who died delirious on the eighth day, presented pulsation in the neck on the second day after admission, when the pericardial effusion had reached its acme. This pulsation was partly in the carotids but was chiefly venous and was more marked on the right side of the neck, the veins on that side being fuller than those on the left. On the third day the upper boundary of the region of pericardial dulness was lower, having descended from the third to the fourth costal cartilage, and the venous pulsation was not so perceptible. I will name two other cases of this class: one, a man who came in with an anxious expression of face: on the fifth day friction sound was heard over the heart, and on the seventh he presented extensive double venous pulsation in the neck: the other, already related, a girl who came in with rheumatic pericarditis, and in whom the veins of the neck were full during expiration on the third day, when the pericardial effusion was at its height and deglutition was difficult.

There was visible pulsation of the jugular veins in three of the patients who had been affected with valvular disease of some standing before being attacked with rheumatic pericarditis. In these cases, the venous pulsation was evidently due to the valvular affection.

XVII.—Appearance and Expression of the Face during the course of Pericarditis.

The face was flushed, dusky or very pallid, or its expression was one of anxiety or depression, in 43 of the 63 patients affected with rheumatic pericarditis. In six other cases it is stated that the aspect had improved, although there is no previous description of the face. There was thus a marked change in the appearance of the patient in four-fifths of the cases (49 in 63). The face is not mentioned in the remaining thirteen cases, and in one only of these was the attack severe, while it was so in thirty-six of the patients in whom its appearance was notably altered. The face was similarly affected in three-fifths of the patients attacked by endocarditis (60 in 108), in less than one-half of those who were threatened with endocarditis (27 in 59), and in one-fourth only of those who presented no sign or symptom of endocarditis. The appearance of the face was less and less profoundly altered in these patients, as the class to which they belonged became less and less affected in the heart, and still less in the class made up of those who gave no evidence of affection of that organ.

The face was flushed in 19 of the 63 cases of rheumatic pericarditis. Perspiration was copious in all but three of these, the perspiration often standing in beads upon the face. The flush, instead of being limited to the cheeks, was diffused over those parts that are usually white

even in persons of the most rosy hue, the forehead, namely, the eyelids, the nose, the white skin of both lips, and the chin. I never noticed the color spread at the first blush from feature to feature, but it seemed to tint them all at once. Thus the face was pallid on the day of admission in a fatal case already quoted by me, and on the following day it was flushed all over. But the flush which at first seemed to suffuse the whole face usually vanished step by step; the pink skin of the upper and lower lips first becoming white, then the nostrils and, in succession, the eyelids, the chin, the brow, and the cheeks in several of my cases.

The face was pale during the period of the friction sound in nine cases. One of these, a female servant, was very pallid and sallow, the features being pinched, when admitted with pericarditis; while on the following day, the face was rather flushed, and the fever seemed to be greater. Another case, a servant girl, aged 20, admitted with pericarditis, was flushed on the second day, but on the third, when the fluid in the pericardium had reached its acme, deglutition was difficult, and she was depressed, pallid, and weak. The face was twice as often flushed (19 times), as pale (9 times), during the attack of pericarditis. I have been unable to discover clinical reasons for the difference in these cases of the hue and color of the face. The clinical history of the pallor of the face induced by rheumatic pericarditis is illustrated by a case, the physical features of which I published in 1844;[1] a youth, aged 16, was admitted into the General Hospital near Nottingham, on the 17th of November, 1842, under the care of Dr. Williams, suffering from acute rheumatism, with pericarditis. His countenance was pale, and his surface generally was also pale. On the third day after admission, the general symptoms were milder, although the extent of pericardial dulness had not lessened, and the face was less pallid, the lips being red. On the sixth, the following is my report: "The gums are slightly tender, his general appearance improves, the hue of the skin is clear, and rather red; the reflex influence of disease in contracting the capillaries being removed." He made a complete and rapid recovery. In this case, the general surface was pale as well as the face; but in the cases under analysis, my notes do not, as a rule, describe the hue of the body.

The aspect was dusky, muddy, or glazed in sixteen, and the expression of the face was anxious or depressed in thirty-five of the patients affected with pericarditis.

I would here briefly compare these numbers with the numbers of those thus affected in the other cases of acute rheumatism.

The face was notably flushed in one-fifth of the cases with simple endocarditis (19 in 108), one-eighth of those threatened with endocarditis (8 in 63), and in one-twentieth of those giving no sign of endocarditis (4 in 79). The aspect was dusky or muddy in one-tenth of those with simple endocarditis (10 in 108), in one of those threatened with endocarditis (1 in 63), and in one of those who gave no evidence of endocarditis (1 in 79). The expression was anxious or depressed in one-fourth of those with simple endocarditis (25 in 108), in one-sixth of those threatened with that affection (10 in 63), and in one-twelfth of those who presented no sign of inflammation of the heart (6 in 79).

I have drawn up these numbers from a careful examination of my case books, and they present an accurate return of the symptoms there recorded. These cases are however necessarily reported with varying degrees of minuteness, and the more severe cases, attracting the greatest interest, are naturally observed and related with greater care than those that present no unusual features. These must therefore be taken not as the actual, but the approximate numbers.

Keeping this in view, it must be felt, from what I have said, that rheumatic pericarditis with endocarditis, and to a less degree simple endocarditis, produce a remarkable change on the complexion, aspect, and expression of the face. The attention is at once drawn to the heart by the altered countenance. When the inflammation of the heart is established, the varying hue and expression of the face tell, with remarkable accuracy, the varying state of the powers of the heart, and of the double inflammation with which the organ is affected.

When the tide of effusion into the pericardium has reached its height, as I shall illustrate in the next section, the hue of the face is usually more dusky and livid, and its expression more anxious than at any other time; but when the tide has fairly turned, and, the effusion having lessened, the inflammation ceases to be active, the face becomes often quite suddenly cheerful, while its hue becomes clear; the eye at the same time, instead of being heavy and charged with blood-vessels, becomes bright and clear. After this, if there is no relapse, the powers rally with remarkable quickness and freedom, and the appetite returns. This state is very different from the convalescence of fever, which passes through its period of improvement slowly and with scarcely perceptible steps.

In a patient, to whom I have already alluded, whose heart acted strongly and

[1] Prov. Med. Trans., vol. xii., 1844, p. 532.

rapidly at the time of the first onset of the inflammation, the right side of the face was swollen and flushed, evidently under the influence of the attack of pericarditis.

What are the causes of this remarkable influence of inflammation of the exterior and interior of the heart on the face?

There are probably more causes than one at work to produce the flushing or pallor present in pericarditis. The moderate elevation of temperature present in all cases of inflammation is probably connected with flushing of the face, either as a cause, or rather as a common effect. The question must here be put, what is the cause of the moderate elevation of temperature in cases of inflammation? Is it from general relaxation of the arteries, with elevation of temperature, owing to the influence of the inflammation on the afferent nerves of the part affected? such influence being conveyed to the vaso-motor centre in a manner analogous to that in which relaxation of arteries and elevation of temperature is produced on one side of the head and face by the division of the sympathetic on that side of the neck, or by the pressure of that nerve by an aneurism of the arch of the aorta. This influence would, of course, only account for the moderate rise of temperature in local inflammation, and does not touch the question of the cause of the increased heat in fevers or in cases of acute rheumatism with delirium.

Putting this cause aside, which applies to every case of inflammation, I would suggest that one great cause of the flushing or pallor of the face in pericarditis is the influence of the inflammation on the afferent nerves at the surface of the heart and great vessels, which, being depressed or stimulated, may induce reflex dilatation of the arteries of the head, with flushing of the face, or reflex contraction of the arteries of the head with pallor of the face. I suggested this in principle as the cause of the pallor in the Nottingham case in my note-book in 1842, and am still disposed to do so. In aneurism of the arch of the aorta, pressure on the branches of the sympathetic of one side causes relaxation of the arteries and elevation of temperature on the corresponding side of the head and face. I consider that a parallel effect would result from the excitation or the injury of the sympathetic and sensory nerves, and perhaps of other nerves having, say, a vaso-inhibitory property distributed to the seat of the inflammation of the heart and great vessels in pericarditis; contraction of the arteries of the head and face with pallor being produced on the one hand, and relaxation of those arteries with flushing and perspiration on the other. In one case only, just referred to, was there flushing and perspiration notably limited to one side of the face. It is natural, however, to expect that as the inflammation affects the nerves of both sides in pericarditis, both sides of the face should be equally affected, as it was indeed in all but one of my cases of pericarditis affected with pallor or flushing of the face.

I would here remark that as the reflex contraction or dilatation of the arteries with pallor or flushing, from excitation or injury of the sympathetic or sensory nerves is *continuous*, it differs essentially from the reflex movements of the muscles caused by the excitation of an afferent nerve, such movements being necessarily short and *intermittent*, the withdrawal and renewal of the stimulus to the afferent nerve being needful for their reproduction. In short, the reflex vaso-motor current is continuous, while the reflex excito-motor current (of the muscle) is interrupted.

The increased contraction of the arteries caused by the excitation of a sensory or sympathetic nerve appears to be due to the increased discharge of nervous force directly from the vaso-motor centre when that centre is thus stimulated by the excitation of those nerves. That centre would indeed seem to require, for the exercise of its proper functions, to be reinforced and stimulated through the sympathetic nervous system, and probably by the blood circulating in the arteries, when we consider that the division of the left splanchnic in the rabbit may lower the arterial pressure from 90 millimetres to 41, that excitation of the divided nerve may raise the pressure to 115 millimetres, and that division of the other splanchnic may further lower it to 31 millimetres.[1]

I would here remark that similar effects are produced by analogous causes in pneumonia, and especially in pneumonia of the upper lobe, when the face, besides being congested, presents a dusky hue and a powerless expression that speak of the profound influence exercised upon it by the disease. In this disease also, as in pericarditis, with the turn of the tide of the inflammation and with the removal of its products, the veil is as it were lifted away from the countenance; and a patient, one day under the weight of the inflammation, with an aspect dark, depressed and anxious, presents on the next day, with the removal of the exudation from the affected air-cells, and the renewal of their respiration, a face clear and clean, and an expression bright and cheerful.

The eye is every now and then reported to have been dull, and heavy in appearance during the attack of pericarditis, its minute vessels being congested; but it is more frequently described as becoming

[1] Ludwig and Cyon, quoted by Dr. Burdon Sanderson: "Handbook for the Physiological Laboratory," p. 249.

bright and clear when the effusion into the pericardium was lessening, and the inflammation was becoming inactive and only present in the shape of its results. I had not, until quite recently, made any close observation of this organ, but in one of the last cases of acute rheumatism with endocarditis treated by me in St. Mary's Hospital, I found that during the acme of the disease, when the face was flushed, dusky and anxious, the conjunctiva was crowded with small vessels which ended a very short distance from the cornea, so that round the clear of the eye there was a white zone or ring edged by fine converging vessels. When the inflammation ceased to be active, and the face, in keeping with this improvement, became clear and cheerful, the eye became bright, and the vessels crowding the conjunctiva lessened in number, so that the ball of the eye became again white. This organ requires careful observation in cases of rheumatic pericarditis and endocarditis.

XVIII.—Condition of the Face when the Pericardial Distension was at its Height.

When the pericardium is distended to the full with fluid, under the three-fold influence of (1) what may be termed the "fluid" pressure, induced by the distension of the sac bearing with varying force, outwards upon the œsophagus and trachea, the left bronchus, the lungs, especially the left, and the diaphragm; and inwards on the descending vena cava, the right and left auricles, and the pulmonary veins; (2) inflammation involving the nerves distributed to the surface of the heart and the great vessels; and (3) inflammation of the superficial muscular fibres of the heart itself; as we have seen, point by point, pain may be present around and within the heart, over the pericardial sac and the pleura; swallowing may be difficult; the voice may be hoarse or reduced to a whisper; the action of the heart, which at the beginning of the attack is often forcible, may become feeble and intermitting, or even altogether fail; the respirations may be hurried and laborious, often so as to compel the raised and forward posture; the pulse may be rendered weak and irregular and be quickened, though not in the same proportion, as the breathing, the ratio of the pulse to respirations being two or three and a half to one, instead of, as in health, four to one; and the veins of the neck may be swollen and pulsating. The last effect of the over-distension of the pericardium that I shall illustrate is that upon the circulation of the head and face.

A female servant whose case has already been alluded to was admitted with acute rheumatism and pericarditis of great severity. On the third day, I found that the pericardium was distended to the full, she complained of a sensation of choking, swallowing was difficult, the countenance was anxious, the face was livid and perspiring profusely, and the veins of the neck were full. On the sixth day the pericardial dulness had lessened all round, her face was less dusky, and her aspect had improved. On the tenth, in the evening, she suddenly came over faint, the lips being blue, and the face dusky; but in a few hours the face, though still anxious, lost its dark hue and the lips became again red. Next day it was found that the pericardial effusion had again increased. The fluid, however, soon again diminished. On the twelfth her aspect had again improved, on the fifteenth her face was flushed, and on the sixteenth it was of good color, and its expression was no longer anxious. Here, twice over, the effusion in the pericardium reached its acme, and under the influence of its pressure and the inflammation of the organ, the heart faltered, the venous blood was delayed in its passage, the arterial blood was with difficulty supplied, the face and neck became charged with venous blood, and the lips became livid; and here, twice over, the pressure was removed by the lessening of the fluid, when the color returned to the face and the expression of anxiety disappeared.

XIX.—Affections of the Nervous System in Rheumatic Pericarditis.

Dr. Davis, of Bath, in the year 1808[1] published three cases of acute rheumatism, two of them being affected with pericarditis, and one with endocarditis. One of the cases of pericarditis, which was observed in 1785 by Dr. Haygarth—who curiously does not mention this important case in his "Clinical History of the Acute Rheumatism," published in 1806—was affected with moaning, restlessness, and delirium ending in death. The other case of pericarditis, a young lady, who was under the care of Dr. Davis, had great heats, with perspiration, screaming, and the most violent jactitation of the body. "occasioned by the extreme anguish which she felt in the region of the heart." She was perfectly sensible throughout, and died after the disease had lasted twenty-six days. The patient with endocarditis was affected with want of sleep and violent delirium, for nine days, at the end of which time she died.

[1] "An Inquiry into the Symptoms and Treatment of Carditis," by John Ford Davis, M.D.

In a series of important clinical contributions, Corvisart, Mr. Stanley, Dr. Abercrombie, Dr. Macleod, Andral, Dr. Latham, Dr. Bright, Dr. Mackintosh, M. Bouillaud, Sir Thomas Watson, Sir George Burrows, and Dr. Kirkes, have described cases of pericarditis, some connected with acute rheumatism, but many not so, in which delirium, coma, convulsions, temporary insanity, chorea and choreiform movements, or tetaniform symptoms and rigidity, and even actual tetanus were present.

These observations suggested to several of those authors, including Andral and Dr. Bright, a close connection amounting even to cause and effect, between pericarditis and the affections of the nervous system associated with it.

The affections of the nervous system in cases of rheumatic pericarditis, and acute rheumatism are always serious, often fatal, and comparatively rare. Recent observations have shown in many of those cases the presence of a very high temperature, delirium and coma, ending in death. I shall therefore, in inquiring into the clinical history of those associated affections, examine those cases admitted into St. Mary's Hospital under my care during the twenty years that I have held office, and all the published cases that I can find of this class.

I have brought together from various sources, 180 cases of acute rheumatism with affections of the nervous system, more than one-half of which had pericarditis (92 in 180). The temperature of the body was recorded in one-third of the total number of cases (61 in 180); and although these cases were observed at a much more recent period than those in which the temperature was not recorded, I shall examine the more recent series of cases first, for they throw light upon the old series of cases.[1]

Cases of Acute Rheumatism with Affections of the Nervous System, in which the Temperature of the Body was Observed.

Dr. Sydney Ringer published in the year 1867, three cases of acute rheumatism with pericarditis, in which the temperature rose before death respectively to 109·2°, 110·8°, and 110·0°.[2] These three patients had delirium, followed by coma and death, and one of them was under the care of Dr. Reynolds as early as May, 1862.

Dr. Kreuser related in 1866[1] three fatal cases, of acute rheumatism in which the temperature rose respectively to 109·4°. 110·2°, and 110·4°, and these three patients were affected with delirium, and one of them with coma also.

More recently an important series of cases of this class have been communicated by Dr. Hermann Weber in an important paper, Dr. Murchison, Dr. Burdon Sanderson, Dr. Greenhow, Dr. Southey, Dr. Henry Thompson, Dr. Meding, Mr. Anderson, Dr. Wilson Fox, whose work on the treatment of hyperpyrexia is of great value, and Dr. Andrews.

I have brought together from these and other sources, sixty-two cases of acute rheumatism, affected with coma, delirium, chorea, or convulsive choreiform, or tetaniform symptoms, in which the temperature was observed during the progress of the illness.

Of the sixty-one cases in which the nervous system was affected, and the temperature was ascertained—I. twenty-seven had pericarditis; II. thirteen had simple endocarditis; and, III. twenty-one were free from pericarditis, endocarditis being absent or doubtful.

I.—Cases with Pericarditis in which the Nervous System was affected, and the Temperature, generally very high, was observed.

SUMMARY.

A[1] Had *coma* without *delirium*, maximum temp. 110° . . .	1
A[3] Had *delirium* followed by *coma*, temp. 110·8°–104·6° . . .	11
A[4] Had *delirium* followed by *stupor*, temp. 106°–103°	1
Had *delirium* and *convulsive movements*, temp. 107°–110·2° . .	1
B[1] Had uncomplicated *delirium*, temp. 110·4°–103°	9
Had *delirium* with general stiffness, temp. 103·2–102·2° . . .	1
Had *temporary* or *partial coma*, temp. 101·8°9–9·3°	2
C[1] Had *chorea*, temp. 101·5° . .	1
Total	27

The temperature of the body was observed in twenty-seven cases of rheumatic pericarditis with affection of the nervous system, and was very high in three-fifths of them (15 in 27), their highest temperature varying respectively from 106·8° to 115·8°. Five of these cases were placed in a cooling bath, when their temperature, then at the highest, was ascending

[1] The Tables which here follow are omitted in the present edition, on account of their bulk and complication.—H.]

[2] Medical Times and Gazette, 1867, ii. 378.

[1] "Medicinisches Correspondenz-Blatt des Württembergischen ärtzlichen Vereins," band xxxvi. p. 105.

rapidly, with the effect of arresting its rise, cooling the body, and, in four instances, leading to the recovery of the patient. The bath was employed also in two cases in which the temperature was 105° and 105·5° respectively, with the effect of cooling the body; but as the ascent of the thermometer was neither rapid nor very high, those cases can scarcely be included with those of hyperpyrexia. The temperature was 104·6° and 105·3° respectively in two cases during the period of delirium, but was not observed during that of coma, and I therefore think that both those cases may be included with those of hyperpyrexia—which bring their number up to seventeen, or two-thirds of the total number of cases with Pericarditis.

Seven of the remaining ten cases, or one-fourth of the total number, had a high, but not very high, temperature, varying from 103° to 106°, so that these cases would rank, as regards the heat of the body, with fever or pyrexia. The temperature was only moderately high in the three remaining cases, or one-eighth of the total number, varying from 99·3° to 101·8°.

A^1 Profound coma, without delirium, was present in one case; A^3, ^{4}, delirium that passed into coma in eleven cases, into stupor in one case, and into convulsive movements in one case; *B*, uncomplicated delirium was present in nine cases, one of which had Bright's disease; and delirium with stiffness of jaws, neck, and limbs occurred in one case. Temporary coma occurred in one case, and semi-consciousness in another, both with albumen in the urine; and C^1 chorea and slight continuous contraction of certain muscles existed in one case.

A^1 The case of coma without delirium, a woman, was under the care of Dr. Wilson Fox,[1] with acute rheumatism and Pericarditis. The temperature was about 102° on the morning of the fourteenth day of illness, and had risen to 108·4° at 9.15 P. M., when she became entirely unconscious, and to 109·1° at 9.50 P. M., when she was put into a bath at 96°, and ice was applied to her body. She was unconscious, pulseless, and cyanotic, her respirations were irregular, gasping, and stertorous, and she appeared to be dying. In half an hour her temperature had fallen to 106·2°, when the pulse became perceptible, and she showed signs of consciousness. In ten more minutes the temperature was 103·6°, and she was taken out of the bath, and twenty minutes later it had fallen to 100·1°, when she could speak, and had imperfect consciousness. After various oscillations, this patient recovered. I relate this case here briefly not to illustrate the treatment, but to show that profound coma became established when the temperature was excessively high, and that consciousness was restored when the body was cooled.

A^3 Ten cases, in which delirium was followed by coma, and in which the bath was not used, proved fatal, but one such case recovered after the employment of the bath.

Delirium appeared at a temperature of from 103° to 104·8° in eight of the eleven cases in which coma was preceded by delirium, the temperature in six of these being at or above 104° when the disturbed state of mind was first noticed. In three of these cases delirium was still present when the thermometer was as high as from 107° to 108°, and in one of them when it was as low as 99·6°.

Profound coma declared itself when the temperature had risen from 109° to 109·4° in five of the eleven cases in which complete unconsciousness followed delirium, when the thermometer stood at 108·4° in one of them, at 106·8° in another, and at 106·6° to 107·6° in another, in which the coma, not profound, was transient.

In several of these cases it was noticed that the temperature rose between the supervention of coma and death.

The delirium was violent in five of those eleven patients who passed from delirium into coma, two of whom got out of bed; was active in three of them; and resembled delirium tremens in two, while in another the manner was strange and excited, and the sentences were disconnected and incoherent.

The transition from a state of violent or active delirium to coma was usually gradual. Muttering replaced active delirium in three instances, the muttering delirium being accompanied by restlessness in two of them. A state of semi-consciousness, accompanied by moaning in one and by restlessness in the other, intervened in two cases between the period of delirium and that of coma; and two other cases passed from delirium to a state almost of unconsciousness, and from that to coma. Violent delirium ceased abruptly after venesection in one patient, who was quiet for a short time, but soon passed into a state of perfect unconsciousness.

The duration of the delirium was very various in the different cases, lasting in one case about three-quarters of an hour, and in another eight days. The delirium was more frequent by night than by day, and lasted from one to four nights in four cases in which it was scarcely observed during the day.

The period of coma varied much less than that of the delirium, lasting from a quarter of an hour to seven hours in nine of the eleven cases with delirium and

[1] "Treatment of Hyperpyrexia," by Dr. Wilson Fox, p. 2.

coma. In one of the remaining cases, the duration of the coma was prolonged, death being delayed, and in another of them consciousness was restored, and recovery was established, by the use of the bath.

The two cases were fatal in which delirium preceded semi-stertorous breathing, with violent spasmodic movements of the whole body in one instance, and profound stupor in the other. The temperature in the former case rose to 110·2° before death, but in the latter it was never higher than 106°. Dr. Murchison favored me with the leading features of that case.

B[1] Delirium without coma or other important modifications affected nine cases of acute rheumatism with pericarditis. These cases divide themselves naturally into two groups; in the first group, consisting of four, the delirium was of the usual character, and the temperature was very high, varying from 107·3° to 110·5°, and was kept in check in two of them by the cooling bath; while in the second group, containing four cases, the temperature was not so very high, varying from 103·3° to 105·3°. The delirium was accompanied by tremor, and usually by hallucinations, and a general condition resembling delirium tremens. The remaining case of delirium belongs to neither of these groups, since the delirium was slight, and gave way to general emaciation, ending in death.

Delirium was present throughout in one of the four cases with very high temperature, and in that patient it ranged from 103° to 105·6°, and ascended to 107·4° during the last ten hours. Death was sudden. The second case, a coachman who had lived well, was under the care of Dr. Wilson Fox.[1] His temperature was 107°, his pulse 100–108, respiration 44–45. At 2 A. M. he was put for twenty-five minntes into a bath at 89° to 86°, when his temperature fell from 107° to 103·1°, and he became perfectly conscious. Fifteen minutes after the bath his temperature had fallen to 98°, when his pulse was 84, respiration 20, and he was perfectly rational and conscious. The pericarditis in this case was of unusual duration and severity. The bath, the wet-pack, or the ice-bag was employed during the next six days to keep down the temperature, which had a strong tendency to rise. This patient recovered. In another case, a man, the temperature was lowered by the bath from 108·2°, when he was delirious, to 103·8°, when he could answer questions rationally. A second bath lowered his temperature from 105° to 102°, and thirty-five minutes after his removal from it, to 98·7°, when he was quiet. He recovered slowly.[1]

One of the four patients with tremor, hallucinations, a state resembling delirium tremens, and a temperature not excessively high, who was under the care of Dr. Southey, was an intellectual, nervous man, and a drinker of beer. His tongue and hands were at a temperature of 105°; he was placed in a bath at 71° for ten minutes, when he felt cold, talked rationally, and thought it the queerest treatment for rheumatism. He was wet-sheeted whenever his temperature rose to 104°, when he was always delirious. He died with bronchial symptoms. Sir William Gull saw the case, and suggested that it indicated the association of acute rheumatism with delirium tremens.[2]

The next case resembling delirium tremens was a poorly nourished, pale man. The bath lowered his temperature on the first occasion from 104·3° to 99·8°, and on the second from 105·3° to 101·6°, when he was rational, and after the second bath he had *risus sardonicus*, his limbs were tremulous, and he remained delirious until the fourteenth day (temp. 103·4° to 100·2°). After this he steadily improved.[3] Dr. Southey favored me with the notes of another case of this class, a poorly nourished, anæmic man, a coachmaker, who had been ill ten days. When admitted (temp. 103°), his tongue was tremulous, and he perspired profusely. On the fifth day he had pericarditis; on the seventh night, constant muttering delirium; and next day an abrupt manner. On the ninth, after a delirious night, his hands were tremulous. On the seventeenth day his skin was hot and dry, temp. 103·8°; and the activity of his mind resembled what is observed in delirium tremens, but he had no horrors. The ice-bag was applied to his head on the eighteenth, and as he was sleepless, he had 30 grains of chloral, after which he slept for four hours. On the following day he was conscious, had pain in the knees and shoulders, perspired less, and looked better, but still had some tremulousness and jactitation. His respiration and temperature steadily fell, and he gradually recovered.

The fourth case was a constable, who ten years before had been unconscious after a kick. His highest temperature was 103·3°, but it rarely exceeded 102°. In the course of his illness he had delirious nights, choreal movements of the left hand, on one occasion tremor of the right hand, hallucinations, and frequent rolling of his head from side to side. He im-

[1] "Treatment of Hyperpyrexia," p. 10.

[1] Dr. Andrews, "St. Bartholomew's Hospital Reports, x. 338.

[2] Lancet, 1872, ii. 562.

[3] Dr. Andrews, "St. Bartholomew's Hospital Reports," x. 350.

proved slowly, but remained for some days incoherent and childish in manner.[1]

B[1] Delirium with stiffness of jaws, neck, back and limbs, occurred in a patient of Dr. Bristowe's, a bargeman, aged 21, with slight acute rheumatism, pericarditis, and endocarditis.[2]

Two cases, one affected with temporary unconsciousness, the other with stridor and semi-consciousness, were under my care in St. Mary's Hospital. They had albumen in the urine, and were both fatal. The first case had previous aortic and mitral valvular disease. The second had a presystolic murmur, and mitral and tricuspid systolic murmurs, and the inspection after death showed pericarditis, button-hole contraction of the mitral valve, and acute Bright's disease of the kidneys.

C[1] Choreal and continuous contraction of some muscles occurred in the following case, a delicate, excitable girl, aged eleven, for observing which I am indebted to Mr. Saunders. When I first saw her, about the tenth day of her illness, a loud pericardial friction sound prevailed over the whole front of the chest, extinguishing all other heart-sounds. Ten days later, temp. 101·5°, she took little notice, bent and extended her right arm and hand irregularly, but bent the hand backwards on the forearm, flexed the fingers, and pointed the right great toe downward, by the continuous, but not constant, contraction respectively of the flexors and extensors of the forearm and the muscles of the calf. The face was still, the tongue protruded itself steadily and for long; her body was quiet, and speech was limited. During the night she alarmed her mother by screaming violently, throwing herself about the bed, and tossing her head from side to side. After about twenty minutes she became quiet and fell asleep. Four days later she had a return of pain and swelling in the right knee, friction sound was barely audible over the heart, and the movements of the right arm had lessened and were more simply those of ordinary chorea.

The *affection of the joints* during the early period of the attack of acute rheumatism was severe in three-fifths (15 in 27), and of moderate severity in one-third of the patients (8 in 27), not severe in two instances, and in one, the condition of the joints was not described. The affection of the joints disappeared, or was much lessened in severity at the time of the delirium, coma or chorea in all those cases (20 in 27) in which the condition of the joints is described. In thirteen cases the affection of the joints was well at the period in question; in four it was slight, and in three it was not severe.

The invariable subsidence of the inflammation of the joints in these cases, when affection of the nervous system takes place, shows that there is some connection between the appearance of the one affection and the disappearance of the other. The improvement of the inflammation of the joints generally coincides with improvement of the general symptoms, unless the heart is inflamed. We may therefore, I think, infer that the presence of trouble in the nervous system, whether accompanied or not by a very excessive rise in temperature, has a distinct association with the lessening of the affection of the joints.

The *perspiration*, before the nervous system was affected, in these cases was noted in ten of the fourteen cases with coma, stupor or convulsions preceded in all but one instance by delirium, and during that early period it was profuse in seven, and moderate or slight in three of those ten cases. The perspiration was observed in eleven of the fourteen cases just noticed during the period of delirium or coma, when it was absent in three, slight in four, moderate or considerable in two, and profuse in two of these eleven cases.

The perspiration was noted both before and during the period of the delirium or coma in nine of those fourteen cases. In eight of those nine patients, the temperature was excessively high at the time of the delirium or coma, and perspiration was then absent or lessened. In one case with delirium, the highest temperature observed was only 103·8°, and perspiration, previously moderate, was then profuse.

The perspiration was observed during both periods in four of the nine cases in which delirium was present without coma or stupor, and was profuse in those four cases during the early period of the disease. One of those patients had on previous days perspired freely, but the skin became dry when the temperature rose to 107·3°. In another of them, the skin previously perspiring, felt hot and dry when delirium appeared at a temperature of 103·8°. The perspiration remained profuse in two cases during the period of delirium with hallucinations and tremor, the temperature being then respectively 105° and 102°. Both of those patients were predisposed to affections of the nervous system. Dr. Wilson Fox justly regards the cessation of perspiration while the temperature is still high as a symptom of very great gravity. It would appear from what I have just stated, that the cooling influence of the perspiration may have kept down the temperature in the three latter cases, while in the ten former cases the want of that cooling influence may have allowed the tempera-

[1] Dr. Greenhow, Clin. Soc. Trans. vii. 172.
[2] Path. Trans. xxiv.

ture to rise unchecked when heat was supplied from within by the rapid combustion of the tissues of the body during the disease.

The presence of a *miliary eruption* or sudamina was noticed in nearly one-half of the cases (12 in 26).

The *Pericarditis* was of average intensity or severe in eleven and slight in three of the fourteen cases with coma, stupor, or convulsions, in all of which but one the more grave affection of the nervous system was preceded by delirium. In seven of the nine cases with uncomplicated delirium, the pericarditis was of average severity, and in two of them it was slight. The pericarditis was of average severity in the remaining three patients, in none of whom was the temperature above 101·5°, none of them having transient coma, one of them coma, and the other choreal symptoms.

We shall be better able to consider whether the presence of pericarditis had any influence in producing the excessive rise of temperature in cases with "hyperpyrexia" when we have inquired into the whole chain of cases, those namely without as well as those with that affection.

Endocarditis was present in nearly one-half of these cases, with pericarditis and affection of the nervous system (11 in 26), was absent in almost as many (9 in 26), and was doubtful or not noted in the few remaining cases (5 in 26).

Convulsive, Choreiform and Tetaniform Movements.—Movements of a convulsive, choreiform or tetaniform kind affected nine of the twenty-four patients with acute rheumatism and pericarditis in whom the temperature was observed, including the case just related in which choreal symptoms were present without delirium. Besides these, two patients affected with delirium had distinct *risus sardonicus*.

One of these patients, a shopman in a cigar shop, aged 28, had in the morning muttering delirium, and a temperature of 107°. In the afternoon he had violent spasmodic movements of the whole body, his respirations were semistertorous, his temperature was 110·2°, and an hour later he died.[1] Another of them, a female servant, being violently delirious, temp. 107·8° F., was bled, and became, as I have already stated, abruptly unconscious. Then succeeded a peculiar series of irregular muscular movements of the hands and arms, with chattering and grinding of the teeth, and convulsive movements of the jaw, or trismus. Fully two hours later, after being in the bath, when she had cooled down to 104°, she had an attack of clonic spasms of the muscles of the arms, lasting some minutes.[1] There were muscular twitchings of the limbs in three patients when in a state of unconsciousness.

One patient, a police-constable, aged 23, who, ten years previously, had been unconscious from a kick in the mouth, after little sleep, had wandering, much jactitation, constant movement of the fingers of the left hand, tremors of the right hand, and subsultus. Two days later there was also frequent rolling of the head from side to side. His temperature was not above 102°.[2] Another patient, a woman aged 29, also rolled her head from side to side, contracted her brows, and distorted her face into various grimaces. Her temperature was 107·8°.[3] One patient, a man aged 23. on the evening before he died, temp. 105·4°, was very delirious, and rolled violently about the bed, so that he required to be held down. This violence quickly passed away, and he then lay half unconscious and moaning loudly.

Symptoms of a more or less tetaniform character, that is to say, with continuous rigidity or contraction of muscles, appeared in five of the cases.

Dr. Wilson Fox's patient, already sketched at page 516, after the bath, temp. 100·6°, had at times spasms of rigidity of the muscles of the lips and neck, but not of the limbs. Another patient, a gardener, seven hours before death, became incoherent, and within ten minutes, unconscious; his lips pouted and rubbed incessantly over the teeth, and his whole voluntary muscles twitched constantly.[4] The third is that of Dr. Wilson Fox just referred to, with chattering and grinding of the teeth, and convulsive movements of the jaw, or "trismus."[5] The fourth case is Dr. Greenhow's, already noticed, with choreal movements of the left hand. When that hand was turned on to its back,[6] there were constant twitching movements of the hand and fingers, and the forefinger became flexed towards the palm. The fifth case is my own, already related at page 517, with choreiform movements of the right arm. Her right hand was bent backwards, her right fingers were flexed, and her right toe pointed downwards, owing to the continuous contraction of the corresponding sets of muscles, which offered steady resistance when put on the stretch.

[1] Dr. Murchison, Clin. Soc. Trans. i. 32.

[1] Dr. Fox, "Treatment of Hyperpyrexia," 44.

[2] Dr. Greenhow, Clin. Soc. Trans. vii. 175.

[3] Dr. Sydney Ringer, Medical Times and Gazette, 1867, ii. 380.

[4] Mr. Anderson, British Medical Journal, 1871, i. 529.

[5] Loc. cit. p. 48.

[6] Loc. cit. p. 174.

These five cases seem to suggest a combination of convulsive, choreiform and tetaniform movements.

The question naturally arises, were the cases presenting choreiform movements associated with endocarditis? The answer to that is, however, as regards these cases, definitely in the negative, for endocarditis was absent, or not observed, in those cases, excepting to a slight and doubtful degree in one of those with muscular twitching. Endocarditis was, however, present in Dr. Wilson Fox's case with spasms of rigidity of the muscles of the lips and neck. I shall again briefly consider these cases when I return to the important question of the association of pericarditis with tetaniform and choreiform movements.

Tremor was present in seven of the cases, all of which have been already alluded to.

II.—Cases with Simple Endocarditis in which the Nervous System was affected and the Temperature, generally very high, was observed.

SUMMARY.

A^3 Had *delirium* followed by *coma*, temp. 104·4°–110·2° . . .	4
A^2 Had *delirium* and *convulsive movements*, temp. 111·6° . . .	1
B^1 Had uncomplicated *delirium* in three, temp. 108·5°–111·4°; in two, temp. 102·8°–103·9° . .	5
B^2 Had *delirium*, cerebral embolism and hemiplegia, temp. 103° .	1
B^3 Had *delirium* and *chorea* (minute cerebral embolism) temp. . .	1
Had high temperature without notice of delirium, temp. 105·8° (ice-bag) .	1
Total	13

The nervous system was affected in thirteen cases of simple endocarditis in which the temperature was observed. The majority of these cases, like that of those affected with pericarditis, presented an excessively high temperature; and in three of the whole number the temperature, when undergoing a rapid ascent, was arrested in its rise, lowered, and kept down by the use of the cooling bath or the external application of the wet sheet and ice. The temperature was as high as from 108·5° to 111·6° in three-fifths of the cases (7 in 13); and in the one of those cases in which the temperature was the lowest, 108·5°, the vigorous use of ice-cold water within and without arrested the rise of temperature and induced its permanent lowering, followed by the recovery of the patient. In one patient the temperature was checked at 105·8°, and brought down by the bath; and in another the thermometer was at 104·4° during the period of delirium, but was not employed during that of coma. In four of the cases the temperature was only of a moderate height, being from 103·9° to 102·3°; and we may therefore infer that fully two-thirds of the cases with simple endocarditis (9 in 13) in which the nervous system was affected, had "hyperpyrexia."

$A^{2\text{-}3}$ Twelve of the thirteen cases had delirium, which passed into coma in four instances, ended in convulsive movements in one, B^1 was without complication in five, was associated with B^2 cerebral embolism and hemiplegia in one patient, and with B^3 minute cerebral embolism and chorea in another. In one instance, in which the temperature was high (105·8°), there was no note of delirium.

A^3 One of the four cases in which delirium passed into coma was a delicate, ailing woman. On the seventh day her temperature in the morning was 102°, but it rose in the evening to 109·5°, when she was comatose. For want of a bath, she was taken downstairs, placed, doubled up, in a washing-tub containing water at 80° cooled to 62°, and cold water was ladled over her body. Spasms soon came on, which were more and more continuous until she was taken out of the bath in one of them after being there for forty-five minutes, while her temperature had fallen to 100·3°. Towards midnight she was much convulsed, the teeth closing firmly on the lower lip and drawing blood. On the tenth day the temperature rose to 105·1°, she was again put into the tub for fifty-eight minutes, and at the end of that time was taken out in a state of well-marked opisthotonos, which passed off gradually in about two hours. She died on the twelfth day.[1]

B^1 Three of the four cases with delirium without coma had high temperatures, 111·4°–108·5°; while in two the temperature was comparatively low, 103·9°–102·8°. One of the patients with delirium and high temperature was a female servant, aged 22. On the eighth day of treatment, temp. 108·5°, her sensorium was much disturbed, and her skin, which hitherto had been moist and sometimes covered with sweat, was dry. Cold was used energetically. Ice-cold water and cloths were applied freely to the body, and ice-water enemata were given every half-hour. In an hour's time she breathed more freely, her head was relieved, and the pulse fell. At half-past six in the evening her temperature was 98·6°, skin perspiring, mind clear, and she felt like a new-born person. Two

[1] Dr. Andrews, "Bartholomew's Hospital Reports," x. 346.

days later she sat up in bed, and took food with appetite.[1]

In the two cases with comparatively low temperature the delirium was only present during the night. The temperature was 103·9° in the daytime in one of these patients, and 100·4° in the other. Convulsive movements affected four of the thirteen patients belonging to this group with endocarditis.

The *affection of the joints* was severe in eight and was rather severe in one of the thirteen cases with simple endocarditis before the period of delirium or coma; while it was absent in two and not severe in three; and its condition was doubtful in four of those cases during that period.

Perspiration was profuse in five and absent in one of the cases of simple endocarditis before delirium set in; and it was absent in two, slight in one, probably profuse in one, and doubtful in two of those cases after the appearance of delirium, while it was profuse in another patient who was delirious when admitted and whose temperature never rose above 102·8°.

III.—Cases in which there was no Pericarditis, Endocarditis being absent or doubtful, in which the Nervous System was affected, and the Temperature, generally very high, was observed.

SUMMARY.

A^3 Had *delirium* followed by *coma*, temp. 111·1°–105·8° . . .	6
A^4 Had *delirium* followed by *somnolence*, temp. 106°	2
B^1 Had *delirium* uncomplicated, temp. 110° to 100·4°	10
Very high temperature without *delirium*, temp. 110·8°–106·3° .	2
Twitching of limbs, temp. 102° . .	1
Total	21

Twenty-one cases had no pericarditis, endocarditis being absent or doubtful; and the majority of these cases, like that of those with pericarditis and with simple endocarditis, presented an excessively high temperature; and in five of the whole number the temperature, when undergoing a rapid ascent, was arrested in its rise, lowered, and kept down by the use of the cooling bath, the wet sheet, or ice. The temperature was as high as from 106° to 111·2° in three-fifths of the cases (12 in 21), being kept down in the one of those in which it was the least high by the use of the cooling bath. In one-fifth of the cases (4 in 21), the highest ascertained temperature varied from 106° to 103·4°, and in these the cooling bath was not employed. In one-fourth of the cases (5 in 21), the highest temperature varied from 102° to 100·4°. From this summary it would appear that three-fifths of these cases of acute rheumatism without pericarditis, endocarditis being absent or doubtful, in which the nervous system was seriously affected, had hyperpyrexia.

Pericarditis was absent and endocarditis was absent or doubtful, as we have just seen, in twenty-one cases of acute rheumatism in which there was affection of the nervous system and the temperature was ascertained. A^3 In six of those cases delirium gave place to coma, and in one of these the delirium reappeared; A^4 in two delirium passed into somnolence. B^1 Delirium without coma was present in ten cases. Two patients had very high temperature without delirium, one of whom was restless and talked when asleep, and the other had vomiting and dyspnœa; and in one there was twitching of the limbs and body without delirium, the temperature not rising above 102°.

A^3 The whole of the six cases in which delirium passed into coma were fatal. The delirium was present in these patients when the temperature varied from 102·2° to 108·4°, and coma replaced the delirium in five of them at a temperature ranging respectively from 108° to 110°. The highest temperatures observed in these cases towards or at the time of death was from 109·5° to 111·1°. In a case in which delirium gave place to coma and that again to delirium, the temperature about the period of coma was 104°, but eight hours before death it was 105.8°.[1]

The delirium was violent or active in four of these six patients, three of whom got out of bed or tried to do so; and in two of them it was muttering or quiet.

The duration of the delirium varied much in these cases. In one patient the delirium continued for four days, in another two; in one it lasted four hours, and in another, the most interesting of the series, after it was slight for one day, it became muttering for half an hour. The duration of the coma was more constant. It lasted for from an hour to an hour and a half in four cases, and in one for four hours, while in one there was alternate delirium and coma for two days.

A^4 In two cases delirium passed into drowsiness. One of these, a dull, corpulent woman, aged 32, was strange in manner (temp. 103·4°) on the eighth day after admission, and had low muttering delirium. At 2 A.M. on the following night

[1] Dr. Meding, Archiv der Heilkunde, xi. 467.

[1] Lebert, "Klinik des acutens Gelenkrheumatismus," p. 55.

(temp. 105·3°) she awoke restless; and at 5 A. M. (temp. 106°) she was dull and somnolent. She was put for twenty minutes into a bath at 90° to 81°. When in the bath she felt comfortable, but at length she complained of cold (temp. 102°). After this her temperature never rose above 104·7°, she had bronchitis and pneumonia for some days, and finally recovered.

I was favored by Dr. Murchison with notes of the other case of this class. A lady, aged 35, stout, was attacked with acute rheumatism. At the end of ten days her joints were better, but she became sleepless and delirious. Opium, chloral, and bromide of potassium only made her worse. Her pulse was 108, weak; temp. 102·5°. She gave no signs of peri- or endo-carditis, and had headache. The following is the report of her case ten days later: "The temperature has been as high as 106°, but is now only 101°. She is heavy and drowsy, but has been very noisy and delirious. Respiration is quick and irregular—cerebral. She swallows well. Pulse 64. Heart seems still sound. Urine is made in bed. There are bed-sores, and she has some pains in the joints." She died next day.

B[1] There was delirium without coma in ten cases. In three of these the temperature was very high, being 110° in a fatal case; and 108·2° and 107° respectively in two that recovered after the use of the cooling bath; in one of these the temperature, rarely above 104·6°, once rose to 105°, and this case died in spite of the repeated use of the bath; while the remaining six cases had the comparatively low maximum temperatures respectively of 104·5°, 103·4°, 101·2°, 101·1°, 101·1°, and 100·4°; and of these the first case (temp. 104·5°) and that in which the temperature was the lowest (100·4°), a case with Bright's disease, died, while the four others recovered.

The duration of the delirium was very various in different cases, having ended in death in one instance in two hours and a half, and being prolonged with interruptions in another for twenty-nine days, the high temperature being kept down and lowered and the delirium from time to time suspended by the cooling effects of a succession of twenty baths.

As I have just said, in two of the three cases with delirium and very high temperature, the temperature was kept in check by the cooling bath. One of these cases, a youth, on the morning of the fourth day of treatment, muttered to himself but could be roused, temp. 107·8°, and at 7.45 temp. 108·2°. After being half an hour in a bath at 76°, his temperature was 101°, and half an hour later 98·8°, when he fell asleep, and awoke in a perfectly conscious state. In the evening, a second bath again lowered the temperature from 105·8° to 98°, when he perspired freely and slept. After this the temperature never rose above 99·8°, and he recovered.[1] The second case, a woman, with a temperature of 107°, was put into a bath at 90° cooled to 42°. Her temperature was lowered to 97·5°, and her mind became clear.[2]

One patient, a man, who had been a free liver, presented throughout from time to time profuse sweating, variable delirium, tremor of hands, subsultus, and twitchings of the face, and a temperature varying from 104·4° to 106·4°. The use of the cooling bath invariably lowered the temperature, restored the patient from a state of delirium to one of consciousness, and caused a subsidence of the other nerve-symptoms, tremor, subsultus, and facial spasms. This condition lasted for twenty-nine days, during which time twenty baths were employed, five of them in one day for a combined period of over five hours, and the patient finally died, the temperature at the instant of death being 104·2°.[3]

Among the six cases with delirium in which the temperature was not very high, varying from 104·5° to 100·4°, two died and four recovered.

One of these cases, with a temperature of 103·5°, was a great beer-drinker. His hands were tremulous, he wandered during the day, was very noisy towards the evening, when he screamed out much, continued in a state of variable delirium for fourteen days, and finally recovered.[4]

The highest temperature observed in the four remaining cases with delirium was 101·4° and 100·4° respectively. Two of them had albumen in the urine, and the other one had obstinate diarrhœa, and was delirious when the diarrhœa was checked.

There were three cases of hyperpyrexia in which there was no delirium. One of these was a man whose temperature rose to 106·3°. He had previously been deaf and very restless. Under the influence of a cooling bath his temperature fell to 101·8,° and later to 99·8°. After the bath his deafness left him, and he did well.[5] Another case, a woman aged 24, was suddenly seized with dyspnœa and vomiting, which continued until death; a short time before which event her temperature was 110·8°.[6]

Convulsive Choreiform and Tetaniform Movements.—Twitchings were present in

[1] Dr. Weber, Clin. Soc. Trans. v. 186.
[2] Sir William Gull, Lancet, 1872, ii. 562.
[3] Dr. Greenhow, Clin. Soc. Trans. vi. 7.
[4] Dr. Johnson, Lancet, 1867, i.
[5] Dr. H. Thompson, Medical Times and Gazette, 1873, i. 269.
[6] Dr. Ogle, Lancet, 1876, ii. 154.

four of the twenty-one cases that form this group, in which there was no pericarditis and endocarditis was absent or doubtful. The twitchings affected the body in one instance, the limbs and features in another, the muscles of the face for a long period in another, whenever the temperature rose; and in a fourth, the features occasionally twitched with a sardonic grin. In one case the patient was restless and moved his arms about; but, perhaps with this exception, there were no notable choreiform or tetaniform movements in any of the cases. In two cases there was tremor—in one, of the trunk and limbs, in the other, of the hands.

Twitching movements were present in four of the twenty-six cases with pericarditis, in one of the eleven cases with simple endocarditis, and as we have just seen, in four of the twenty-one cases in which there was no pericarditis and endocarditis was absent or doubtful. Twitching movements were therefore distributed in nearly equal proportion in those three groups of cases, and were therefore not due to pericarditis. Twitchings were present in eight cases with hyperpyrexia, and it is therefore probable that they were associated with the very high temperature. This is borne out by a case of Dr. Greenhow's, in which twitchings came on, and were again and again renewed when the temperature became very high, and were again and again almost or quite suspended by the cooling bath.

In the remaining case with twitchings, a man who was under my care, the temperature was never above 102°. On the fifth day, temp. 100·2°, he had muscular twitchings all over the body, which continued for several days, and reappeared on the twenty-eighth day. There was albumen in the urine on both occasions when the twitchings were present. His recovery was slow.

There were choreiform or tetaniform symptoms — or both — in seven of the twenty-four cases with pericarditis, but in only one of the eleven cases with simple endocarditis, and in none of the twenty cases in which there was no pericarditis, endocarditis being absent or doubtful. The question how far the choreiform and tetaniform movements observed in these cases was connected with pericarditis will be considered when we review the larger series of cases of acute rheumatism with and without pericarditis in which the temperature was not observed.

The *affection of the joints* during the early period of the disease was severe in ten, and moderately so in five of the twenty-one cases in which there was no pericarditis and endocarditis was absent or doubtful. The affection of the joints was more severe before than during the delirium or other affection of the nervous system, in all but three cases, in which the joint-affection was equally severe during the two periods. In two of these three exceptional cases the temperature never rose above 102°, in one of these the delirium was only present during the night, and in the other there was no delirium, but twitchings were present for a short time during the early days of the illness.

Perspiration.—The state of the skin is described in one-half of the cases belonging to this group (10 in 21), and all of these had profuse perspiration before the nervous system became affected. In seven of those cases there was either no perspiration, or it was much lessened during the period of delirium. In three cases, perspiration was equally copious during the two periods. In three of these cases the skin, which had been perspiring profusely before the excessive rise of temperature, and the occurrence of delirium, was hot and dry when the temperature was 110° to 111·1°; coma was present, and death approached. These clinical facts correspond with those which, as we have already seen, occurred in the analogous cases affected with pericarditis. One of the cases in which there was no affection of the heart was observed by Mr. Anderson night and day. This patient, of a nervous, excitable temperament, a laborer, aged 29, had a hot, dry skin, and rambled during the night for four succeeding nights; but during the three intervening days his skin was covered with a profuse acid perspiration, and his mind was unaffected. On the morning of the fourth day his manner was wild and excitable, not unlike that of a patient in the early stage of delirium tremens, and his skin was hot and dry, and covered with a miliary eruption. After a bath, he sprang out of bed, ran into the grounds, and struggled violently. His temperature at that time was 107°, and later in the evening, ten minutes before his death, it was 110·3°.

Dr. Greenhow's case, already referred to at page 518, offers a contrast in some respects, but not in others, to Mr. Anderson's case. In this man, perspiration was absent with delirium at a temperature of 104·8°, and was absent without delirium after the bath at 100·2°, and was present afterwards with obscured intellect and intermediate temperatures.

Perspiration, which is not present at ordinary temperatures, is indeed an index of the internal production of great heat, and a safety-valve for carrying away a large portion of that heat. When perspiration takes place from an exposed skin in a dry air—in motion—its evaporation

tends to keep down the heat. In these patients, however, lying, as they do, in their own perspiration, covered by bedclothes, in a still air saturated with moisture, evaporation can do comparatively little towards cooling the body.

We must look, then, to some other influence than evaporation to account for the cooling effect of perspiration in acute rheumatism. Such an influence we find in the welling out of hot liquid from every part of the body—a liquid charged with a portion of the surplus heat generated by the rapid combustion or disintegration of the internal structures in that disease. It is self-evident, that if the temperature of the body be 103° or 104°, the fluid poured out from the body must likewise have a temperature of 103° or 104°, and that this fluid during its steady universal expulsion must carry away with it a corresponding proportion of the heat generated within, and so tend to keep down the temperature of the whole of the structures that compose the body.

If, on the other hand, the skin is dry, the chemical heat generated in the rapidly-changing tissues tends not to escape, and may be stored up in accumulating quantities in the blood and the tissues, with the effect of producing an excessively high temperature, or "hyperpyrexia."

Respiration.—I have not made an analysis of the rate of respiration in cases of acute rheumatism with affection of the nervous system, with and without high temperature. One well-observed and well-treated case of hyperpyrexia is sufficient for our present purpose, which is to illustrate the influence of an excessively high temperature of the body on the one hand, and of the cooling of that body on the other, on the frequency of respiration. In Dr. Wilson Fox's case, already related at page 516, when the temperature of the body was 107°, the patient was delirious, and the respiration was 45 in the minute, but when the patient's body had been cooled down by the bath to 98°, the mind was clear, and the respiration was 20 in the minute.

It is evident, therefore, that during hyperpyrexia, the cooling effect of respiration is stimulated to its highest degree by the excessive heat of the body, but that this cooling effect is quite inadequate to keep down the temperature of the body below that of hyperpyrexia.

There were some conditions common to the three sets of cases—those namely with: 1, pericarditis; 2, simple endocarditis; 3, without pericarditis, endocarditis being absent or doubtful—and I shall now briefly notice those conditions.

Restlessness affected a considerable proportion of the patients before the occurrence of delirium. Six of the twenty-seven cases with pericarditis; three of the thirteen cases with simple endocarditis; and ten of the twenty-one cases that had no pericarditis, endocarditis being absent or doubtful, were thus affected with restlessness.

A *miliary eruption* or *sudamina* appeared in a considerable number of the cases, being noticed in twelve of the twenty-seven cases with pericarditis; in three of the thirteen cases with simple endocarditis; and in eight of the twenty-one cases in which there was no pericarditis and endocarditis was either absent or doubtful.

An *abundant secretion of urine* took place in a few of the cases, at the time of the great rise in temperature. The urine was very abundant under those circumstances in three of the twenty-seven cases with pericarditis; in three of the thirteen cases with simple endocarditis; and in two of the twenty-one cases in which there was no pericarditis, endocarditis being either absent or doubtful.

Diarrhœa, sometimes profuse and offensive, was present when the temperature was very high in seven of the twenty-seven cases with pericarditis; in four of the thirteen cases with simple endocarditis; and in five of the twenty-one cases in which there was no pericarditis, and endocarditis was either absent or doubtful.

Excessively high temperature or "hyperpyrexia" in acute rheumatism with and without pericarditis. We have just seen that in sixty-one cases of acute rheumatism in which the temperature of the patient was observed, the nervous system was affected, and we shall now inquire how many of them presented an excessively high temperature, and what was the influence of pericarditis in those cases of hyperpyrexia. The temperature was excessively high, ranging from 106·8° to 111·1° in thirty-one of those sixty-one cases, and was arrested during its rise when it was at from 105° to 106·3° by the use of the cooling bath, or cold externally, in six cases. In three of those six cases, the tendency of the temperature to rise was great, but in three of them it was not so. The temperature was not observed during the period of coma or the last hours of life in three fatal cases in which the temperature was 104·6°, 104·8°, and 105·8° respectively at the time of the last observation, and I consider that these three cases and three of the six in which the high temperature was kept in check by the bath, ought to be added to the thirty-one cases in which the temperature was very high, thus bringing up the number of those with "hyperpyrexia," to thirty-seven of the total number of sixty-one

cases. Thus estimated, we find that seventeen of the twenty-seven cases with pericarditis, nine of the thirteen with simple endocarditis, and eleven of the twenty-one without pericarditis, endocarditis being absent or doubtful, either had, or were threatened with "hyperpyrexia." Among these thirty-seven cases of hyperpyrexia, one had coma without delirium, twenty-one, delirium followed by coma, or, in one instance, stupor, two, delirium with convulsive movements, ten, uncomplicated delirium, and three had neither coma nor delirium.

The case of simple coma, and all but one of the twenty-one cases in which delirium passed into coma, were affected with actual (18) or threatened (3) hyperpyrexia. The temperature observed soon rose above 106° in three cases with delirium and stupor, but in one of these it was kept down and lowered by the cooling bath, while in both the cases which ended fatally with convulsive movements, the temperature was very high. Of the twenty-four cases with uncomplicated delirium, only two-fifths had hyperpyrexia (10 in 26).

Coma preceded by delirium is, as we have just seen, the typical effect of rheumatic hyperpyrexia, and one-half of those with hyperpyrexia and coma preceded by delirium, had pericarditis (10 in 20). From these clinical facts it would appear that hyperpyrexia attacked cases of acute rheumatism almost as frequently when they had pericarditis, as when they were not so affected (17 in 27 with pericarditis, 20 in 37 without pericarditis). When we consider that pericarditis usually attacks only one in every five or six cases of acute rheumatism, we must multiply the cases of pericarditis with hyperpyrexia by five or six if we would make a parallel comparison between those cases with pericarditis and those without it. It would appear from this that the presence of pericarditis in a case of acute rheumatism increases the chance of the occurrence of hyperpyrexia with delirium and coma, in the proportion of four or five to one. An important case successfully treated by Dr. Wilson Fox by the cold bath had pericarditis in its worst form. The dulness or percussion over the region of the pericardium filled the whole left front of the chest from apex to base. In that case the tendency to the renewed excessive rise of temperature after it had been brought down again and again, by the cold bath, the ice-bag, or the wet-pack, continued until the seventh day; when the pericardial dulness fell to the first rib mid-sternum, and the tendency to the increase of temperature lessened. It is a clinical fact that here the renewed rise of temperature continued so long as the pericarditis was severe, and gave way when the pericarditis gave way, and it is probable that the continued severity of the pericarditis had an influence in keeping up the tendency to the rise of temperature. It must, however, not be lost sight of that as a rule, cases of acute rheumatism with pericarditis are in all respects worse than those without it, and that, not only at the time of the pericardial inflammation, but usually also before it. It becomes therefore a question whether or not the same severity of the acute rheumatism itself that brought the pericarditis into existence brought also the excessively high temperature with its attendant delirium and coma into existence, the two affections being affiliated, and due to a common cause.

The occurrence of a high temperature of the body in cases of acute rheumatism, corresponds in essential features with the high temperature observed in sunstroke, in certain exceptional cases of tetanus, and in injuries to the cervical portion of the spinal marrow. In sunstroke the temperature varies from 112° to 105·5°, the skin is hot and dry, coma supervenes, preceded occasionally by delirium, and death tends to ensue unless the temperature of the body is lowered by cold.[1]

The temperature in tetanus, though variable, does not as a rule rise to a very great height. Wünderlich, however, gives a case in which it attained to 44·75° C. (112·55° F.) before death.[2] This instance resembled in all its main features the cases of hyperpyrexia in acute rheumatism. The patient, before the time of the fatal rise of heat, was very restless; had profuse perspiration and an abundant miliary rash; then came on delirium, night trembling, contracted pupils, and death. Wünderlich, without giving any reason for it, gives the name of rheumatic tetanus to another but less extreme case of the same kind.

Injury of the spinal cord from the fourth to the sixth cervical vertebra from fracture or caries of the spinal column has induced an excessively high temperature in several cases published since the first observation to that effect by Sir Benjamin Brodie.[3] The symptoms in these cases closely resemble those of hyperpyrexia in acute rheumatism, but in only one of them was it stated that the final and fatal coma was preceded by delirium. One of Dr. Hermann Weber's two cases was a

[1] Dr. Levick, "Pennsylvania Hospital Reports," 1868, p. 371; Dr. Gee, Gulstonian Lectures on Pyrexia, Brit. Med. Journal, 1871, i. 302; Dr. Maclean on Sunstroke, Reynolds' "System of Medicine," i. 661.

[2] Wünderlich, Archiv der Heilkunde, ii.

[3] Sir Benjamin Brodie, Med.-Chir. Trans. xx. 118; Reineke, Berliner Klinische Wörterbuch, 1869, 113, 301; Billroth, Archiv für Klin. Chirurgie Langenbeck, ii. 482.

youth, who could walk supported, but like a drunken man, and could move his arms twenty minutes after the accident, which caused fracture and incomplete dislocation of the third, fourth, and fifth cervical vertebræ. He voided urine frequently and in great quantities. An hour after admission his temperature was 100·4°. Two hours and a-half after the accident he passed liquid motions unconsciously, had occasional convulsive twitches in the arms and legs, his skin was slightly moist and hot, and his temperature was 109·58°. Four and a-half hours after the accident there was complete coma, and his temperature was 111°, and it was 111·2° at the time of death, eight hours after the accident.[1]

Dr. Burdon Sanderson, who has favored me with the use of the manuscript notes of his lectures delivered at Manchester, gives an account of experiments made by him in which the cervical portion of the spinal cord was injured. He found that there was no increase of temperature for two hours after the injury to the cord, but that at the end of that time it began to rise and to rise rapidly, attaining a very great elevation, 42° C. or 107·6° F., or higher than that of fever. Dr. Burdon Sanderson considers that this experiment shows conclusively that the process of which the higher temperature is the outcome, must consist in a gradual modification of those processes on which heat production depends, must have as wide a localization as they, and cannot therefore be attributed to any sudden interruption of the relation between the centre and the periphery of the nervous system. These experiments correspond remarkably with Dr. Hermann Weber's case just reported.

Cases in which the temperature of the body was below that of hyperpyrexia.—We have just seen that of the sixty-one cases of acute rheumatism associated with affection of the nervous system in which the temperature was observed, in thirty-seven there was actual (31) or threatened (6) "hyperpyrexia." In the remaining twenty-four cases, the maximum temperature of the body observed in the different instances varied from 99·3° to 106·3° temp. Ten of the twenty-seven cases with pericarditis, four of the thirteen with simple endocarditis, and ten of the twenty-one without pericarditis, and in which endocarditis was absent or doubtful, belong to this group in which the temperature was not excessively high. In twelve of these twenty-one cases, the maximum temperature varied in the different cases from 103° to 106°, and in nine of them from 99·3° to 102·8°.

A considerable proportion of those who were attacked with delirium at comparatively low temperatures were either habitual drinkers, or of a nervous temperament, or had been subject to anxieties and privation, or to lowering diseases, or had received injuries affecting the nervous system, and in several of those cases the affection was closely allied to delirium tremens; several such cases occurred among those affected with pericarditis. This was so in Dr. Southey's two cases with pericarditis, referred to at page 516, in Dr. Greenhow's case, given at page 517, in Dr. Murchison's two patients, quoted at page 521, and in a patient of my own.

To these must be added the case with which Dr. Southey favored me since the above was written, given at page 516, and two of Dr. Andrew's cases.

Most of these cases had pericarditis.

Cases of Acute Rheumatism with Affections of the Nervous System in which the Temperature of the Body was *not* observed.

There were 119 cases of acute rheumatism with affections of the nervous system in which the temperature was not observed. Of these 65 had pericarditis; 16 had simple endocarditis; and 38 were free from pericarditis, endocarditis being absent or doubtful.[1]

[1] Dr. Hermann Weber, Clin. Soc. Trans. i. 163.

[1 Several tables under this heading and under Pericarditis have been omitted, as they are too complicated in their arrangement to be followed by the ordinary reader, and as the results deducible from them are fully stated in the text.—H.]

Summary of cases of Acute Rheumatism, with Affections of the Nervous System, in which the temperature was not observed.

	With Pericarditis.	Simple Endocarditis.	No Peric. Endocard. absent or doubtful.	Total.
A^1—Coma with delirium or convulsions	3	0	2	5
A^2— " with convulsions	2	1	2	5
A^3— " preceded by delirium	5	0	8	13
A^4—Stupor preceded by delirium	0	0	2	2
A—Had Coma or Stupor. Total	**10**	**1**	**14**	**25**
Had Semi-consciousness	1	.....	.....	1
B^1—Delirium, uncomplicated	21	8	14	42
B^2 " passing into temporary insanity	*11*	*5*	*3*	*20*
" with chorea or choreiform or tetaniform symptoms	*5*	*0*	*2*	*7*
" Total	16	5	5	27
B^3—Delirium, with chorea and choreiform movements, not including those with temporary insanity	4	*0*	1	5
B^4—Delirium with tetaniform movements, not including those with temporary insanity	2	*0*	1	3
B—Delirium without Coma. Total	**43**	**13**	**21**	**77**
Delirium. Totals (including those with coma)	**48**	**13**	**31**	**92**
C^1—Chorea without delirium	7	.....	.....	7
C^2—Choreiform movements (jactitation), without delirium	1	1	.....	2
C^3—Tetaniform symptoms, without delirium	2	.....	.....	2
D—Had slight fit	1	.....	.....	1
E—Had embolism, hemiplegia	.....	1	.....	1
F—Had Paraplegia	.....	.....	2	2
G—Had agitation and prostration	.....	.....	1	1
Total	**65**	**16**	**38**	**119**

I.—Cases affected with Pericarditis.

There were sixty-five cases of acute rheumatism with pericarditis, in which the nervous system was affected. (*A.*) Ten of these had coma, of which (A^1,) three had uncomplicated coma; (A^2,) two had coma with convulsions; and (A^3,) in five the coma was preceded by delirium. (*B.*) Forty-three cases had delirium without coma; of these (B^1,) twenty-one had uncomplicated delirium, one of which had "symptoms of inflammation of the brain," and one apparently had pyæmia; (B^2,) sixteen, of which five had choreal or tetaniform symptoms, had temporary insanity, lasting from two weeks to three months, or in three instances, insanity was cut short by death; (B^3,) four had chorea or choreiform movements, and (B^4,) two had tetaniform symptoms without temporary insanity. (C^1.) Eight of the cases had chorea or choreiform movements, and (C^2,) two had tetaniform symptoms without delirium. (*D.*) One of them had a slight fit with ptosis.

II.—Cases affected with Simple Endocarditis.

There were sixteen cases of acute rheumatism with simple endocarditis, in which the nervous system was affected, excluding cases of ordinary chorea, but including cases of chorea rapidly fatal, or with delirium.

(A^2.) One of these cases had coma with convulsions, associated with acute Bright's disease from embolism. (A^3.) One had delirium ending in coma, with embolism of the minute cerebral arteries. (B.) Twelve of them had delirium without coma, of these (B^1,) seven had uncomplicated delirium; and (B^2,) five passed into a state of temporary insanity, lasting from three weeks to four months. (C^1.) One had chorea ending rapidly in death. (E.) One had embolism with hemiplegia.

III.—Cases in which there was no Pericarditis and Endocarditis was absent or doubtful.

There were thirty-eight cases of acute rheumatism without pericarditis, endocarditis being absent or doubtful, in which the nervous system was affected, exclusive of cases of ordinary chorea. (A.) Twelve of these had coma or stupor, of which, (A^1,) two had uncomplicated coma, (A^2,) two had coma with convulsions, (A^3,) in eight the coma was preceded by delirium; and there were also (A^4,) two cases in which delirium passed into stupor. (B.) Twenty of the cases had delirium including two with "cerebral rheumatism," and one that had pus in the pia mater; of these (B^1,) fourteen had uncomplicated delirium; (B^2,) five passed into temporary insanity, two of which had chorea also; (B^3,) one had chorea; and (B^4,) one had tetanic spasms. (F.) Two of the cases had paraplegia. (G.) One of the cases had agitation and prostration ending in rapid death.

A.—Coma.

I.—*Cases with Pericarditis.* A^1.—*Uncomplicated Coma.*—Three cases with Pericarditis had coma without convulsions or delirium, all of which proved fatal.

A^2.—*Coma with Convulsions.*—In the two cases of coma with convulsions, death was speedy.

A^3.—*Coma preceded by Delirium.*—Four of the five cases in which delirium passed into coma, died; and one recovered. One of the cases passed rather into stupor than coma. The duration of the coma in these cases was variable and uncertain, and that of the delirium lasted for from one or two nights to nine or ten days.

II. — *Cases with Simple Endocarditis.* A^2.—*Coma with Convulsions.*—One fatal case of coma preceded by convulsions had simple endocarditis with embolism of the spleen and kidneys, the coma and convulsions being evidently associated with acute Bright's disease.[1]

III.—*Cases without Pericarditis, Endocarditis being absent or doubtful.* $A^{1,2}$.—*Coma without and with Convulsions.*—There were four fatal cases of coma without delirium among the cases without pericarditis, endocarditis being absent or doubtful, two of them having convulsions. In three of them death was very rapid, and in one coma, coming on after convulsions, lasted for twelve hours before death.

These cases did not differ materially in character and history from those with coma and pericarditis.

A^3.—*Coma and Delirium.*—Coma was preceded by delirium in eight fatal cases that presented no sign of affection of the heart.

The delirium was more frequent by night than by day, being present in three of the cases from two to five or six nights, while it was absent in the daytime, and it lasted in the other five cases from two to four or five days.

The coma, as a rule, soon ended in death. In one-half of the cases, or four, the delirium became violent, and in the other half, its character was not described.

These cases do not differ materially in essential character from those with pericarditis that were affected with delirium and coma. There were, however, nervous symptoms in the form of agitation, twitchings, and choreiform and tetaniform movements in a much greater proportion of those with pericarditis than of those not so affected.

A^4.—*Delirium and Stupor.*—One of the two cases in which delirium preceded stupor recovered after the employment of the wet sheet, and the other died.

B.—Delirium.

B^1.—*Uncomplicated Delirium.*—1. *Cases with Pericarditis.*—Twenty-one of the sixty-five cases with rheumatic pericarditis had uncomplicated delirium, including one with "symptoms of inflammation of the brain," and one with probable pyæmia. Eleven of these cases died and ten recovered.

The duration of the delirium varied much. The delirium was more active by night than by day, and in five cases was present from one to three or four nights, but was absent during the day. In the rest of the cases it lasted from for a few hours to four or five days. The delirium was noisy or violent in eleven instances, moderate in four, and slight in five cases.

One case, a female servant, felt much better at the evening visit, but a quarter of an hour later became delirious, with loud continuous cries. A varied treatment, including wet packing, was employed, and she recovered.

Another case, a workman in Messrs. Guinness's Brewery, drank largely of their XX porter besides whiskey. He had pericarditis, and "delirium tremens," and recovered after taking opium.[1]

2. *Cases with Simple Endocarditis.*—Seven of the sixteen cases with simple

[1] Frerichs, "On the Diseases of the Liver," New Sydenham Soc. Edition, vol. i. p. 164.

[1] Dr. Graves, "Clinical Lectures on the Practice of Medicine," vol. i. p. 531.

rheumatic endocarditis had uncomplicated delirium. Three of these cases died and four recovered.

The duration of the delirium varied from, for a single night in one patient, to at least nine days in another. It was present more often, and, as a rule, with greater violence by night than by day. In four of the cases the delirium was active or violent, in one the delirium was wandering, and in another, it was accompanied by somnambulism.

One of these cases was observed by Dr. Boisragon and Mr. Tudor, and reported by Dr. Davis, and is, so far as I have discovered, the first case in which endocarditis was well described.

Three of the cases of endocarditis with delirium were under my own care, and of these, one died and two recovered.

3. *Cases without Pericarditis, Endocarditis being absent or doubtful.*—Fourteen of the thirty-eight cases without pericarditis, endocarditis being absent or doubtful, had uncomplicated delirium. Ten of the fourteen cases died, and four recovered.

The duration of the delirium varied much in the different cases. It prevailed more during the night than the day. In three instances it was only present during the night for from one to three nights. In one case the delirium was only present for a quarter of an hour before death, in four cases it existed for one day, and in four others from two to five or six days. The delirium was violent or lively in five of the cases, and five were simply "delirious."

These cases corresponded in essential features with those that had delirium with pericarditis.

"*Hyperpyrexia*" *in cases of acute rheumatism without and with Pericarditis in which the temperature was not observed.*—The ten fatal cases belonging to the last group of fourteen with delirium, the twelve with coma and the two with stupor, all of which had neither pericarditis nor endocarditis, evidently belong to the important group of cases of acute rheumatism with hyperpyrexia. All of those twenty-four cases except one with stupor, died, and that patient recovered after the external use of the wet sheet. The ten cases with coma, and the eleven fatal cases that recovered under the use of wet packing that had uncomplicated delirium among the patients with pericarditis, and three fatal cases of delirium with simple endocarditis, may also be ranked among the cases of hyperpyrexia. According to this estimate, twenty-two of the sixty-five cases with pericarditis, three of the sixteen with simple endocarditis, and twenty-four of the thirty-eight without pericarditis or notable endocarditis, in which the temperature was not observed, were affected with "hyperpyrexia."

These forty-nine cases corresponded in their broad features as regards coma, delirium, and death with those cases of "hyperpyrexia" in which the temperature was observed. As in those also so in these, in the few cases where these conditions were observed, the affection of the joints ceased when the delirium appeared, and the perspiration, copious during the earlier stages, was absent or much lessened during the stage of delirium or coma, when the skin was usually dry and hot.

Convulsive, choreiform, and tetaniform movements in the cases with hyperpyrexia.—There was an important difference in the two sets of cases with and without pericarditis, as regards the presence of convulsive, choreiform, and tetaniform symptoms in combination with the far more important symptoms of "hyperpyrexia."

Convulsive movements, jactitation, agitation, choreiform movements without actual chorea, and tetaniform symptoms appeared more frequently in the cases of coma or delirium with pericarditis, than in those without pericarditis. Involuntary movements of the muscles occurred in one, and general agitation in three of the twenty-five cases of hyperpyrexia that had neither pericarditis nor notable endocarditis. A convulsive fit occurred in one, jactitation of the limbs or body in two, tetaniform symptoms in two, and great general agitation in three, of the twenty-two cases with hyperpyrexia that had pericarditis. Besides these eight instances of convulsive, choreiform, or tetaniform affections among the fatal cases of coma and delirium with pericarditis, there were two with jactitation, and one with twitchings of the muscles of the face, among the cases of delirium with pericarditis that were not fatal. Four of the eleven cases with pericarditis thus affected with convulsive, choreiform, or tetaniform movements had endocarditis, three had no endocarditis, and in four the presence of endocarditis was doubtful.

We have already seen that among the cases of "hyperpyrexia," in which the temperature was observed, choreal and tetaniform symptoms occurred much more frequently among those with, than among those without, pericarditis; while on the other hand twitching movements were as frequent among those without, as among those with, pericarditis.

Delirium resembling Delirium Tremens.—Among the cases of delirium in acute rheumatism without pericarditis or evident endocarditis as among those previously analyzed with pericarditis, there are several that present symptoms partly allying them to delirium tremens—partly to the delirium of rheumatic hyperpy-

rexia, and that are associated with previous habits of drinking, or with some affection of the nervous system. One of these patients, a hard drinker, complained of being unable to see, called out "thief," rushed out of bed and fell down. After this he struggled with two attendants, and then dropping back, died. All this took place in less than a quarter of an hour.[1]

Two of my own patients belong to this class, one of whom recovered, the other died. One was a stout florid waiter, aged 40, who perspired profusely, slept but little, and became very violent. On the seventh night he slept with an opiate. He recovered rapidly.

The patient who died was a barman, aged 23, who was rather restless, and hurried in speech on the day after admission, became more restless on the third day, and died suddenly.

B². —Temporary Insanity with Taciturn Melancholy and Hallucinations.

B². I.—*Cases with Pericarditis.*—The series of cases that I have now to consider present a remarkable succession of symptoms. In eleven cases of acute rheumatism with pericarditis, delirium, usually desponding and taciturn, often with hallucinations, came on when the heart was inflamed; but instead of passing away quickly, this sombre delirium lasted for from two or three weeks to three months. Of these eleven cases of temporary insanity, ten recovered, and one died; eight of those cases were females, six below the age of twenty, and three were males. All but one of these patients were affected with endocarditis as well as pericarditis.

The duration of the insanity varied considerably, and the return to a healthy state of mind was gradual, and never sharp. The temporary insanity lasted for above a fortnight in three cases; for about a month in three; for two months in one; for ten weeks in one patient, whose mind was not yet clear at the end of that time; and one died with her intellect still confused at the end of two months.

The prevailing feature of the delirium was a state of taciturn melancholy. Only one patient, a young woman, the fatal case, was at times in wild delirium, at times taciturn and almost idiotic, and at times quite rational.[2] Eight of the patients were taciturn, and two others were confused in mind or speech. Four of them had hallucinations; one saw her mother at her side; one a knife and poison; one was followed and insulted, and then reached out his hand as to an old friend; and one complained of vermin. Another patient had delusions.

Two of my patients belong to this series of cases. One of these, a potman, aged 21, on the seventeenth night after his admission was in a state of partial stupor and delirium. On the following day he answered no questions, and as he would not take food, stimulants were given by enemata. On the twenty-sixth day he again took food, but he continued to be taciturn. On the thirty-ninth day he recovered the powers of nature, was up on the forty-seventh, and left on the seventy-fifth day, his heart-sounds being healthy.

The other case, a laborer, with endopericarditis, had a vacant, torpid, and wandering mind for three weeks, which followed an attack of hemiplegia from embolism affecting the right side, with loss of speech, which was apparently a mixture of aphasia and a taciturn character of mind. On the tenth day his face was paralyzed on the right side, and the pupils were irregular. On the thirteenth he would not or could not speak, but muttered slightly, and tried to get out of bed. He improved daily and his speech returned, but his expressions were incoherent. On the thirty-eighth day he had more command over his articulation, and on the forty-second had almost regained the use of his right side. He improved steadily, and on the seventy-second day he went out well, the heart-sounds being healthy.

Besides the eleven cases just spoken of with temporary insanity of a taciturn melancholic type, there were five others in which a similar condition was associated with chorea or choreiform movements (in 4) or with tetaniform symptoms (in 1). Three of these cases were fatal, and two of them recovered. The whole of the five cases were below the age of twenty-one, and two of them were male and three were female patients. All of them had endocarditis as well as pericarditis. The affection lasted in one of the two that recovered about a month, and in the other for a shorter period. The three fatal cases died respectively in about twenty-three, sixteen, and nine days after the beginning of the mental trouble. One of those patients was taciturn, then delirious, and finally had the most violent choreiform movements, ending in death. Another fatal case had difficult utterance, incoherence, tossing of the head from side to side, and choreiform spasms which put on the character of the most violent convulsions. A third case spoke loud and low, after, in succession, being excited and stubborn, weeping, seeing a dead

[1] Trousseau, "Lectures on Clinical Medicine" (New Sydenham Soc.), vol. i. p. 513.

[2] Sir Thomas Watson, loc. cit. ii. 307.

man, and grimacing as in chorea: death took place an hour after an attack of universal convulsions. One of the two cases that recovered had a rather childish appearance; her answers were sometimes irrelevant, sometimes rational, and she had choreal movements of the right arm and leg.

The last case had delirium with tetanic spasms; at first he had an excited manner, with wild rolling of his eyes, then furious delirium, followed by firm clenching of the hands, sleep, and a more tranquil state. After this he was idiotic and violent by turns, until the twenty-eighth day after the first disturbance of the mind, when he became tranquil.

These sixteen cases with taciturn melancholy, often with hallucinations, lasting for from three weeks to three months, and then usually getting well, present a group of conditions that seem to separate them from the twenty-one cases of delirium that were not followed by coma, and the five that were so. In those cases the delirium was often violent, generally active, sometimes muttering; in these it was melancholic and taciturn. In those cases the delirium was often exclusively by night and was then almost always most noisy; in these it was present day and night, though it was usually more active by night. In those cases the delirium lasted for from one or more hours to five or six nights and five days; in these it lasted for from three weeks to three months. In those cases perspiration was generally profuse before the appearance of the delirium, the skin usually becoming hot and dry as the temperature rose to the fatal height; in these perspiration was only noted as being profuse in two cases, and slight in one. In those cases death was the natural result; in these, all but one of the eleven without chorea recovered, while three of the five with chorea died. In those endocarditis was absent in three-fifths of the cases (11 in 32) with coma and delirium, but three more of those cases probably had endocarditis; in these endocarditis was present in all but one.

We saw that in the two sets of cases, in one of which the temperature was, and in the other, was not observed, delirium presented itself in two forms: (1) one, and the leading form, of delirium with hyperpyrexia, ending in death; (2) the other, the secondary form, with a less high temperature in which a condition resembling delirium tremens associated itself with and modified the delirium of hyperpyrexia, often occurring in persons who had been intemperate, anxious, nervous, or in want, and ending generally in recovery.

In these cases of temporary insanity with taciturn melancholy we have clinical evidence of a third kind of delirium, differing from the two other kinds of which we have just spoken.

These cases resemble in some of their symptoms, cases of insanity with settled taciturn melancholy; but from those they differ in this essential point, that while in those the insanity is obstinate, often indeed for life; in these the insanity comes definitely to an end in from two or three weeks to three or even four months.

The features, then, that characterize these cases of temporary insanity are youth and previous good health; or in a few cases intemperate habits; the absence of hyperpyrexia; the existence of endocarditis; the settled though varying and even intermittent character of the taciturn delirium, which is present, though modified, by day as well as by night; and the dying out of the affection in a limited period. These conditions point, not to a rapidly progressive and varying cause, which marks hyperpyrexia, which is kept in check or suspended by a perspiring skin, or the external use of cold, and is promoted by a hot dry skin; but to a continuous cause, that is excited during the height of the disease, but that varies in operation for from two weeks to three months after the acute rheumatism and the acute stage of the endocarditis have passed away. In one of my own cases there was embolism, evidenced by the loss of power in the right side, and taciturn aphasia, in combination with endocarditis; and it appears to me that in embolism of the minute cerebral arteries of the convolutions, we have a series of conditions that correspond with those occurring in the whole of these remarkable cases. Embolism of the cerebral arteries comes on with endocarditis, and arrests for a time the circulation of the blood through the parts of the brain supplied by the affected vessels; its effects remain after the acute stage of the originating endocarditis has passed away; and, if death does not cut short the clinical history of the case, those effects usually gradually lessen and disappear in from two or three weeks to several months, unless the extent of the plugging of the vessels be such as to cause extensive softening of the part of the brain supplied by those vessels. I therefore consider that to embolism we may have to look for the explanation of these cases. We shall find other instances of a like nature among the cases without pericarditis, in which endocarditis was present, and in those also in which it was doubtful or absent.

B².—II. *Cases with Simple Endocarditis.*—Five of the sixteen cases that had endocarditis without pericarditis were affected with delirium of a desponding type with taciturn melancholy. Two of these died and three recovered. In addition to these five cases with taciturn melancholy, there

was another analogous case of embolism of the basilar artery, with headache and agitation, and in the evening apoplectic symptoms, right hemiplegia, and difficulty of speech.[1] As this case did not survive the first great attack, I shall not add it to the rest. The length of time that the mind was disturbed varied in the different cases from three weeks to four months; one of the fatal cases lasted twenty-three days, and another two months; while those that recovered were affected for one, two, and four months respectively. Four of them were restless; three were taciturn, especially during the night; another answered slowly; and the fifth case in a low voice; three had hallucinations, including one of those that were also taciturn, and two would get out of bed. Three of them were desponding or melancholy; one was apathetic; and the remaining one, a fatal case, was for ten days in a state of quiet delirium, and afterwards preserved a dogged silence. Two of them were confused; and one of them was violent. If we compare these five cases of temporary insanity, with simple endocarditis; with the sixteen cases of the same class with pericarditis and endocarditis, we find that the two sets of cases correspond in their main features. Both had disorder of mind, by day as well as by night, though with greater accentuation at night in those with simple endocarditis; in both early restlessness, obstinate silence, melancholy, apathy, and hallucinations prevailed; and in both the affection of the intellect commenced during the attack of acute rheumatism, and of the accompanying endocarditis, and lasted for a variable period after the acute affections had ceased.

As we have just seen, five out of the sixteen cases, or one-third, with simple endocarditis, not including the fatal case of embolism, difficult speech, and right hemiplegia, and another case with embolism of the minute cerebral arteries and delirium that died on the eleventh day; and sixteen of the sixty-five with pericarditis, all but one of them having endocarditis also, or one-sixth, were thus affected with taciturn melancholy lasting for a limited period after the cessation of the acute affection. We may, I think, consider that the existence of endocarditis in so large a proportion of such cases adds to the probability of embolism being the cause of the temporary insanity.

Since the above was written Dr. Broadbent has favored me with his notes of an important case of acute rheumatism and endocarditis, with chorea and delirium, in which there was capillary cerebral embolism. I have also met with a case observed by Dr. Dickinson of acute rheumatism and endocarditis with delirium and minute cerebral embolism, and red softening of some of the convolutions; and with another case of chorea and endocarditis with delirium and minute cerebral embolism. These three cases afford direct evidence of the association of embolism of the minute arteries of the convolutions of the brain with delirium.

Dr. Broadbent's patient, a laundryman, aged 17, when attacked, had severe affection of the joints, and was light-headed; two days later his right limbs twitched and jumped, and he was delirious. On admission, after being ill a week, he seemed stupid, had to be spoken to loudly, his answers were confused, his articulation was indistinct, and his limbs still twitched, but especially on the right side. T. 103°. During the two following nights he had no sleep, was very delirious, talked, screamed, and jumped out of bed. He slept after a dose of chloral, but was soon pale and prostrate, and died on the fourth day after his admission. Recent loose clots were found in the minutest arteries and capillaries of the *corpora striata* and of some of the convolutions.

B². —III. *Cases without Pericarditis, Endocarditis being absent or doubtful.*—Five of the thirty-eight cases in which there was no pericarditis, and endocarditis was absent or doubtful, or one in eight of the whole number, became delirious during the acute stage of the disease, and remained of unsound mind for two months and a half in one, and for about a month in four instances. Two of these patients were also affected with choreiform movements. Four of these were men, and one was a girl, aged 16. Two of the men had been at one time drunkards, one of them had suffered in health from losses and excesses, and the other man was a servant, and probably lived generously. The speech was affected in all of them. One stammered, one answered slowly, one was taciturn, one refused to answer, and the girl did not reply to the question put to her, but spoke of something else. Two of them had hallucinations; one was despondent; another, after being noisy, became sulky; one was morose by day, and had lively delirium. One, with choreal movements, after being confused and delirious in paroxysms, became so continuously; and the fifth, also having chorea, was strange in manner.

In none of these five cases was there any notable sign of endocarditis, and the disturbed state of mind and speech could not therefore be attributed to embolism.

B². *Temporary Insanity—General Summary.*—There were altogether twenty-one cases of acute rheumatism with temporary insanity; and six of delirium, usually of the low melancholy type, in which the in-

[1] Bouillaud, "Maladies du cœur," vol. i. p. 405.

sanity was cut short by death. Of these twenty-seven cases, sixteen had pericarditis, six simple endocarditis, and five had apparently neither pericarditis nor endocarditis.

Four-fifths of the cases had endocarditis (21 in 27), and one-fifth of them gave no evidence of endocarditis (6 in 27).

I have already given clinical reasons for thinking that the temporary insanity may have been due to embolism of the minute cerebral arteries in those cases with endocarditis, and direct evidence that in two cases that condition coincided with delirium.

In six of those cases with endocarditis the temporary insanity, delirium, or melancholy was associated with chorea, and their clinical history would seem to suggest that in those cases the temporary insanity and the chorea were due to a common cause acting perhaps on different parts of the nervous centre. This view is strengthened by Dr. Tuckwell's important remarks on Muscular Chorea and its probable connection with Embolism. This memoir is illustrated by a case[1] in which there were two large patches of red softening affecting the cortex, and in one of them the white substance also, of the right hemisphere of the brain. The arterial branches leading to one if not both of these softened patches were occluded by coagula, and very fine granular particles were dotted along the small bloodvessels in the softened cerebral gray matter. This patient, a boy, was attacked with chorea nine days before admission, and became delirious during the first night after it. On the third day he had wild maniacal delirium, and furious choreic movements. This wild state soon quieted itself, but was renewed on the eighth night, and on the ninth day he became comatose and died.

More than one-half of the cases were below the age of twenty-one (14 in 27), and of these all but one had endocarditis, while one-third of them were above the age of twenty-five (9 in 27) and of these nearly one-half (4 in 9) presented no sign of endocarditis.

Although the majority of these cases, and especially those with endocarditis, were young people of previously good health, yet a small but definite group of the cases form an important exception to this typical series. Six of the cases, all men, were either known to be of habits of intemperance, or were of occupations in which such habits are possible. Three of those male patients were drunkards or given to excess, and of the rest, one was a policeman, one a man-servant, and the sixth was a postboy. Four of these patients, all of whom recovered, presented no sign of endocarditis, and the two others had endocarditis.

The question arises here, Whether the temporary insanity in these four men who had not endocarditis, one of whom had chorea also, may have been due to thrombosis or the spontaneous collection of fibrine in the minute arteries of the convolutions? I simply put this as a question, but in support of the possibility of this condition I find an important case that was closely observed by Dr. Charlton Bastian during life and after death. The patient was a strong man, a gate porter, who had been accustomed to drink a great deal of late, and was attacked with erysipelas of the head and face following a fall, when he cut his head on the curbstone. He became violently delirious, was then quieter, became comatose at night, and died early on the following morning. The heart was healthy; the pia mater and brain were abnormally vascular; the consistence of the brain was good. Minute embolic masses were present in the small arteries and capillaries of the brain in every specimen looked at.[1]

B^3 & C $^{1, 2, 3}$.—Chorea and Choreiform and Tetaniform Movements, with and without Delirium, in Cases of Acute Rheumatism, with especial reference to Pericarditis.

The occurrence of chorea without delirium in cases of acute rheumatism when connected with endocarditis will be considered when we inquire into that affection. The present inquiry will be limited to (1) cases of chorea and of choreiform movements with delirium, or ending in sudden death, occurring in acute rheumatism with or without pericarditis; and (2) cases of chorea and choreiform movements without delirium, occurring in acute rheumatism with pericarditis; and in inquiring into these cases, I shall briefly include the cases of combined chorea and temporary insanity that have already been considered.

B^2 & $C^{1, 2}$.—I. *Cases with Pericarditis.*—Chorea occurred as a definite accompanying affection in six instances with delirium, and in seven without delirium; and choreiform movements not amounting to definite chorea occurred in two instances with delirium and in one instance without delirium among the sixty-five cases of acute rheumatism affected with pericarditis now under examination. In addition to these cases so affected, there were six patients with pericarditis who had

[1] British and Foreign Medico-Chirurgical Review, xl. p. 506.

[1] Path. Trans. xx. 8.

delirium, or coma, or both, as the principal affections, and who had choreiform movements as a subsidiary affection. There were thus twenty-two cases of rheumatic pericarditis with chorea or choreiform movements, not including several who had also tetaniform symptoms.

The thirteen cases with chorea, and two of the three with limited choreiform movements, were below the age of twenty-one, nine of these being girls and six youths. The remaining case with limited choreiform movements was a man aged 22. Nine of these sixteen choreal cases died and seven recovered.

Thirteen of these cases, including the whole of those with delirium, had endocarditis as well as pericarditis; in two cases endocarditis was probable, and in one it was absent or doubtful.

In eight of the cases the chorea appeared after the commencement of the pericarditis; in seven of them the two affections probably came into existence about the same time; and in one exceptional case, recorded by Dr. Ormerod, the chorea appeared first, then the pericarditis, and finally the affection of the joints, thus reversing the usual order of succession of those affections.

The chorea appears to have continued up to the time of death in most of the fatal cases when the pericarditis was active; but the reports of several of them are, in this respect, imperfect.

The relation of the termination of the chorea to that of the pericarditis varied much in the cases that recovered. In one case the choreic movements were violent on the day that the frottement diminished, and were absent four days later. In another case the chorea improved with the improvement of the state of the heart.[1] A patient of my own made objectless movements with his hands when the pericarditis was at its acme; and two days later those movements ceased. In a boy with pericarditis, violent chorea appeared when the rheumatic and cardiac affections rather suddenly disappeared. Six days later with return of pain in the joints the chorea ceased.[2] In another patient, a girl, chorea appeared four days after the disappearance of friction sound.[3]

Partial choreiform movements, usually of short duration, appeared in six of the cases that were affected with delirium with and without coma, and in all of them the movements appeared when pericarditis was present.

The character of the choreiform movements was peculiar in some of the cases. Two of the patients rolled the head from side to side; one smacked his lips, another pursed his mouth, a third snapped, grimaced, and cried out; two moved the left hand and arm constantly; and in five the spasmodic movements of the body were very violent, so that in three of them personal restraint was demanded. In one of those five patients, on the second and third days, the spasms put on the character of the most violent convulsions. The cases of chorea with delirium presented considerable variety; and several of them, as we have already seen, had temporary insanity.

Five of the eight cases with delirium were fatal, and three recovered. The duration of the cases varied considerably. The five fatal cases died at various periods from the fourth to the sixteenth day of the delirium. Of the three that recovered, one had delirium for a month, one had chorea for three weeks, and in one, a man under my care, quick and needless movements of the hands, and occasional muttering, lasted two or three days.

B^3.—II. *Cases with Simple Endocarditis.*—No case of chorea with delirium, and only one with rapid death, occurred among the cases of acute rheumatism with endocarditis.

B^3. — III. *Cases without Pericarditis, Endocarditis being absent or doubtful.*—I have already given two cases of this class with chorea and delirium that were affected with taciturn melancholy of limited duration. The third case, a girl, aged 14, also had chorea and delirium.[1]

B^3. *Cases with Choreiform Movements* in which those movements were partial and of secondary importance, and occurred in patients already included among those with delirium or coma. B^3. — I. *Cases with Pericarditis.*— Six cases with delirium, one of which had coma also, among the sixty-five cases of rheumatic pericarditis had movements of a choreiform character for a single day in the course of the disease. Three of these patients died and three recovered. Four of them were male and two were female patients, and of these, five were above the age of twenty-five, and only one below that of twenty-one.

B^4, C^3.—*Cases with Tetaniform Movements sometimes associated with Choreiform Movements.*—I. *Cases with Pericarditis.*—In a small but important group of cases tetaniform symptoms occurred in connection with rheumatic pericarditis. Seven of the sixty-five cases of pericarditis had tetaniform movements, or continuous contraction or rigidity of muscles, of greater or less intensity. Some of these affections

[1] Guy's Hospital Reports, vi. 1841, pp. 420, 421.

[2] Mr. Land, Lancet, 1873, i. 38.

[3] Dr. Kirkes, Trans. of the Abernethian Society, 1850, p. 57.

[1] Tüngel, loc. cit. 1860, p. 125.

approached in their severity and characteristic form to tetanus, others could only be indistinctly associated with that disease.

The first case was an excitable man, aged 19, a gardener, who had, when first seen, twitching of the muscles, and of the right side of the face, increased by speaking. He had increasing agitation, indistinct articulation, and a difficulty in opening his mouth, which he closed with a snap. After this he threw his head from one side of the bed to the other, his convulsions resembled tetanus and opisthotonos, and his distress in swallowing was like that in hydrophobia. Four days later he rolled his eyes, ground his teeth, and smacked his lips; and he died exhausted by laborious spasm and probably by want of sustenance. His brain was vascular, and there was questionable softening around a vascular spot in the spinal cord opposite the first dorsal vertebra. There was pericarditis and endocarditis.[1] The next case, an over-worked girl, aged 19, at a late period of its history, seemed to plunge almost at once into the tetaniform condition. She rolled her eyes wildly, had furious delirium, and violent tetanic spasms with firm clenching of the fingers. After a week the delirium subsided, but she talked incessantly and incoherently, and was half maniacal, half idiotic up to the forty-sixth day, but from that time her progress to recovery was steady.[2] The third case, a youth, had pericarditis, but no endocarditis, inflammation of the kidney, occasional delirium, and slight opisthotonos; and on the eleventh day he died.[3]

The two following cases, both of which were fatal, were under my own care. The more important case was a youth aged 17. On the eighteenth day, the left side and the tongue were affected with choreiform movements, which extended, with stiffness, to both arms. On the thirty-eighth the left arm, which still jerked and shook about, was rigid, the forearm being bent on the arm, the hand on the forearm. On the forty-seventh day, stiffness of the neck appeared, and he moved his arm with difficulty; and on the following day he died. He had both pericarditis and endocarditis. In this case the rigidity of the limb points to an affection of the nervous centre, probably due to embolism.

The other case, a man aged 27, a carpenter, came in with pericarditis at its acme, and endocarditis. On the third day he frequently slumbered and, as the eyes were half-closing, the arms and legs started. On the evening of the seventh day he was restless, and not quite rational, trembled, and kept moving his lower jaw and biting his lips. Half an hour later he was more noisy, and knocked about, still shaking his jaw. His pupils were dilated and very sluggish, and at eleven o'clock he died. I can find no notes of his *post-mortem* examination.

The two remaining cases with tetaniform symptoms belong to the group of cases of endo-pericarditis with delirium and coma. One of these, a young man, had pain in his right temple, followed by wild delirium. During the night general convulsions came on in occasional spasms of a tetanic character, and in the intervals he lay in a state of coma. He remained in this condition for three or more days, when he died.[1] The remaining case, a young woman, became restless and flighty on the sixth day of her illness, and next day pericarditis and endocarditis declared themselves. She then became very violent. The right arm and leg were never still; at times, however, this state became aggravated into one of general convulsions of a tetanic character. She continued thus for nine days, the convulsions being incessant. On the twelfth day she became comatose, after jumping up and falling out of bed with her forehead on the floor. She finally recovered.

Pericarditis — neither Rheumatic nor from Bright's Disease — accompanied by Affections of the Nervous System.

An important series of cases of pericarditis in which there was neither acute rheumatism nor, so far as was directly ascertained, Bright's disease, have been published from time to time by Rostan, Dr. Abercrombie, Dr. Bright, Bouillaud, Andral, and Sir George Burrows.

The cases of this class that I have gathered together from the records of various observers, and from the note-books of St. Mary's Hospital amount to twenty-six.

These cases present examples of the whole series of affections of the nervous system that have been observed in cases of rheumatic pericarditis, with the exception of those with temporary insanity, and these were possibly represented by one fatal case that had obstinate taciturnity.

Among these twenty-six cases, (A^1) one had coma without delirium; (A^3) four had delirium and coma, the delirium in two of them preceding, and in two of them following the coma; one had delirium and convulsions; (B^1) eleven had uncomplicated delirium which was slight and of short duration in seven of them; and of

[1] Dr. Yonge; Dr. Bright, Guy's Hospital Reports, v. (1840) p. 276.

[2] Dr. Fuller, loc. cit. 201.

[3] Dr. Fuller, loc. cit. 289.

[1] Sir Thomas Watson, loc. cit. ii. 306.

those without delirium, (C[1, 2]) three had chorea or choreiform movements; (C[3]) two had tetanus, and (D) one had slight convulsions. The affections of the nervous system in these cases of pericarditis, instead of being similar in character were thus very various.

A[1]. *Coma.*—The patient with coma was a woman who was suddenly seized with complete loss of consciousness, remained in this state four days, and died. Pericarditis was the only appreciable lesion.

A[3]. *Coma with Delirium.*—Among the four cases with coma associated with delirium, in two instances the delirium, as usual, preceded the coma, while in two the delirium followed the coma. One of the former class, a boy, aged 12, affected with pyæmia, was delirious, and after a night without sleep, became unconscious and died in the afternoon. Pericarditis was associated with small deposits of pus in the walls of the heart, the fibres of which were soft and almost black.[1] The other case in which delirium was followed by coma, presented tetaniform symptoms. A woman aged 26, was admitted soon after a false conception in a state of delirium and obstinate taciturnity. After this she frequently reversed her head backwards, had convulsive movements of the face, and the arms presented from time to time a rigidity almost tetanic. On the fifth day the arms when raised fell as if paralyzed, she became comatose, and died in the evening. Pericarditis was the only morbid state discovered after death.[2]

Three of these cases may have been affected with "hyperpyrexia." There is, however, no indication that their temperature was raised.

In the other patient, a house-painter, in whom the coma preceded the delirium, I think that Bright's disease, not noticed after death, when the kidneys were not examined, was the probable cause of the fatal conditions spoken of.[3]

The patient with delirium and convulsions was a schoolboy with evident pyæmia, who had, in the opinion of all who saw him, severe inflammation of the brain. His brain was healthy, but his pericardium was inflamed, and innumerable small patches of pus oozed from among the muscular fibres of the heart.[4] The case with convulsions without delirium was also a boy, aged 7, who had pain in the left side and the epigastrium, and on awaking next morning was seized with slight convulsions, sank into a low exhausted state, and died in half an hour. There was universal pericarditis, and when the heart was cleared from its soft gelatinous envelope, it was covered with small irregular granulations.[1]

B[1]. *Delirium* without coma or other complications was present in eleven cases of pericarditis not occurring in acute rheumatism or Bright's disease.

The most important of these cases was a man aged 36, under the care of Sir James Alderson. Three and a quarter pounds of dark amber-colored fluid were found in his pericardium, the heart being covered and the sac lined with a thick honeycombed layer of new membrane. The supra-renal capsules, especially the right one, were enlarged with tubercular deposit. He had excessive distress and pain over the heart, the whole front of the chest was dull on percussion, and the impulse and sounds of the heart were absent. From the presence of these signs Sir James Alderson concluded that his patient was affected with pericarditis. On the twenty-second day after admission he became delirious, and on the twenty-fifth he was maniacal, and died.

Another case of this class was a shoeblack, aged 67, who had delirium, with great loquacity. He raised himself suddenly, went to the window to breathe, returned to bed, lay down, and soon died. There was extensive pleurisy on the left side, spreading to the pericardium, which contained a pound of purulent liquid; the walls of the heart being soft and its fleshy substance yellow.[2] A third case, a man, had pericarditis associated with pyæmia, following an operation for stricture.

Seven of the remaining cases of this series presented only slight delirium, and may be conveniently grouped together. One, who had pleuro-pneumonia and pericarditis, recovered.[3] Another had slight delirium and fever, and pericarditis. One had empyema and pericarditis. Two cases under my care were delirious the day before death. One had pyæmia, and purulent dots were scattered through the fleshy substance of the heart; the other had empyema of the left side, and pericarditis. In the two remaining cases the delirium appeared a short time before death; one had extensive phthisis of the right lung and a vast cavity, the other had empyema of the left side, the pericardium being inflamed and thickened. In all these cases the delirium appeared to be quite as much connected with the disease upon which the pericarditis had grafted itself as upon the pericarditis itself, and in most of them it was little more than the wandering of mind

[1] Mr. Stanley, Med.-Chir. Trans. vii. 322.
[2] Andral, Clinique Medical, i. 25.
[3] Bouillaud, loc. cit. i. 319.
[4] Dr. Latham, London Medical Gazette, iii. 1829, p. 209.

[1] Dr. Abercrombie, Trans. Edin. Med.-Chir. Soc. i. 1821–24.
[2] Corvisart, loc. cit. p. 239.
[3] Bouillaud, loc. cit. i. 367.

incident to illness of so lowering and fatal a character.

The remaining patient of this group, a coachman, aged 51, and a drunkard, who was under the care of Dr. Chambers, presented a condition resembling delirium tremens. He had extensive pleuro-pneumonia of the left lung, and pericarditis. Two of these cases with brief delirium, were under the care of Sir James Alderson, and two under that of Dr. Chambers.

C[1], [2]. *Chorea and Choreiform Movements.*—Two cases of non-rheumatic pericarditis had chorea, and four presented movements of a choreiform character.

C[1]. *Chorea.*—One of the cases, a well-grown girl of 15, had chorea for six weeks before admission, and on the twenty-seventh day after it was suddenly seized with obstructed respiration followed by a convulsive fit, and died. There was pericarditis, and the mitral valve was somewhat thickened, but the endocarditis was not noted. The other case, a little girl, was under my own care. She was brought to the hospital in her mother's arms, in great distress. She presented prominence over the region of the pericardium, dulness on percussion extending up to the clavicle, and a pericardial friction sound. There was evidence also of pleurisy of the left side. Choreal symptoms appeared in the face, beginning in the corrugator supercilii, on the third day after admission, and chorea was established on the fifth day. On the ninth day she was much quieter; her face was pale, her lips were blue, and the veins of the neck pulsated, being full during expiration and during the ventricular systole; and a loud mitral murmur was audible at the apex. She died on that day, but I have found no notes of the examination after death.

C[2]. *Choreiform Movements.*—Four cases of non-rheumatic pericarditis presented in the course of their illness movements of a choreiform character. These cases hold an intermediate place between those with well-developed chorea, and those with regularly repeated local convulsive movements. The most important case ought perhaps to be included among those with chorea, but it developed certain characteristic symptoms of the choreiform type, that are rarely or never present in uncomplicated chorea. This patient, a young lady, after a fortnight of extreme restlessness, and a good deal of delirium, fell into a state resembling chorea with convulsive agitation of the limbs, constant motion of the head, and wild rolling of the eyes. Cold was applied to the head, her symptoms subsided in a few days, and she gradually recovered. Three months and a half after the commencement of her illness she took cold, became suddenly worse, and died on the seventh day. The pericardium was universally adherent to the heart by lymph, and a deposit of lymph covered its outer surface.[1]

The other three cases, all fatal, of non-rheumatic pericarditis were reported by Corvisart, and the most remarkable symptom was a state of extreme agitation amounting to jactitation. They all had pleurisy as well as pericarditis, and one had pneumonia also. One of them had delirium, in another the mind was affected, and in the third disturbance of the intellect was not noted.

C[3]. *Tetaniform Symptoms and Tetanus.*—Two of the cases of non-rheumatic pericarditis were affected with tetaniform movements, or rather with actual tetanus, some of the symptoms of which were unusual. One of these cases was a boy who when admitted had cramps, and a threat of suffocation. His fingers, arms, and forearms, his legs, and feet were strongly bent, and the muscles of his limbs and abdomen and his masseters were so hard that they felt like touching a stone, especially during the paroxysms. A warm bath and cold affusion gave great relief, and the paroxysms of suffocation ceased half an hour later. After this the jaws were slightly closed, he had a return of the suffocation, especially when he drank, and occasional cramps. On the fifth day he had a cold bath by accident, and was seized with cramp when in the bath. He had spasmodic contractions of great intensity on the following day, and died in a paroxysm of suffocation. There were two ounces of pus in the pericardium, the surface of which was injected.[2]

The other patient with tetanus was a gentleman of middle age who, when first seen, was suffering from a violent spasmodic contraction of his limbs. On the fifth day he had cramps of his extremities and occasional spasmodic rigidity of the whole body, which was sometimes bent backwards, being supported by the occiput and the heels in a state of complete opisthotonos. During the night his spasms were so severe that he could scarcely be kept in bed, and he died suddenly on the following day. He had pericarditis, and the brain and spinal cord were healthy.[3]

I have ranked these cases with those of tetanus because they presented universal rigidity of the limbs and body; which, in the first case, extended to the masseters; and in the second, caused, during the paroxysms, complete opisthotonos. There were, however, certain conditions in which they both differed from ordinary tetanus. In neither of them did the affection commence with trismus, and in the

[1] Dr. Abercrombie, Trans. of Med.-Chir. Soc. of Edin. i. 1.

[2] Bouillaud, loc. cit. i. 333.

[3] Dr. Mackintosh, Practice of Physic, ii. 25.

second case its presence is not mentioned. In both of them at the outset of the attack the muscles of the extremities were involved; and in the first of them, besides cramps of the legs, the fingers, arms and forearms were strongly bent. In tetanus I need scarcely say that the reverse conditions prevail, for trismus is usually the earliest symptom, and the affection of the limbs is comparatively late, while that of the hands and arms is usually slight, the extensor muscles being more affected than the flexors. In tetanus the advance of the disease is steadily progressive, but there was a suspension of the spasmodic contraction of the limbs in both of these cases.

Dr. Bright describes a case of tetanus occurring in a man affected with inflammation of the pleural surface of the right side of the pericardium, involving the phrenic nerve, in which there was no pericarditis. In this instance the tetanus advanced rapidly through its usual progressive course. On the first evening he complained of difficulty in opening his mouth, and swallowed with a convulsive catch. During that night his teeth were completely closed, and next morning he could get nothing into his mouth, and could not even swallow his saliva. There were slight indications of opisthotonos, and spasmodic action of the muscles of the back. In the afternoon there was no relaxation of the spasm; he had several epileptiform seizures, during which his face was purple, his eyes stared, and his whole body was convulsed. He rambled occasionally, and died twenty-four hours after the first seizure of dysphagia. Dr. Bright suggests that in this case tetanus may have been caused by the phrenic nerve being involved in the seat of the inflammation.[1]

Convulsive Movements, Chorea, and Choreiform and Tetaniform Symptoms in Cases of Acute Rheumatism with and without Pericarditis, and in cases of Non-Rheumatic Pericarditis, in which Bright's Disease was Absent. Summary. Convulsive Movements.—I do not consider here cases of coma with convulsions, of which there were five, three with and two without pericarditis, one with and three without endocarditis, which was probably present in the remaining instance; nor those with convulsions associated with albuminous urine, of which there was but one patient, affected with both pericarditis and endocarditis.

There were altogether nineteen patients with convulsive movements among the whole series of 180 cases of acute rheumatism with affection of the nervous system; twelve of whom had pericarditis and seven had no pericarditis, while eight or perhaps nine of them had endocarditis. Fourteen of these cases had twitchings of the limbs or face, and of these, eight had pericarditis, and five endocarditis.

From this *résumé* it is evident that although these convulsive movements are probably influenced, and may in some instances have been caused by the co-existence of pericarditis or endocarditis; yet they may, and often do occur quite independently of either of those affections, and in the absence of both of them.

Hyperpyrexia (actual in seven cases, inferred in four) was present in eleven of the nineteen cases with convulsive movements or twitchings. In Dr. Greenhow's important case, given at page 521, twitchings of the face were generally present when the temperature ranged from 102·1° to 106·2°, but they were suspended by the cooling bath, and returned after removal from the bath.

The general affection of the nervous system varied in the different cases with convulsive movements and twitchings. In one patient a convulsive fit preceded coma without delirium. In seven cases there was delirium followed by coma. In four of these, twitchings occurred during the delirium; while in two of them, twitchings, and in one, convulsive movements, accompanied the coma. Convulsive movements of the whole body in one instance, and of the face in another, followed delirium and preceded death. There were general convulsive movements in one, and twitchings of the face in two cases of uncomplicated delirium.

There were twitching movements in four cases with chorea and delirium, in one of which there was also a state resembling tetanus and opisthotonos. In these four cases the twitchings were probably choreiform in character.

In one of the two remaining cases, a slight fit with ptosis preceded death by a few hours; and in the other twitching was present with albuminuria.

Convulsions were present in three, and convulsive agitation of the limbs in one, and of the lips or face in two of the twenty-six cases of non-rheumatic pericarditis, in which there was no Bright's disease.

Chorea, and Choreiform, and Tetaniform Symptoms. Chorea.—Twenty-one of the 180 cases of acute rheumatism with affections of the nervous system had chorea. Fifteen of those patients with chorea had pericarditis, six had no pericarditis; while fourteen of them had endocarditis; three had no endocarditis; and in three of them, endocarditis was probable or doubtful. Pericarditis and endocarditis attacked three-fifths of these patients conjointly (13 in 21). It would appear from this, at first sight, as if pericarditis favored or influenced the production of chorea,

[1] Med.-Chir. Trans. xxi. 4.

thus apparently supporting the view of Dr. Bright that the more frequent cause of chorea in conjunction with rheumatism is inflammation of the pericardium, the irritation being probably communicated thence to the spine through the phrenic nerve.[1] This view is apparently strengthened by the history of several of the cases in which the chorea and the pericarditis appeared, improved, and disappeared simultaneously. On the other hand, in one case, chorea preceded pericarditis, and in at least two others it came into play when the pericarditis was vanishing. The united presence of inflammation without and within the heart in so many of these cases, complicates the question as to the influence of pericarditis on the production of chorea; and these clinical statistics favor the view that endocarditis may be the cause of the chorea, quite as much as that pericarditis may be its cause. I will not pursue this question in this place farther, excepting to repeat that in Dr. Broadbent's and Dr. Tuckwell's important cases of chorea and delirium, there was embolism of the most minute cerebral arteries, associated with endocarditis. These two cases seem to show that it is possible that in some of the above cases also, chorea may have been associated with minute cerebral embolism due to endocarditis. I have already illustrated the possible or probable connection of temporary insanity in cases of acute rheumatism with endocarditis and minute cerebral embolism, or with minute cerebral thrombosis, the convolutions being affected; and five of these cases of chorea had also temporary insanity, in three of which there was endocarditis, while in two there was no endocarditis.

Chorea was present in two cases of non-rheumatic pericarditis without Bright's disease. In one of these the onset of the chorea preceded, and in the other followed, that of the pericarditis. In one of those cases endocarditis was also present, and in the other it was doubtful.

Choreiform Movements. Jactitation.—Chorea, as we have just seen, affected twenty-one of the 180 cases of acute rheumatism with affection of the nervous system. Besides these there were fourteen cases that had choreiform movements without definite chorea. One patient moved automatically, as in chorea, and another made objectless movements with his hands. Both of these cases had endo-pericarditis. Eight patients were affected with jactitation, which was general in six instances, and limited to the right or left side in two. The whole of these patients had pericarditis, while endocarditis was present in three of them, was absent in one, and probably absent in four.

There was extreme jactitation of the whole body in three cases of non-rheumatic pericarditis, probably without endocarditis, observed by Corvisart; two of these had pleurisy, and the other one pleuro-pneumonia; those affections in two of the cases being the probable cause of the pericarditis.

The invariable presence of pericarditis and the frequent apparent absence of endocarditis in these cases of general jactitation, would appear to point to pericarditis as a possible cause of that condition, and perhaps by inducing reflex movements.

Agitation.—Fourteen of the cases with affection of the nervous system in which the temperature was not observed had agitation, which is a condition allied to general jactitation, which was also present in two of them. I find no express mention of agitation in the sixty-one cases in which the temperature was observed. Five of the fourteen cases with agitation had pericarditis; eight of them had endocarditis; while five of those cases had neither endocarditis nor pericarditis.

Ten of the cases with agitation died and four recovered.

Rolling of the Head from side to side.—A peculiar, regularly repeated rolling of the head from side to side occurred in eight of the 180 cases of acute rheumatism with affection of the nervous system. Five of these cases had well-developed chorea, and two others had limited choreiform movements. Six of these cases had pericarditis, while five of them had endocarditis, and in one its presence was doubtful. All of them had either endocarditis or pericarditis. This peculiar oscillating movement of the head, though generally connected in these cases with chorea or choreiform movements, is not, so far as I know, ever present in ordinary chorea, and it forms, therefore, a feature of difference between those cases and these. One patient, who had endo-pericarditis, delirium, and coma, rolled violently about the bed so that he required to be held down.

Choreiform movements were present in four cases of non-rheumatic pericarditis without Bright's disease. In one of these the state resembled chorea, there being convulsive agitation of the limbs and constant motion of the head, with delirium; in another patient there was slight convulsive agitation of the face; and in two other cases there was violent general jactitation.

Tetaniform Symptoms and Tetanus.—Thirteen, or if the presence of "risus sardonicus" alone be included, fifteen cases presented symptoms of a tetaniform nature.

In eight of those cases the tetaniform symptoms were general. Some of these

[1] Med.-Chir. Trans. xxii. 15.

had also chorea, or choreiform movements. In one such case the choreal convulsions put on a character resembling tetanus and opisthotonos, and the distress in swallowing was not unlike that in hydrophobia. Another case had opisthotonos and tetanic spasms; and a third had slight opisthotonos. Three other cases had spasms or convulsions of a tetanic character, which were accompanied in one instance by firm clenching of the hands. One patient under my care had stiffness of the neck; and rigid jerking and shaking about of the left arm; the forearm, at a later period, being bent on the arm, the hand on the forearm. The eighth case, a woman, had a temperature of 109·5, and after being put into a tub of cold water was attacked with tonic spasms. Two cases had spasms of rigidity of the muscles of the neck, in one after being cooled in the bath from t. 109° to 103·6°, and two had stiffness of the neck, one of which has been already alluded to.

One patient who was violently delirious at a temperature of 107·8° to 109°, after being bled, immediately passed into a state of unconsciousness, and was attacked with trismus, and convulsive movements of the jaws, hands, and arms. Two cases were affected with stiffness of the jaw; which was accompanied in one of them by swelling of the temporo-maxillary articulation; this being the patient just spoken of who was seized with tonic spasms after the bath; and who closed her teeth firmly over her lips, drawing blood. Another patient, a man, was continually moving his lower jaw and biting his lip; and another, also a man, kept incessantly pouting his lips and rubbing them over his teeth. One patient, spoken of above, with spasms of rigidity of the neck after the bath, had also spasms of rigidity of the lips. Another case with opisthotonos and tetanic convulsions, closed the lips in snaps before, and smacked the lips after having convulsions.

"Risus sardonicus" was observed in five cases, in three of which there were, and in two there were not, other tetaniform symptoms.

Of the above thirteen cases with tetaniform symptoms, not including the two with simple "risus sardonicus," ten had endo-pericarditis, one had pericarditis, endocarditis being absent, in one both of those affections were doubtful, and in one they were both absent. These clinical facts make it probable that pericarditis or endocarditis, or both, may sometimes be concerned in the production of tetaniform symptoms. Other influences were, however, at work connected with hyperpyrexia in some of the cases. Thus trismus appeared in one patient just alluded to who became unconscious after being bled, the temperature rising from 107·8° to 109°; and in three cases the tetaniform symptoms came into play after the excessive temperature had been cooled down by the bath.

We have already seen that in one case of non-rheumatic pericarditis ending in coma, the arms presented occasionally a rigidity as of tetanus; and that in two other cases of the same kind, there was actual tetanus of a peculiar type. These three fatal cases had no endocarditis.

Andral, in commenting on the first of these cases, or that with occasional rigidity of the arm, and delirium ending in coma, asks whether the cause of the affection of the nervous system in these cases is not in the inflammation of the pericardium itself? We have already seen that Dr. Bright looks to the communication of an influence from the inflamed pericardium, through the phrenic nerve to the spine, as a cause of choreal and tetaniform affections. I would here remark that tetanus may be caused by a wound and by exposure to cold, and there is nothing therefore inconsistent, so far as I can see, in the idea, that it may be caused also by an internal inflammation affecting local nerves, and through their channel acting on the spinal marrow.

Tetanus is not, so to speak, an intermittent contraction of the muscles of the reflex type, but a continuous contraction of the muscles, due to the direct continuous action of the spinal cord. In tetanus, as Dr. Lockhart Clarke has shown, there are areas of disintegration in the spinal cord. In traumatic tetanus, the cause of the affection is the injury to the nerve, and in these cases the nerve must carry from its periphery to its centre an influence that sets into action the disintegration of the cord. If the inflammation of the peripheral ends of the nerves of the pericardium excites tetanus, it would perhaps do so in some such manner as that just suggested. The cases of tetanus and tetaniform affection associated with pericarditis, though striking are very rare, and we may fairly ask whether in those cases in which the two affections coincided, some other cause may not have been at work to induce the tetanus. I know of no instance in which tetanus was induced by any other internal inflammation, and if this be so, it is not easy to see why pericarditis or pleurisy affecting the phrenic nerve should be the only internal inflammations capable of inducing that affection in its typical or modified form.

THE PHYSICAL SIGNS OF RHEUMATIC PERICARDITIS.

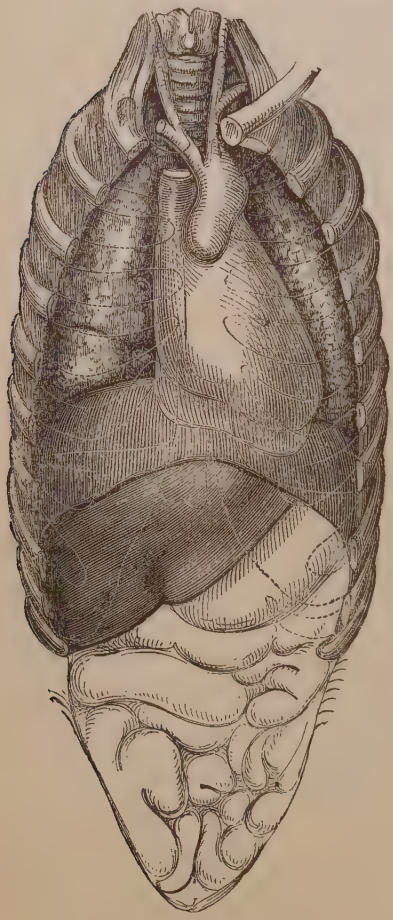
In every case of rheumatic pericarditis there is an increase in the amount of

fluid in the pericardium, and a layer of ridged, roughened, or honeycombed lymph is spread over the opposing surfaces of the heart and the pericardial sac. The amount of the fluid poured into the sac is made known by the extent of dulness on percussion, the prominence of the sternum and costal cartilages, and the widening of the intercostal spaces over the region of the pericardium, and by the position of the impulse; while the presence of lymph covering the heart and lining the sac is told by a friction sound.

Effusion of Fluid into the Pericardium in Rheumatic Pericarditis.—Although in the prescribed order, the examination of the chest by the eye and the application of the hand rightly precede that by percussion I shall here reverse this order, and begin with percussion, for by it we really judge of the extent of the fluid in the sac.

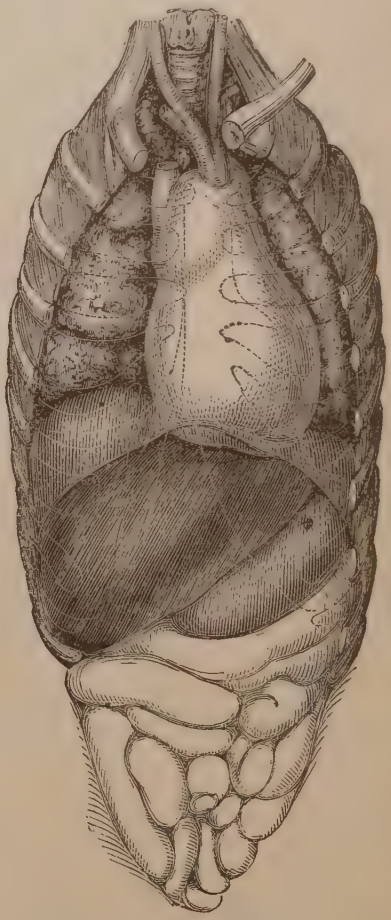
The pericardium of an adult man with a healthy heart is capable of holding from fourteen to twenty-two ounces of fluid, and that of a boy of from 6 to 9 years old, about six ounces, when the sac is distended

Fig. 79.



Pericardium not distended.

Fig. 80.



Pericardium artificially distended with fifteen ounces of fluid.

to the full by injecting water into it by a syringe, through an opening made in the anterior wall of the pericardium.

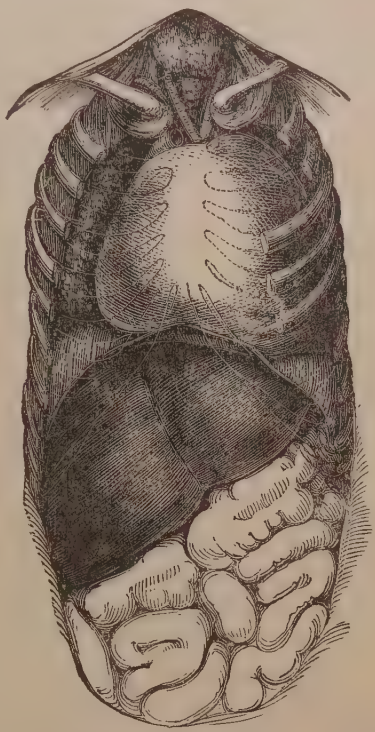
The effect of this artificial distension of the pericardium on the size, form, and position of the sac and on the situation of the surrounding parts is shown in the accompanying figures (79, 80). The pericardium, thus distended, is pyramidal or pear-shaped. It is formed, so to speak, of a larger and a smaller sphere, the smaller one resting on the top of the larger. The larger and lower sphere contains the heart, the ascending vena cava, and the pulmonary veins; and the smaller sphere holds the great vessels. The distended sac occupies the whole centre of the chest, filling up the space between the sternum

in front and the spinal column behind; and extending across the chest from a little within the right nipple to a little beyond the left nipple. The whole sac is lengthened; its smaller end reaches upwards almost to the top of the sternum; and its floor, being formed by the central tendon of the diaphragm, presents a large spherical prominence that bulges downwards into the abdomen, occupies the epigastrium, and reaches as low as the tip of the ensiform cartilage and the lower edge of the sixth costal cartilage. The enlarged and swollen sac displaces all the organs and parts surrounding it. In front it separates the two lungs from each other, so as to uncover the pericardium in front of the heart and great arteries. It pushes forwards the two lower thirds of the sternum, the ensiform cartilage, and the adjoining costal cartilages, especially the left, from the third to the sixth; and by counter-pressure backwards it compresses the œsophagus, the descending aorta, the bifurcation of the trachea, and the left bronchus between itself and the bodies of the vertebræ upon which those parts rest. It displaces the lungs to either side and backwards; and the central tendon of the diaphragm where it forms the floor of the pericardium, the stomach, and the left lobe of the liver downwards.

The artificial distension of the pericardium closely corresponds in general form with its natural distension from pericarditis, when the amount of the effusion has reached its acme. I have already sketched at page 496 what I believe to be the usual course of the increase of the effusion from the beginning of an attack of pericarditis to the period of its acme. When, however, the inflammation of the pericardium has existed for some time, the walls of the sac, so thin, tough, and firm in health, become comparatively thick, soft, and yielding; and as the sac cannot expand to a material degree either upwards towards the neck, or downwards towards the abdomen, it yields sideways and backwards, and widens to the right and especially to the left, so as to encroach on both lungs, but more seriously on the left lung; as may be seen in the accompanying figure, which is taken from a case of chronic pericarditis of long standing, in which the sac contained three pounds and a quarter of fluid (fig. 81). When thus distended, the sac seems to occupy the whole front of the chest; and it completely conceals the left lung, which is pushed backwards and compressed by it so that comparatively little air is admitted into that lung at its lower and posterior part; this effect being increased by the compression of the left bronchus.

There is another effect of this distension of the pericardium to which I have already alluded, its inferred effect namely upon the heart itself. The muscular walls of the ventricles are so thick, and their action is so powerful, that the direct effect of the fluid pressure upon them cannot be very great. But the pressure of the fluid tells inwards upon the weak and unresisting walls of the auricles, the vena cava descendens within the pericardium, and the pulmonary veins, so as to com-

Fig. 81.



Case of pericarditis in which one sac contained 3¼ lbs. of fluid. The patient was under the care of Sir James Alderson.

press and lessen those vessels and the auricles, and to resist and impede the currents of blood, on the one hand from the system along the cava, and on the other from the lungs along the pulmonary veins. This partial blocking of the double stream from the system and the lungs to the heart lessens the contents of the organ, and tends to diminish the size of its cavities. At the same time the supply of blood to the aorta is lessened, and the ascending aorta is therefore also compressed by the fluid. The pulmonary artery, however, owing to the obstacle to the flow of blood through the lungs, tends to resist the pressure of the fluid in the swollen sac, and to remain distended.

While, however, this influence on the part of the fluid pressure of the distended pericardium is at work compressing the auricles and veins; a second influence is

at work, also set up by the inflammation, to counteract the first influence, and to shield to some extent the weaker parts of the heart. The auricular appendices shrink at an early stage, and the walls of the auricles and veins are thickened and somewhat protected from the pressure of the effused fluid by a leathery coat of mail in the shape of the roughened and honeycombed coating of lymph that clothes and strengthens the feeble natural walls of those parts. Thus the double march of the inflammation supplies at the same time a compressing fluid, and a sustaining covering of lymph.

The distension of the pericardium with fluid produces two other effects on the heart. 1. The heart is heavier than the fluid in which it plays, and its ventricles consequently tend to sink backwards so that the left ventricle rests upon the posterior wall of the pericardium, just as the liver sinks backwards when the abdomen is distended with fluid in cases of ascites. 2. The other effect of pericardial distension on the heart is the lifting or tilting upwards of the organ within the sac. The heart is attached by its great vessels to the posterior and upper parts of the sac, and the whole organ, therefore, tends to shrink upwards and gravitate backwards towards its points of attachment. At the same time the accumulating fluid which occupies in volume the space between the lower surface of the heart and the central tendon of the diaphragm, displaces the organ upwards into the higher part of the pericardium.

The natural effect of this gravitation, shrinking, and upward displacement of the heart, owing to the great accumulation of fluid in the sac, would be, I conceive, if not modified by other agencies, to cause a layer of fluid to be interposed between the front of the heart and the anterior walls of the chest. Practically however we find that this is not usually the case over the mass of the ventricles; for with one or two rare exceptions we can always feel the impulse of the heart beating sometimes with force, sometimes with a thrill, in the second and third, or third and fourth left spaces, extending from the edge of the sternum to above and beyond the nipple. A layer of fluid is, however, evidently interposed between the lower portion of the front of the heart and the anterior walls of the chest.

The reasons for the presence and pulsation of the heart in the upper intercostal spaces when the pericardium is distended, I believe to be, firstly, the distension of the pulmonary artery, and to a less extent, of the right ventricle, owing to the difficulty with which the blood flows through the lungs; and, secondly, the raised position of the heart, which having left the broader space of the chest below, where it enjoyed free play, occupies its narrower space above, where the heart and pericardium are as it were grasped between the walls of the chest in front and the bodies of the vertebræ behind. The result is that under the combined influence of the elevation of the heart; the distension of the pericardium; and the contracted area of the upper part of the chest in which the heart is lodged, the left lung is displaced from before the organ and the right and left ventricles, and the apex and the great arteries beat against the higher intercostal spaces with which they come into direct contact. In consequence of the withdrawal of the lung from before the heart, and the narrowing compass of the portion of the chest in which the organ is situated, its impulse besides being raised, is also widened outwards, so that the apex beats against the third or fourth space, at or above the level of the left nipple, where it extends beyond the nipple line.

Although the upper portion of the right ventricle is in immediate contact with the walls of the chest, I am satisfied that a portion of the fluid effused into the sac is interposed between those walls and the lower portion of the right ventricle over its anterior surface.

We shall afterwards see that the impulse is raised in position when the fluid in the pericardium increases, and is lowered in position when that fluid diminishes, so that under these circumstances the varying amount of the fluid is told by the varying position of the impulse.

Cases that form the subject of this inquiry into the physical signs of pericarditis.—I possess notes of 44 of my 63 cases of rheumatic pericarditis, of the increase, acme, and diminution of the quantity of fluid in the pericardium, as shown by the enlarging and lessening area of the dulness on percussion over that region; the progressive changes in the position of the impulse; and the variations in the tone, intensity, and area of the friction sound; all of which signs are at once the effects and the witnesses of the advance and decline of the inflammation. I shall now briefly analyze, point by point, these parallel effects in those cases.

Percussion.

The enlarged Area of Dulness on Percussion over the Pericardium, caused by the Increase of Fluid in the Sac.—In 22 of the 44 cases under examination, the increased amount of fluid in the pericardium, as indicated by the extended area of dulness over that region, had already at the time of its first observation reached its acme,

and from that time, the amount of fluid with its area of dulness steadily declined. One of these cases had a relapse and proved fatal on the 14th day. In the remaining 22 cases the period of the greatest distension of the sac was preceded by a gradual increase, and was followed by a more gradual decrease, in the amount of the fluid; the periods of increase, acme, and decrease of the amount of fluid, being shown by the corresponding gradual enlargement, greatest area, and lessening of the region of dulness on percussion over the pericardium. In 11 of these 22 cases there was a single rise and fall of the tide of the effusion; but in the 11 remaining cases there was a relapse, and the amount of effusion, after lessening considerably, again increased and attained to a second acme. In five of those cases, indeed, there was a second relapse, so that the fluid in the pericardium presented a third, and in one of them even a fourth wave of increase.

The duration of the whole period of increase of dulness on percussion over the region of the pericardium varied much in different patients. Of the 22 cases in which the region of dulness had attained to its greatest area at the time of the first observation, the average duration of the increased dulness from the effusion into the pericardium was eight days, the extreme duration varying from three days on the one hand, to seventeen on the other. The average duration of the period of increased dulness in the 11 cases in which there was a gradual increase, single acme, and a decrease in the amount of fluid effused into the pericardium, amounted to fully eight days, the extreme variation ranging from four to thirteen days. The average duration of the whole period of increased pericardial dulness was more than twice as long in the 11 cases of relapse, as in the cases with a single acme, since in them it amounted to eighteen days, the extremes varying from fourteen to twenty-four days.

The increase of fluid in the early stage was usually rapid. In one-half of the cases in which this increase was watched, the area of dulness had reached its maximum, on the second or third day after the first observation (11 in 22), and in all but two or perhaps three of the remainder, on the fourth or fifth day. The early advance of the dulness was, as a rule, more slow in those patients who suffered from a relapse than in those who did not do so.

The time during which the effusion into the pericardium remained at its height was, as a rule, very short, In 39 of the 44 cases the region of dulness extended over its greatest area for about a single day. It may have lasted longer, but on the next examination, made usually on the following day, but sometimes later, the tide had turned and the extent of dulness had lessened. The acme of the pericardial dulness lasted two days in three of the remaining cases, and three days in two of them.

The period of the decrease of the effusion in the pericardium was much longer than that of its increase, its average duration having been, as we have already seen, eight days in the 22 cases in which the effusion was at its acme on the first examination.

We thus see that the period of the advance of the effusion, dating from the time of its first observation in the early stage, usually lasted about three days; that the period of the acme of the effusion was usually observed during only one day; and that the period of the decline of the effusion generally lasted about eight days.

The fluid in the pericardium begins to increase on the first day of the inflammation; but, as it necessarily gravitates backwards during the early stages, the effusion does not appear in front until it has accumulated so as to occupy the natural hollow at the back of the sac, and the space between the lower surface of the heart and the floor of the pericardium. Dulness on percussion over the region of the pericardium therefore does not declare itself until the inflammation has lasted for a day or two. I have no exact indications telling how soon the fluid advances to the front of the heart in sufficient quantity to push aside the lungs. That it must, however, have been rapid in certain cases is I think shown by the instance given on next page.

The effusion had reached its acme in one patient three days after the beginning of the attack of acute rheumatism; and the increased cardiac dulness was observed for the first time on the fifth or from that to the seventh day after the beginning of the illness in nine cases. Pain attacked the heart in three cases the day before, and in one three days before the first appearance of increased dulness over the pericardium; and from one to four days before the effusion had reached its acme in eight other cases.

Friction sound, like increased pericardial dulness, is not present at the first blush of pericarditis, and in my cases the two signs usually appeared at the same time. Thus they did so in 16 of the 22 cases in which the dulness on percussion was detected in the early stage; while in only one of those cases did the first brush of the friction sound precede, and in the remaining five it followed the onset of the increased pericardial dulness.

The upper boundary of the pericardial dulness when first observed, was limited by the space between the third and fourth left cartilages in 11 out of 22 cases, by the fourth cartilage in three cases, and by the

Fig. 82.

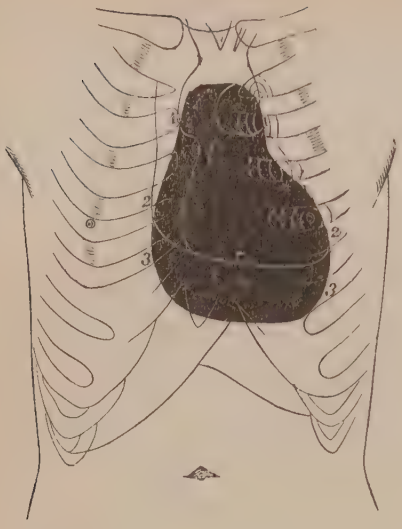


Fig. 83.

Figure 82, from a youth aged 17, affected with rheumatic pericarditis, who recovered in nine days from the time of his admission.

Period of the rapid increase of the effusion into the pericardium, just before the occurrence of its acme. The effusion completely distended the sac.

Day of admission.

The pericardial effusion distends, lengthens, and widens the sac, to the same extent and with the same effect as when the healthy pericardial sac is artificially distended with fluid (see figs. 79, 80, p. 540). The swollen pericardium is pyramidal or shaped like a pear, as in figure 80. Its smaller and higher portion (1, 1) contains the great arteries; and its larger portion is occupied above (2, 2) by the heart, and below (3, 3) by the great volume of fluid which accumulates between the under surface of the heart and the floor of the pericardium. The distended pericardium displaces the lungs upwards, and to each side; and the diaphragm, liver, and stomach downwards: and the fluid in the sac compresses the auricles; and that in the lower portion of the sac, between the under surface of the heart and the floor of the pericardium, elevates the heart. Owing to the displacement of the lungs from before the pericardium, the whole of the anterior surface of the heart and great arteries is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the ascending aorta within the pericardium and the pulmonary artery; and, owing to the elevation of the heart by the fluid, that organ presses and rubs with increased force against the walls of the chest in front of it; the anterior surface of the heart at its lower portion is, however, separated from the sternum and cartilages by a thin layer of interposed fluid.

This explanation, and that which follows, given once for all, will apply to figures 83; 86, p. 551; 88, p. 553; 91, p. 559; and 94, p. 576, which represent, each of them, the single, or first or second acme of the pericardial effusion.

There is *prominence over the region of the pericardium*, the left costal cartilages and ribs from the third to the eighth being raised and moved outwards.

The region of dulness on percussion over the distended pericardium ("pericardial dulness," see the black space) indicates the extent of the pericardial effusion; has the pyramidal or pear-shaped form of the distended sac; and extends from a little above the lower end of the manubrium, where it displaces the lungs, down almost to the tip of the ensiform cartilage, where it intrudes on the epigastrium. The lower and larger portion of the region of pericardial dulness over the heart and the great body of the effusion is more than twice the width of its upper and smaller portion over the arteries. This narrow upper portion forms therefore a peak which gives to the region of pericardial dulness its pear-shaped form, and which rises high behind the sternum, and occupies the lower portion of the manubrium. The wider portion of the region of pericardial dulness bears chiefly to the left; and its upper border, starting from the foot of its narrower portion, is on a level with one of the higher left costal cartilages or spaces. The upper and left boundary of the region of pericardial dulness is therefore indented; and its upper border is much higher behind the manubrium, than behind the adjoining left costal cartilage or space that may form its higher limit. The higher and narrower region of pericardial dulness (1, 1) over the ascending aorta and pulmonary artery, is about two inches in width, and is situated behind the sternum, on a level with the first and second spaces, and for about half an inch to the left of it in those spaces. The lower, larger, and wider region of pericardial dulness that extends over the heart itself

(2, 2) and over the accumulated fluid that occupies the depending portion of the sac below the heart, and that bulges downwards into the epigastric space (3, 3), extends from the upper edge of the third left costal cartilage, and the corresponding portion of the sternum, down to the lower edge of the sixth left cartilage, and almost to the tip of the ensiform cartilage; and from about an inch to the right of the lower half of the sternum, to half an inch or more to the left of the nipple. The lower border of the fifth cartilage, and a line running thence across the sternum to the fourth right space, *probably* forms the lower boundary of the heart (2, 2), and the upper boundary of the depending space (3, 3) occupied by the volume of the fluid distending the pericardial sac.

The *impulse of the heart* occupies the third and fourth left spaces (see the curved lines in those spaces), and extends in the latter space to just beyond the nipple; and the pulsation of the pulmonary artery is felt in the first and second spaces to the left of the sternum; where the first impulse is followed by a sharp second stroke, which is synchronous with the loud second sound of the pulmonary artery, and which gives the effect of a double impulse, one systolic and gradual, the other diastolic and sharp.

Figure 83, from the same patient as figure 82.

Period of the acme of pericardial effusion.

Third and fourth days after admission.

The explanation of pericardial effusion and dulness given with figure 82, applies also to this figure.

The pericardial effusion, which distended the sac on the day of admission (see fig. 82) has steadily increased in quantity since then, so that the whole pericardium has become enlarged, and has yielded sideways, and especially to the left; but it has not lengthened from above downwards. In this patient, therefore, the *region of pericardial dulness* (see the black space) during the acme is unusually wide, and especially along its left border; this increased width being fully as great above over the great vessels (1, 1), as lower down over and below the heart (2, 2, 3, 3). The left boundary of the region of pericardial dulness over the great arteries (1, 1) extends about an inch to the left of the sternum, in the first and second spaces; while the left boundary of the large region of pericardial dulness over and below the heart, extends fully half an inch to the left of the mammary line (2, 2, 3, 3). In all other respects, except the increase of the dulness to the left, the region of pericardial dulness corresponds with figure 82, taken on the day of admission. The apex of the left ventricle seems in this case to be behind the fourth left rib or space, and the lower boundary of the heart *probably* extends along the upper edge of the fifth left cartilage, and across the corresponding portion of the sternum; the heart having been much elevated by the increase of the fluid, which interposes itself between the anterior surface of the heart at its lower border and the walls of the chest.

The prominence over the region of the pericardium has increased.

The impulse is peculiar; it is felt beating (4th day) from the first to the third left costal cartilages, while the third and fourth spaces are retracted during the systole (see the curved and straight lines in those spaces). These movements give to the impulse the appearance of an undulation. The interposition of the fluid between the apex and lower border of the front of the heart and the walls of the chest has combined with the elevation of the organ to raise the impulse.

For later views of this case see figures 84, 85, p. 549.

third cartilage in seven cases. In one patient only did the dulness on its first observation reach as high as the second space.

The increase of the region of dulness over the pericardium was sometimes gradual, sometimes rapid. In rare instances the gradual ascent was slow and irregular. As a rule, however, the ascent was rapid.

The contour of the area of dulness on percussion over the pericardium when swollen with fluid in acute rheumatism corresponds very closely with the outline of the sac when distended with water after death. (See figures 79, 80, p. 540). In a paper in the Provincial Medical Transactions I gave illustrations of the area of pericardial dulness in which the boundary lines of the effusion were ascertained with care, and I here give figures of those cases (figs. 82, 83, p. 544; 84, 85, p. 549; 86, 87, p. 551; 88, 89, p. 553); and elsewhere, views taken from a case of pericarditis in St. Mary's Hospital, which show the same point during various stages of the affection. (See figures 90, 91, p. 559; 92, 93, p. 575; 94, p. 576.)

The form of the region of pericardial dulness changes as its area increases, its upper boundary being then on a higher level over the sternum than over the costal cartilages, instead of being, as in health, on the same level. The pericardial dulness, at the same time, extends further downwards in the manner shown in the figures just referred to, so as to intrude on the abdomen, and to replace the liver and stomach to a degree proportionate to the amount of the effused fluid.

When the increase of fluid in the pericardium reaches its height, and the sac is completely distended, the area of dulness over the affected region is pyramidal, or, more exactly, pear-shaped, and it extends over and beyond the heart, and in front of the great vessels. The inner borders

of the right and left lungs are pushed to each side by the distended sac, so as to expose the whole of the heart and the great vessels.

The region of dulness over the great vessels extends upwards from the level of the third cartilages, sometimes as high as across the middle of the manubrium, or within an inch of the top of the sternum, but more usually to a little above the junction of the manubrium with the long bone of the sternum, or about two inches below the upper end of the bone. This space of dulness over the aorta and pulmonary artery extends across the whole width of the sternum and reaches some distance to the left of it, in the first and second spaces.

The area of the region of dulness over the heart itself and the lower portion of the distended pericardium, may extend across the chest from an inch or more to the right of the lower portion of the sternum to an inch beyond the left nipple; and from above downwards from the second cartilage to the lower edge of the sixth cartilage. The extreme measurement from side to side of the whole region of pericardial dulness may vary from four and a half to six inches, and somewhat diagonally from above downwards, from five and a half to seven inches.

The lower portion of the region of dulness, from side to side, for the extent of about two inches from above downwards, is situated below the lower boundary of the heart; and is entirely occupied by the effused fluid, which here, as I have before shown, displaces the heart upwards, and the diaphragm, stomach, and liver downwards to an extent corresponding to the amount of the effusion.

The width of the region of pericardial dulness in front of the great arteries is usually about two inches, and this region usually ascends above the upper boundary of the heart to an extent varying from one inch to an inch and a half.

This upper region of pericardial dulness over the great arteries, which is two inches wide, is much narrower than the great region of dulness over the heart itself, which at its upper portion is above four inches wide, the greater width of the cardiac portion of the region of dulness being gained chiefly to the left. This sudden widening of the area of pericardial dulness from distension of the sac gives that area a peaked form above, and an indented outline along its left upper border, that distinguish it from the equally high and extensive area of cardiac dulness due to adherent pericardium and valvular disease, when the heart is enlarged in all directions and especially upwards and to the left, and when the upper left border of the region of cardiac dulness presents a very gradual inclination downwards and to the left without a break. (Compare figure 88 with figure 89, p. 553.) This pear-shaped outline of the region of dulness over the pericardium is quite characteristic, and indicates with certainty the presence of extensive effusion into the sac.

Among the forty-four cases, the upper boundary of the region of dulness when the effusion had reached its acme was over the first space or second cartilage in ten cases, over the second space in twenty-two, and over the third cartilage in twelve. In those cases that suffered a relapse, the first acme was as a rule higher, and the second, and still more the third acme were lower than the single acme in cases that had no relapse.

If the position of the upper boundary of the pericardial dulness over the cartilages and their spaces is known, the whole area of the region of dulness over the pericardium may be inferred with considerable accuracy; since the whole outline of that area shrinks when its upper boundary is lowered, and widens when it is raised. In this respect with certain definite reservations, the upper border of the region of pericardial dulness over the cartilages and spaces to the left of the upper half of the sternum, serves to measure the whole area of dulness and to define its complete outline; just as the ebb and flow of the tide, or the rise and fall of a flood indicated on a measuring post, will tell any one accurately acquainted with the coast, or the contour lines of the country, the exact area over which the land is covered by water.

If the upper boundary of pericardial dulness reach to the second space, the contour line defining the dulness extends —to within an inch of the top of the sternum; an inch beyond the right edge of the lower half of that bone; and more than an inch below its lower end, where it may descend as far as the tip of the ensiform cartilage; to the lower edge of the left sixth cartilage; and about an inch beyond the left nipple. (See figures 88, p. 553; 91, p. 559.) If the upper margin of dulness be limited by the third space, the boundary line extends—across the sternum on a level with the third costal cartilages; to the right edge of that bone; and to fully half an inch below its lower end; to the upper edge of the sixth cartilage; and to the left nipple. (See figures 84, p. 549; 90, p. 559.) The lungs, the diaphragm, the liver, and stomach are all correspondingly displaced, to a greater degree all round when the upper limit of dulness is over the second cartilage; and to a lesser degree all round when that limit is over the third space. The intermediate position of the upper

edge of dulness over the other cartilages and spaces gives an intermediate outline of the whole area.

The restrictions to this rule are due to age and sex, to previous affections of other organs, to valvular disease of the heart of old standing, to coinciding affections of the lungs, especially the left lung, to the duration of the attack of pericarditis and the occurrence of relapses, to accompanying endocarditis, to the progress of the disease, and to its terminations, whether in complete restoration to health, the valves being intact, in valvular disease, or in pericardial adhesions. These restrictions are numerous in appearance, but practically they seldom interfere with the rule just stated of the correspondence of the whole area of dulness with the boundary of a particular part of it.

The rule that the region of pericardial dulness in rheumatic pericarditis enlarges over corresponding areas in different cases, holds good in young persons of both sexes, and in women. In men, however, the bony framework of the chest is larger, the lungs are more ample and cover the heart to a greater extent, and the diaphragm is lower than in boys, youths, or women. The result is, that in men both the upper and lower boundaries of the region of pericardial dulness are lower than in the classes just spoken of. Thus the upper boundary of dulness during the acme was over the third cartilage in 8 out of 14 cases of rheumatic pericarditis in men; while in the whole of those of the female sex so affected, except one, that boundary was above the third cartilage. In nearly one-third, or 3 in 11 of the male youths with rheumatic pericarditis, the upper boundary of the region of dulness during the acme was over the third cartilage. This is due to the fact that in the male sex, the lungs at a comparatively early period are more largely developed than in the female sex.

When rheumatic pericarditis attacks a heart enlarged from previous valvular disease, the pericardial sac, being more ample, is capable of containing a larger amount of fluid, and the region of pericardial dulness is of greater relative width than when the affection attacks the virgin heart.

If the lower lobe of the left lung shrinks, owing to the combined effect of the compression of that lobe and of the left bronchus by the swollen sac, and of pleurisy with or without pulmonary apoplexy, a condition of things by no means unusual, the whole area of pericardial dulness tends towards the left, and its left border comes into direct contact with the ribs at the side.

Changes in the Form of the Outline of Pericardial Dulness caused by Variations in the Progress and Termination of the Affection.—If the attack lasts long, the pericardial sac, as I have already stated, becomes softened, it yields sideways, and becomes widened to the left and right, while it is not proportionally lengthened above and below (see figure 81, p, 541). This is especially to be noted when relapses take place, and when the effusion, after lessening in quantity, again increases. (See figure 94, p. 576.)

If the affection passes quickly through its stages, and the recovery is perfect, the heart being restored to health, the changes of the increase, the acme, and the decline of the pericardial effusion and of the area of pericardial dulness pass through the course I have described. (See figures 82, 83, p. 544; 84, 85, p. 549.)

If, however, the heart becomes enlarged owing to the establishment of valvular disease, the lessening and disappearance of the effusion are delayed, and the area of dulness is somewhat widened and lowered, especially towards the left.

If along with valvular disease, adhesions of the heart are established, the whole organ is enlarged, upwards, downwards, and sideways. The outline of the area of dulness loses its characteristic pear-shaped form, and its peaked outline over the great vessels gives place to a gradual widening of that area from above downwards, that corresponds with the enlarged outline of the heart itself. (Compare figure 88 with figure 89, p. 553.)

Prominence over the Region of the Pericardium.

Increased dulness on percussion over the region of the pericardium is the only reliable sign of the increase of fluid in the sac. Increased prominence of the costal cartilages over the heart, with widening of the spaces between them, form, however, a secondary sign of some interest and value.

In my paper before alluded to, I state that the distension of the pericardial sac by fluid, besides displacing the surrounding organs, pushes forward the sternum, elevates the costal cartilages from the second to the seventh, widens the spaces between the cartilages and ribs from the second to the sixth or seventh, pushes outwards the sixth left rib, and causes some degree of prominence over the left side.

This condition was observed with care in one or more of the cases of pericarditis examined by me in the Nottingham Hospital. I find that prominence over the region of the pericardium was noticed by me in 19 of 63 cases of rheumatic pericarditis under my care in St. Mary's Hospital. More than three-fourths of those

patients (15 in 19) were males, while only 4 were females. The cardiac prominence is obscured in women by the mamma; that sign having been observed in only one-seventh of the female cases of rheumatic pericarditis (4 in 27), while it was noticed in nearly one-half of the male cases (15 in 36).

The increased prominence over the region of the heart was usually noticed when the effusion into the pericardium was at its height, and it lessened when the effusion declined. In the greater number of the cases (12 in 19), the prominence over the region of the heart is described in general terms, but in seven its area was specified. In one of these it extended from the second cartilage to the sixth; in two, from the third to the sixth; in three, from the third to the fifth; and in the remaining one, from the fourth cartilage to the sixth.

In these cases the cartilages yielded to the distension of the sac, and were displaced by it forwards and upwards; with the good effect of somewhat relieving the pressure exerted by the swollen sac on those important structures, the bifurcation of the trachea, the left bronchus, the œsophagus, and the aorta, that are situated between the back of the pericardium and the bodies of the dorsal vertebræ. The prominence over the cardiac region caused by the forward pressure of the enlarged pericardium, points out that a serious counter-pressure backwards is exerted at the same time on the three vital tubes that I have just named, which convey air to the lungs, and especially the left lung, food to the stomach, and blood to the lower half of the frame. Indeed, the true value of this sign is that its presence reveals to us at the surface, the existence of deep and serious pressure on important internal parts, a pressure that is augmented when the superficial prominence increases, and that is relieved when that prominence lessens.

It is to be remarked that at the same time that the sternum and cartilages over the region of the distended pericardium are rendered prominent with the effect of somewhat lessening the pressure of the swollen sac upon the bifurcation of the trachea, the left bronchus, the œsophagus and the aorta—the dorsal portion of the spinal column deepens itself and curves backwards so as to afford increased space for the swollen sac, and those important tubes that are compressed by it. At the same time the patient sits up, and even leans forward, so as to allow of the gravitation downwards and forwards of the fluid in the pericardium. By this attitude, and the deepened spinal curvature, indeed, the pressure of the distended sac upon those vital parts is materially lessened, breathing and swallowing are rendered less difficult, and blood is supplied through the descending aorta with greater freedom to the body and lower limbs.

The Position of the Impulse of the Heart in Cases of Pericarditis.

When the amount of fluid in the pericardium has increased so as to enlarge the area of dulness on percussion over the region of the heart, the seat of the impulse is raised and extended outwards.

I gave figures of three cases of pericarditis with great increase of fluid in the sac, in my paper on the position of the internal organs, in which the impulse was present in the third and fourth spaces, instead of occupying its usual position in the fourth and fifth spaces. In that paper, attention was I believe called for the first time to the elevation of the impulse in cases of pericarditis with effusion into the sac.

In thirty-seven of the forty-four cases of rheumatic pericarditis, the exact position of the impulse during successive visits is stated, in five others the impulse is described, but its situation is not specified, and in the remaining two the impulse was almost or quite imperceptible.

In examining these cases I shall study the position of the impulse from two points of view, (1) the elevation of its lower boundary; (2) its diffusion into the higher intercostal spaces during the period of the increase of fluid in the pericardium.

(1) *The Elevation of the* Lower Boundary *of the Impulse.*—In fourteen cases, the extent of dulness on percussion over the region of the pericardium increased, and the effusion attained to its acme after the first observation; and in twelve of these the impulse occupied a higher position at the time of the acme than at that of the first observation, while in two its position was unchanged.

In twenty-two of the patients the amount of fluid in the pericardium was at its greatest height or acme at the time of the first observation; and as the effusion lessened, in eighteen of these the lower boundary of the impulse fell, in three it was stationary, and in one it became higher in position.

We thus see that in thirty of these thirty-seven cases of rheumatic pericarditis, the lower boundary of the impulse was raised in position when the amount of effusion in the pericardium was at its acme.

Fig. 84.

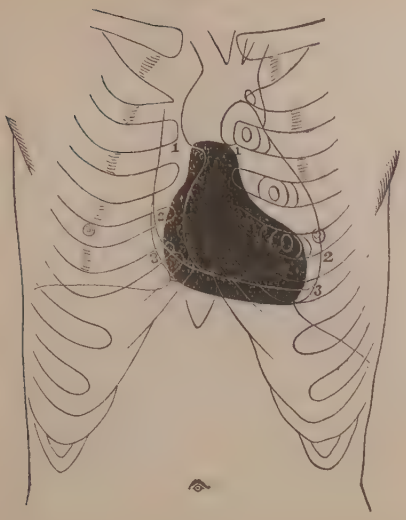
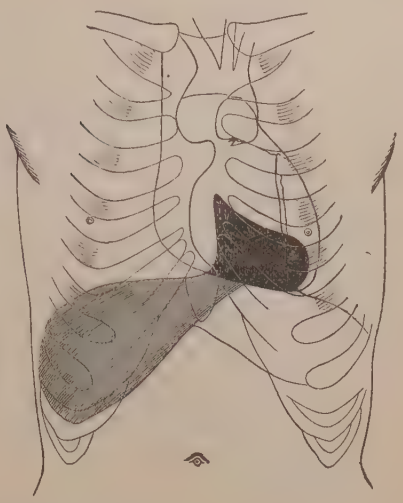


Fig. 85.



For previous views of this case, see figures 82, 83, page 544.

Figure 84, from a youth aged 17, affected with rheumatic pericarditis.

Period of the decline of the pericardial effusion.

Sixth day after the acme of pericardial effusion, eighth day after admission.

The pericardial effusion has diminished to a great extent, and the sac, no longer distended, has contracted, so that it has lost its pear-shaped form, and resumed more nearly that of the heart itself, a little modified and enlarged by undue fulness above. The lower border of the heart is much lower than during the acme, being situated behind the fifth cartilage, and the lower boundary of the pericardium is much higher; it no longer protrudes into the epigastric space, but has shrunk upwards, being situated behind the upper third of the ensiform cartilage, and behind or above the upper edge of the sixth left cartilage. The right ventricle and the apex of the left ventricle are exposed; but the upper part of the conus arteriosus and of the front of the left ventricle, the pulmonary artery, and the ascending aorta, are covered with lung.

The prominence over the pericardium has almost disappeared and the left side has nearly resumed its natural shape.

The region of pericardial dulness (see the black space) corresponds with the lessened amount of the pericardial effusion, and instead of being pear-shaped, or longer than it is broad, as it was during the acme, it has now more nearly the contour of the natural region of cardiac dulness, and is broader than it is long. It still, however, presents a peaked form at its upper border behind the sternum, where that border is on a level with the third cartilage, and where it is still much higher than its upper border to the left of the sternum, which is situated at the third left space. Its lower border is situated behind the upper third of the ensiform cartilage; and its right and left borders are respectively behind the right margin of the sternum, and within the left nipple.

The *impulse* is felt in the first, second, third, and fourth left spaces, being feeble in the fourth space. (See the curved and circular lines in those spaces.)

Figure 85, from the same patient as figures 82, 83, 84.

Period of the disappearance of the pericardial effusion and restoration of the heart to its natural position, which is, however, still rather high.

Eighth day after the acme of pericardial effusion, tenth day after admission.

There is no pericardial effusion, and the chest has resumed its natural shape.

The *region of cardiac dulness* (see the black space) has regained its natural form, and is no longer preternaturally higher behind the sternum than to the left of it. Its upper boundary is situated behind the fourth cartilage, and the adjoining portion of the sternum, its lower boundary, is behind the fifth space and the upper end of the ensiform cartilage; its right margin is a little to the left of the middle line of the sternum, and its left border is fully half an inch within the mammary line.

In one-fifth of the cases (7 in 37) the lower boundary of the impulse was pushed up as high as the third space, and in three-fifths of them it was present in the fourth space (21 in 37). In two patients, one with disease of the aortic and mitral valves, the other with that of the mitral valve alone, of some standing, the impulse was seated in the sixth space, in three cases it occupied the fifth space, and

in three it was felt over the third cartilage.

The existence of previous valvular disease, owing to the increased size of the heart in such cases, exercised a marked influence on the position of the lower boundary of the impulse, and as a rule lessened or prevented its ascent during the acme of the effusion. Thus, of five patients of this class, all of whom had affection of the mitral valve, and one of them of the aortic valve also, in two the lower boundary of the impulse occupied the sixth space, in two the fifth space, and in one it was seated in the fourth space.

If we deduct from the thirty-seven cases these five with valvular disease, which are exceptional both in their nature and as regards the influence of the effusion on the seat of the impulse, we find that in only one of the remaining thirty-two patients was the lower boundary of the impulse as low as the fifth space during the acme of the effusion.

These cases of previous valvular disease are exceptional in another point of view. In three of these five patients the position of the lower boundary of the impulse was not higher during the acme of the effusion than at other times. If we deduct these five cases from the thirty-seven under review, we find that in only three of the remaining thirty-two cases was the position of the lower boundary of the impulse unchanged during the acme of the effusion, while in twenty-nine of them it was definitely higher than in health.

Extent to which the Lower Boundary *of the Impulse was Raised, when the Effusion into the Pericardium was at its Height or Acme.*—In the twelve patients in whom the acme of the effusion was reached after the first observation of increased dulness on percussion, and in whom the lower boundary of the impulse was then elevated, the impulse at its lower boundary ascended two spaces in two instances (compare figure 90 with figure 91, p. 559), a space and a half in one, one space in six, and less than a space in three cases; and it descended after the acme two spaces in five instances, one space in five, less than a space in one, and in the remaining case its descent was not observed.

In the eighteen cases in which the effusion had attained to its acme at the time of the first observation the lower boundary of the impulse subsequently descended two spaces in three patients, one space in thirteen, one rib's breadth in one, and half a space in one case.

If we combine these thirty cases in one group, we find that the lower boundary of the impulse was higher during the acme of the effusion than in the natural state by two spaces in eight cases, by one space in nineteen, and by less than a space in three cases.

Time occupied during the Ascent and the Descent of the Lower Boundary of the Impulse in connection respectively with the Increase, the Acme, and the Decline of the Fluid in the Pericardium.—In the twelve cases in which the impulse at its lower boundary ascended to its highest point after the first observation, and during the period of the increase of the pericardial effusion, the time occupied by its ascent was from one to two days in nine cases, and from four to six days in three cases.

The lower boundary of the impulse fell from its highest position to its natural one in from one to two days in ten cases, in from three to nine days in eighteen, and in sixteen days in two out of a total of thirty cases. The ascent of the lower boundary of the impulse was therefore more rapid than its descent.

Relation between the Extent of the Effusion in the Pericardium, and the Height of the Lower Boundary of the Impulse.—The clinical facts just given show that the lower boundary of the impulse was raised by the increase of the fluid in the pericardium; and we find, therefore, as a rule, a relation between the extent of the effusion and the height of the impulse in these cases of pericarditis. But this rule is reversed in a small group of exceptional cases, amounting to seven, in which the upper limit of the effusion was as high as the first space or the second cartilage; while the lower boundary of the impulse was present in the sixth space in one, in the fifth space in two, in the fourth space in three, and in the third space in only one of these cases. Three of these patients in whom the impulse was low had valvular disease of old standing, a condition that, as I have already shown, prevents or lessens the ascent of the impulse.

(2) *The Diffusion of the Impulse over the* Higher *Intercostal Spaces during the Acme, and Decline of the Fluid in the Pericardium.*—In three-fifths of the cases (22 in 37) the impulse, at the time of the acme of the effusion, extended upwards above its lower boundary to the extent of one or more of the higher intercostal spaces. In more than one-half of these cases the impulse was felt beating as high as the second space (12 in 22), while in less than one-half of them its upper limit was the third space (10 in 22). The extent to which the impulse was felt in the higher spaces was naturally regulated by the position of its lower boundary. Thus, the impulse was bounded below by the fourth space in ten cases, and in eight of these it extended up to the third space or cartilage, and to the second space in only two; while in eight other patients the impulse, which was bounded below by the third space or cartilage, spread upwards to the second space. According, therefore, to the degree to which the impulse was

Fig. 86.

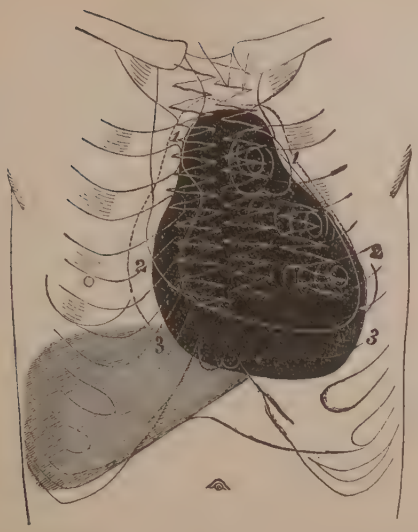


Fig. 87.

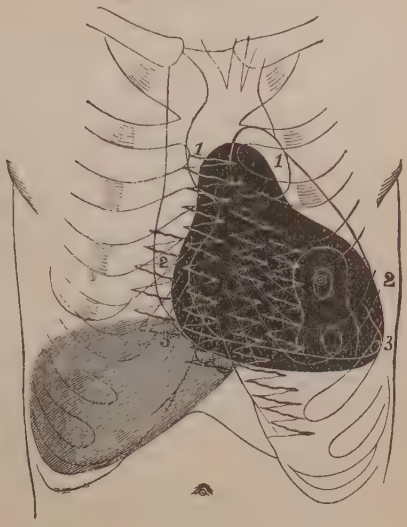


Figure 86 from a housemaid aged 17, affected with rheumatic pericarditis.

Period of the first acme of pericardial effusion, fifth day after admission.

The explanation of pericardial effusion and dulness given with figure 82, page 544, applies also to this figure.

The *pericardial effusion* extends less to the left and more to the right than in figure 83, page 544 (acme of pericardial effusion), and is of about equal extent in the two figures from above downwards. The heart, which is enlarged, is elevated by the fluid, but to a less degree than in figure 87, its lower boundary being *probably* situated behind the lower border of the fifth cartilage, and just above the lower end of the sternum.

The whole front of the heart is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the ascending aorta within the pericardium, and the pulmonary artery.

The region of pericardial dulness (see the black space) extends from a little above the lower end of the manubrium and the second left space, down to the tip of the ensiform cartilage, and the middle of the sixth cartilage; and from a little over an inch to the right of the lower half of the sternum, to a little beyond the left mammary line. The area of dulness includes (1, 1) the region of the great arteries; (2, 2) that of the heart; and (3, 3) that of the volume of the effused fluid below the heart, and projecting downwards into the epigastric space.

The *impulse* is less elevated than in figure 83 (acme), being situated in the second, third, and fourth spaces. (See the curved and circular lines in those spaces.)

The *friction sound* (represented by zigzag lines, the systolic lines being thick, the diastolic thin), is heard, double, over the whole length of the sternum, being audible, with pressure over its upper third (the great arteries), and without pressure over its lower two-thirds; and is also audible with pressure from the third to the fifth left cartilages (right ventricle); and over, but not beyond the apex of the left ventricle.

A loud mitral murmur is audible extensively to the left of the heart.

Figure 87 from the same patient as figure 86.

Period of the decrease of the pericardial effusion after the first acme.

Eighth day after admission, third after the acme—for the sounds. Eleventh day after admission, sixth after the acme—for the pericardial effusion and dulness, and impulse.

The pericardial effusion has lessened considerably, but is still present in considerable quantity. The right ventricle and the apex and front of the left ventricle are completely exposed; and the left border of the right auricle, and the lower portions of the ascending aorta and pulmonary artery, are also brought into view. The heart (2, 2), which is enlarged, has dropped down into its natural place, and even extends beyond that place, at its lower and left boundaries. The amount of effusion between the under surface of the heart and the floor of the pericardium (3, 3) is very small.

The region of pericardial dulness (see the black space) has lessened considerably in area; it extends from between the second spaces, behind the sternum, down to the lower third of the ensiform cartilage; from the third left space to the upper border of the sixth cartilage; and from the right edge of the sternum to a point an inch beyond the left mammary line. There is reason to believe that adhesions have formed at the apex, so that the latter boundary is

not pericardial but cardiac. The region of dulness over the great arteries (1, 1) is still very marked but has materially lessened; that over the heart (2, 2) being still extensive; and that over the depending portion of the pericardial effusion between the under surface of the heart and the floor of the pericardium (3, 3) being very narrow, indeed a mere strip.

The impulse of the apex is felt in the sixth space, considerably to the left of the nipple. The position of the impulse elsewhere is not mentioned in the report, but I have given it in the figure as being present in the fourth and fifth spaces, because three days later, at the time of the second acme, it was felt in those spaces, as well as in the second and third spaces. (See the circles and curved line in those spaces.)

The friction sound (see the zigzag lines, systolic thick, diastolic thin) on the seventh day had increased considerably below and to the right, and lessened above and to the left. It was audible over the sternum from below, but not above, the level of the second spaces, and thence down to the tip of the ensiform cartilage; to the right of the lower half of the sternum; and over the left cartilages, from the third to the seventh, where it extended about two inches below the heart; but it was inaudible over the region of the apex, where there were probable adhesions.

For the later views of this case, see figures 88, 89, p. 553.

raised by the increased amount of fluid in the pericardium, it was felt beating in the second and third spaces, or the third and fourth spaces, instead of, as in health, the fourth and fifth spaces.

In these cases there were two agencies at work: one, the increase of fluid in the pericardium, which elevated the heart and its impulse both at their lower and upper boundaries into the contracted space at the higher part of the chest, and caused the heart to beat against the left upper spaces; the other, the enlargement from distension of the right ventricle and especially of the pulmonary artery, owing to the difficulty with which the blood passes through the lungs from the combined effect of the pressure upon the auricles by the fluid in the swollen sac, and the existence of endocarditis with mitral regurgitation. The enlarged right ventricle and pulmonary artery displace the lungs, and pulsate, the former against the third, the latter usually against the second space; and in that space the double beat of the artery is then felt, the first being feeble, the second sudden and like a shock, coinciding with a feeble first and intensified second sound heard over the same situation. When the heart is much raised, it is evident that the conus arteriosus must sometimes occupy the second space, the pulmonary artery being elevated into the first space.

After the acme, when the amount of the fluid in the pericardium lessened, the position of the impulse, as we have just seen, as a rule descended at its lower boundary, but it generally retained its place at its upper boundary. Sometimes, indeed, the impulse extended upwards as well as downwards during the period of the lessening of the effusion.

The clinical facts that I have just related as to the extension of the impulse into the upper region during the successive periods of the increase, the acme, and the decrease of the effusion into the pericardium; while its lower boundary steadily rose during the increase, and fell during the decrease of the fluid, are to be traced I consider to a succession of causes. I have just considered the two agencies that are at work to extend the impulse into its higher region during the periods of the increase and acme of the effusion; the increase namely of the pericardial fluid elevating the heart into the contracted space of the chest above; and the enlargement of the right ventricle and pulmonary artery from obstruction to the flow of blood through the lungs. During the decline of the fluid the first of these influences is reversed, for the heart descends into its natural place, where it beats with comparative freedom; but the second influence, the enlargement of the right ventricle and pulmonary artery from obstruction through the lungs, often remains in full force to retain the impulse in its higher position; and this influence is frequently added to by other causes that have a like effect. These additional influences include the thickening and matting of the inflamed pericardium; the possible adhesion from pleurisy of the left lung to the pericardium at its upper border; and the deficient or absent expansion of this portion of the lung from adhesion and other causes, such as pulmonary apoplexy, and the imperfect general use of the left lung. These views derive additional confirmation from the fact that in all the cases save one in which the impulse extended over the higher spaces during both the acme and the decline of the effusion, there was endocarditis with mitral incompetence, and in several of them, aortic incompetence also.

Position of the Impulse after the Decline of the Pericardial Effusion during the Later Stages of Rheumatic Pericarditis; and after its Cessation.—When the effusion disappears and the heart resumes its natural position, and when the lungs again cover the great vessels and the upper part of the organ in front, the impulse as a rule descends into its natural position, and is again felt in the fourth and fifth spaces.

In those patients in whom the heart be-

Fig. 88.

Fig. 89.

For previous views of this patient see figures 86, 87, page 551.

Figure 88, from a housemaid aged 17.

Period of the second acme of pericardial effusion owing to a relapse of pericarditis.

From the fourteenth to the eighteenth day after admission, from the tenth to the fourteenth day after the first acme (figure 86), and from the third to the seventh day after the period of decrease of the effusion illustrated in figure 87. The period of the acme lasted four days.

The explanations of pericardial effusion, prominence and dulness, given with figure 82, at page 544, apply also to this figure.

The pericardial effusion has increased again to a very great extent. The heart is considerably enlarged, and is probably adherent at the apex; its lower boundary is therefore much lower than during the first acme, figure 86, and apparently reaches down to the sixth cartilage, and the middle of the ensiform cartilage. The effusion has increased very much, especially upwards, downwards, and to the right; but owing probably to adhesions at the apex, it has been stationary or has lessened in area at the left side—compared with its amount and area during the period of decrease of the effusion after the first acme shown in figure 87. The effusion extends much higher and more to the right than during the first acme (figure 86), but it is of the same extent at its lower and left boundaries in this as in the first acme. The area of the effusion was much wider in relation to its length, and especially towards the left, in the single acme shown in figure 83, owing to the enlargement of the sac from long-continued distension, than it is in this instance, in which the expansion of the sac to the left has been apparently stopped by the probable adhesion of the apex and front of the left ventricle.

The whole front of the heart and great arteries is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, and the ascending aorta and pulmonary artery.

The region of pericardial dulness (see the black space), corresponding to the pericardial effusion, extends very high, or to within an inch of the episternal notch; far to the right, or nearly two inches to the right of the sternum; low down, or below the tip of the ensiform cartilage; and owing probably to adhesions at the apex, proportionately less far to the left, or fully half an inch to the left of the mammary line. The region of dulness over the arteries is unusually high and narrow. Its width on the first day of the acme was little more than one inch; but it had increased to about two inches on the fourth day, when its upper border was not quite so high as on the first day of the acme at its upper part.

The impulse is extensive but not strong, the double pulsation over the pulmonary artery being felt over the second and third spaces; and the impulse of the heart, over the third, fourth, and fifth spaces, where it extends beyond the nipple (see the curved lines and circles in those spaces). The lower boundary of the impulse has therefore been elevated from the sixth space to the fifth since the period of the decrease of the effusion following the first acme, shown in figure 87; it is, however, lower in this second acme than it was in the first acme, when it occupied the fourth space.

The friction sound (see the zigzag lines, the systolic lines being thick, the diastolic thin) is scarcely audible anywhere without pressure, but with pressure it is heard, double, over the whole region of the pericardial dulness except over the apex and front of the left ventricle, where there are probably adhesions, and where a loud mitral murmur prevails. The rub-

bing sounds are louder over the two lower thirds of the sternum and to each side of it, than higher up.

Figure 89, from the same patient as figures 86, 87, 88.

Period of complete adhesion of the pericardium to the heart.

For pericardial dulness—fifty-two days after admission, thirty-nine to forty-three days after the second acme.

For the impulse—eighty-eight days after admission, when the dulness, tested by post-mortem examination, was about the same as on the fifty-third day after admission.

The region of pericardial dulness (see the black space) is very extensive, measuring about seven inches from left to right, with a slight downward inclination, and nearly five inches from above downwards. Its upper boundary was behind the lower border of the manubrium; its lower boundary, behind the lower end of the ensiform cartilage, the sixth left space and the seventh left rib: its right boundary was situated midway between the right nipple and the edge of the sternum; and its left boundary extended to the sixth and seventh ribs at the outer side of the chest.

The impulse on the fifty-third day was present in the fourth, fifth, and sixth spaces from two inches within, to two inches without, the nipple line, and was quite absent from the sternum and the spaces between the cartilages; since that time the patient has been getting gradually worse; and the impulse has been becoming gradually stronger and more extensive, and is now, on the eighty-ninth day, felt over the whole sternum, the epigastrium, and the cartilages to each side, and on the left side down to the seventh left rib, where it beats against the outer side of the chest (see the curved lines occupying all that region). The impulse heaves up rather slowly during the systole, and immediately after it, falls suddenly backward. The impulse in the first and second spaces, over the pulmonary artery, is double, protruding slightly during the systole, and going back with a flapping rapid movement during the diastole, conveying the impression of a sharp impulse or shock, synchronously with the second sound. Ninety-first day. The impulse is still felt over the sternum, but feebler than two days ago, similar in character, but not felt.

comes again healthy after the attack, the size, position and customary beat of the organ are restored: but in those in whom valvular disease is established, the nature and extent of the disease are made apparent by the force, extent, and position of the impulse. When the resulting mitral disease is severe, the impulse of both the right and left ventricles is extended, and is felt beating from the lower half of the sternum to the left nipple. When, however, the mitral affection is slight, and such as scarcely or not at all to interfere with the function of the organ, then the impulse resumes its natural boundary and strength; and thus the impulse becomes a true measure of the extent of the valvular disease. When both the aortic and mitral valves are affected, the apex-beat and the impulse generally of the left ventricle become more markedly developed, the action of the right ventricle being still unduly strong. In those comparatively rare cases in which the aortic valve is alone affected, the right ventricle is untouched; but the size and force of the left ventricle are increased in exact proportion to the increased labor thrown upon that cavity by the degree of the crippling of the valve. The apex-beat and general shock of the left ventricle become extended outwards beyond the left nipple, and downwards into the sixth space, when the valvular affection is great; but they are held almost within the natural limits when it is slight. When the heart becomes adherent and there is disease of one or more of its valves, the impulse of the organ becomes extended in every direction—to the right, over and beyond the sternum; to the left beyond the line of the nipple; downwards, over the ensiform cartilage, and even below it in the epigastrium; and especially upwards, to the second space and to the adjoining portion of the sternum. In some cases the whole impulse bears at first forwards during the systole, and then drags the walls of the chest in a characteristic manner backwards; while in other cases, in which there is complete fibrous attachment of the adherent pericardium to the sternum, that bone and the adjoining costal cartilages are steadily drawn inwards during the systole, and spring forwards with a shock during the diastole. An essential difference is also established between the influence of respiration on the area of the impulse of the adherent and the non-adherent heart. When the heart is not adherent, a deep inspiration, by drawing down the heart and covering it with the expanded lungs, causes a complete transfer of the impulse from the fourth and fifth spaces to the epigastrium and the sixth and seventh cartilages; but when the heart is adherent, the outspread dragging impulse almost retains its position during a deep inspiration, neither materially lessening its area over its upper borders, nor materially increasing it below. There is, in short, no transfer, such as occurs when there are no adhesions, of the impulse during a deep breath from the intercostal spaces to the ensiform cartilage and epigastrium and the adjoining left costal cartilages. Thus in a patient who has recovered from rheumatic endo-pericar-

ditis we are enabled to judge by the position and force of the impulse, whether the valves, if affected, are seriously or only slightly affected; and, by the extent to which the play of the impulse is influenced by respiration, whether the valvular affection is combined or not with extensive and binding adhesions of the heart.

Vibration or Thrill felt by the Hand over the Region of Pericardial Friction.

A sense of vibration or thrill was felt over the seat of the friction sound at the region of its greatest intensity in fully one-fifth of the patients with rheumatic pericarditis (13 in 63).

In seven of the cases, or more than one-half of them, the thrill was felt over the whole region of the impulse, extending in two instances over the second and third left spaces, in one, over the spaces from the second to the fifth, in three, over those from the third to the fifth, and in one, from the fourth to the sixth spaces.

In two other instances the thrill was confined to the second space, apparently over the pulmonary artery, in three to the region of the apex, and in the remaining case it was present both over the second space and the apex. In all these patients the friction sound was harsh and grating, vibrating, or creaking in character.

In those cases in which the vibration was felt over the whole region of the impulse, the thrill was present at the time of the acme of the effusion, or in one instance two days after it; and the same may be said, with one exception, of those in which the vibration was felt in the second space.

The duration of the thrill was short. It was observed for only one day in seven cases, for two days in three, for three days in two cases, and for four consecutive days in the remaining one. In two cases, the thrill, after being absent from its previous seat over the body of the heart for several days, returned over a limited space when the surfaces were comparatively dry, the effusion having disappeared.

The character of the friction thrill or vibration is peculiar, and differs from the thrill due to altered blood-currents, chiefly in the following points. The blood-thrill presents a succession of equal vibrations, often like those made by the vibrating musical cord; is diffused; has a focus of greatest intensity, from which it lessens and fades away all round; gives the impression to the hand of being deeply seated as well as superficial; begins, when diastolic after the impulse ends, and often continues, when systolic, for a short period after the cessation of the beat of the ventricle; retains its character, position, focus of intensity, and general outspread, unchanged or with only slight modifications from day to day: and finally, has a long previous history pointing to an affection of the heart, and probably dating from an attack of acute rheumatism. The friction thrill or vibration, on the other hand, is shallow, giving a sensation as if it were made just under the hand by the rubbing together of two rough surfaces; has often a grating, rasping, or irregularly vibrating character; presents no focus of intensity, but is spread, with varying force, over the region of the impulse; begins and ends rather abruptly, being limited to the period of the impulse and not passing beyond or preceding it; does not end with an abrupt shock; is short-lived and transient, and, if felt on one or two following days, it always changes in extent, and perhaps in position, and alters in character; and finally has a short previous history of local pain, extended dulness on percussion, increased prominence over the region of the pericardium, and elevated impulse. Sometimes, however, the blood-thrill and the friction-thrill are so much alike that they cannot be distinguished by the hand. The character of the thrill is, however, at once cleared up by the ear; the friction-thrill being accompanied by a friction sound which is in all cases increased by pressure, and is most vibrating, grating, or creaking and harsh at the very seat of the vibration; while the blood-thrill is accompanied by the murmur, usually musical, that distinguishes the valvular affection.

The thrill of presystolic murmur is distinguished by the position of the thrill over and to the left of the interventricular septum, the peculiar large vibrating character of the murmur; the abrupt shock with which the thrill and murmur terminate; the persistency of the thrill, murmur, and shock from day to day; and the long previous history.

The character of the friction sound presented in the various cases a close approximation to the character of the thrill or vibration.

The sensation conveyed to the hand when applied over the seat of thrill in the thirteen cases under examination was not always of the same character. Thus, under these circumstances the hand felt a sense of grating or rasping in two, of vibration in four, and of thrill in seven of the cases.

On listening over the region of the thrill or vibration in these cases a loud harsh friction sound was heard in seven patients, in five of whom the sound was described as being "to and fro;" in five others of them there was a noise resembling the creaking of leather; in three the sound was grating, in one rasping, in two

vibrating, in one grazing, and in one "churning." In several of these cases the friction sound presented, as we have already seen, different phases at different periods of their progress. In all of them the friction sound became less harsh and extensive when the vibration or thrill over the region of the pericardium ceased to be perceptible.

It is to be remarked that when the thrill was perceptible in these cases, especially if it extended over the ventricles, and was not limited to the region of the apex or that of the pulmonary artery, the area of the friction sound was increased as well as the intensity. In one of the cases the rubbing sound was audible over the whole front of the chest, and in several of the patients it spread downwards to the ensiform cartilage and to the left and right seventh and eighth costal cartilage.

The character of the friction sound, associated with the presence of a thrill over the heart and great vessels, whether creaking or grating, vibrating or rustling, or to and fro, will be considered in the next section.

Auscultation.

Position and Character of the Sounds heard over the Heart and Pericardium during the Early Stages of Pericarditis.—In more than one-half of my cases of rheumatic pericarditis (33 in 63), I observed the character of the sounds of the heart at or soon after the commencement of the attack, and before the effusion into the pericardium had arrived at its height. I was frequently surprised by the rapidity with which the affection attained to its acme. In twenty-three of these patients friction sound was heard for the first time before the fluid in the pericardium had reached its greatest amount; and in fifteen of these the rubbing sound was detected only one day, and in four two days before the time of the acme.

Modification of the Sounds of the Heart at the Commencement of Pericarditis, before the Occurrence of Friction Murmur or Friction Sound.—There were five cases in which the sounds of the heart were modified before the occurrence of a friction sound, or the period of the acme. In one of them the heart sounds were muffled two days before the occurrence of the friction sound and the acme; in three of them those sounds were ringing in character from three to four days before the acme; and in one of these the systolic sound was rough and unduly prolonged four days before that period. All the cases of this group but one presented on pressure either a single or double murmur or a rubbing sound subsequently to this modification of the heart sounds, and before the occurrence of the acme.

Position and Character of the Friction Murmur, influenced by Pressure, heard at the Beginning of Pericarditis.—A murmur, which was excited or rendered more intense by pressure, was heard over the region of the heart before the period of the acme of effusion into the pericardium in eight cases.

Pain was felt directly over the seat of the pericardial inflammation in seven of the cases, being excited by pressure on the surface of the chest in three of them. In five of the cases the pain was present at the same time as the appearance of a murmur on pressure, and in two the pain preceded the murmur by a day or two.

In four cases the friction murmur was single and systolic. In four cases a double murmur, excited or intensified by pressure, preceded the friction sound and the acme of pericardial effusion. In the last case of this group, a youth aged 17, a fatal case, the friction murmur prevailed more or less through the whole of the illness until the heart became adherent.

The double friction murmur, heard during the early period of pericarditis, is thus distinguished from the double murmur caused by aortic incompetence, combined as it usually is with mitral regurgitation. It is accompanied, and often preceded, by pain over the heart, usually increased by pressure; it comes into play suddenly; its area is limited to the middle, or lower half of the sternum, and the adjoining left, and, on rare occasions, right cartilages; it is accompanied by the natural heart sounds, but is not rhythmical with them, the heart sounds and murmur being heard as it were side by side; it does not begin with a double accent, or shock, the double accent or shock of the natural heart sounds, but is of equal intensity throughout; it is invariably rendered more intense by pressure, which often converts it into a true to and fro *frottement*, and which always obscures or silences the natural heart sounds. It is not accompanied by marked visible pulsation of the great arteries in the neck, or by the sudden pulse at the wrist of aortic regurgitation, audible when the arm is raised; it is accompanied by extended dulness on percussion over the region of the pericardium; and as a rule it speedily gives place to a friction sound, with which, however, it may coexist, being audible beyond the circumference of the friction sound especially below, and on either side.

In all these respects the double friction murmur contrasts notably with the double aortic murmur; which is not usually accompanied by pain over the heart; does not come into play suddenly; is not limited in its area to the middle or lower

half of the sternum and the adjoining cartilages—but extends also to the upper portion of the sternum and to its right; is rhythmical with the natural heart sounds; commences with a double accent or shock; is not rendered to a material degree more intense by pressure, which never converts it into a friction sound, and which never abolishes the double accent with which the double murmur begins; is accompanied by marked visible pulsation of the carotid and radial arteries, the pulse of the latter becoming audible as a shock when the arm is raised; is not accompanied by extension of dulness over the region of the pericardium; and does not give place suddenly to friction sound, but is persistent.

The single systolic friction murmur is not so easily distinguished from the tricuspid murmur as from other systolic blood murmurs, but their differences are sufficiently marked. The systolic friction murmur is accompanied or preceded by pain over the heart, usually increased by pressure; comes into existence suddenly; is limited usually to the base of the right ventricle, being heard over the middle or lower sternum, or over the fourth left space; is accompanied by the natural first sound, but is not rhythmical with it, the heart sound and the murmur being distinctly heard side by side; does not begin with an accent or shock, the accent or shock of the natural first sound, but begins and ends with a single note of equal intensity throughout; extends rarely beyond the period of the systole into that of the diastole; is usually produced, and invariably rendered more intense by pressure, so that it obscures or masks the natural first sounds; is accompanied by extended dulness on percussion over the region of the pericardium; and speedily gives place to a double friction murmur or a friction sound.

The several systolic blood murmurs may be thus distinguished from the single or systolic friction murmur.

The tricuspid murmur is more likely to be taken for a friction murmur than any other systolic murmur, for it is situated over the front of the right ventricle—over and to the left of the lower half of the sternum—and, like the friction murmur, it is a shallow sound, and it may appear and vanish quickly. It differs, however, in these respects; it is rarely accompanied by pain and tenderness over the heart; is never accompanied by the natural first sound over the right ventricle, for that sound is converted into the murmur; always commences with an accent, the accent or shock of the first sound of the right ventricle; may be intensified, but is not changed in character by pressure, which, however, brings the ear more close to the murmur; is not accompanied by extended dulness on percussion over the pericardium; and does not give place to a double friction murmur or a friction sound.

The systolic mitral murmur is readily distinguished from the friction murmur by the intensity with which it is heard to the left of and below the apex; and its great relative feebleness, or silence over the right ventricle—to the left of the lower portion of the sternum; and by its persistence. When the mitral murmur is audible in the situation just spoken of it is feeble, and is accompanied by the natural sounds of the right ventricle. The heart sounds and the murmur are rhythmical and go well together; and pressure, though it makes the mitral murmur somewhat more clear, does not mask or obliterate the healthy sounds of the right side of the heart.

The direct aortic, and pulmonic systolic murmurs are distinguished at once from the systolic friction murmur by their situation above the level of the third cartilage; the pulmonic murmur, which is often scratching in character, and is therefore apt to be mistaken, when first heard, for a friction sound, being limited to the second left space; and the direct aortic murmur being heard over the upper sternum, and to the right of it, and in the neck over the carotid.

The essential features of difference between the friction murmurs and the blood murmurs are these: The friction murmurs do not begin with an accent, but usually maintain the same tone and pitch throughout; while the blood murmurs begin with an accent or shock: the friction murmurs are intensified and altered by pressure, becoming sometimes rubbing in character; while the valve murmurs are only intensified by pressure: the friction murmur and the natural heart sounds are heard at the same time, but they do not play together or in unison, being audible as it were side by side, each having its own rhythm; and on pressure the friction murmur becomes so loud and even rubbing in character as to mask and extinguish the heart sounds; while the blood murmurs are in perfect accord with the heart sounds: the friction murmurs come suddenly, with pain and increased pericardial dulness, and are transient; the blood murmurs come gradually, without pain or increased dulness, and are permanent.

Friction Sound in Pericarditis before the Occurrence of the Acme of the Effusion into the Pericardium. — Friction sound was heard during the early stage of Pericarditis, in every gradation from a sound scarcely to be distinguished from a murmur up to a grating, vibrating, or creaking noise.

In a few of the cases, the early friction

sound was not audible until pressure was made over the heart. In nearly all the cases, the friction sound was double from the first, but in two, and perhaps three patients the sound was single and systolic when first heard. In a small group of four patients, a smooth or feeble double friction sound, intensified by pressure, came into play from one to four days before the occurrence of the acme of the affection, when the friction sound became louder and more harsh.

In the last great division of cases of pericarditis with friction sound before the acme, the double friction sound, as a rule, was loud and harsh, was intensified by pressure, and set in suddenly; and the effusion into the pericardium speedily attained to its acme after the first observation of the friction. This set of cases divides itself into three groups; in the first group (1), the friction sound became inaudible during the acme; in the second (2), the friction sound became less loud and harsh during the acme; and in the third group (3), the friction sound remained during the acme with little or no change.

(1) In two cases, the friction sound, harsh at the onset, disappeared during the acme. It is difficult to explain the disappearance of the friction sound at the time of the acme of the effusion in these two remarkable cases on physical grounds, but the following circumstances show that it was mainly due to lowering of the power of the heart. It is natural to expect that when the fluid increases, it should interpose itself between a portion of the right ventricle and the anterior wall of the chest, and so limit the area of the friction sound, and lessen its intensity. This will not, however, account for the disappearance of the rubbing sound at the period of the acme, since the impulse was then still perceptible, though higher in position and less forcible.

(2) The second group of this division, in which a loud double friction sound appeared suddenly before the acme of the effusion, and became less loud during the acme, consists of five patients.

The case of this group that I shall relate, is illustrated by the accompanying figures (90, 91, p. 559); during its later stages, by figures 92, 93, 94, pp. 575, 576. A housemaid, aged 20, came in on the fifth day of her illness, the heart sounds being natural. On the third day there was increased dulness on percussion over the region of the heart; and a to-and-fro friction sound over the whole of the region of cardiac dulness, to which it was exactly limited. The impulse was present, as before, in the fifth space, but was higher in position. The dulness and the friction sound extended from the sternum almost to the nipple, and from the third left cartilage to the sixth, but did not pass beyond the sternum to the right, so that the rubbing sound was limited to the right ventricle. It was stronger over the sternum than the cartilages, and became everywhere much harsher on pressure. On the fourth, the double friction sound was heard over the greater part of the sternum, and was audible over the manubrium during the expiration only. The friction sound had somewhat the character of a bellows murmur over the fourth space. It was not quite rhythmical with the sounds of the heart, which were also audible. It was harsher and louder during the systole than the diastole, and was rendered more intense by pressure. On the fifth day, the effusion into the pericardium was at its acme—reaching up to the second space and the manubrium. The impulse was raised from the fifth to the third space. The area of the friction sound was more extensive upwards, but more limited below. It was heard over the whole sternum, being louder over the manubrium on expiration, over the lower portion of that bone on inspiration, and was most harsh and strong over the middle third of the sternum. The rubbing sound was heard from the second to the fourth cartilage, but not apparently below it, and was harsh in the third space. A bellows murmur was audible over the fourth cartilage on the light application of the stethoscope; but when pressure was made, a creaking noise was heard there during the systole, and a rubbing sound during the diastole.

I believe that this group and this case represent the natural progress of the friction sound from the commencement of pericarditis to its acme when the effusion is at its height. During the first blush of inflammation, the surfaces of the heart and the sac are crowded with vessels, but are as yet scarcely coated with lymph. A single or double friction murmur, induced or intensified by pressure, may then be the only sound excited by the rubbing of the heart against the pericardium. Speedily their surfaces become coated with a finely honey-combed rugose covering; and the amount of fluid in the sac increases so as to enlarge the area of dulness over the pericardium, and to expose the whole of the right ventricle and the apex, but neither the right auricle nor the great vessels. The heart is slightly raised and the apex beat ascends from the lower to the higher part of the fifth space. A double friction sound is audible over the whole region of pericardial dulness, to which it is exactly limited, louder and more continuous during the systole than the diastole, and rendered more intense by pressure, which brings into full play both sounds, exciting a to-and-fro rustle or *frou-frou*.

Fig. 90.

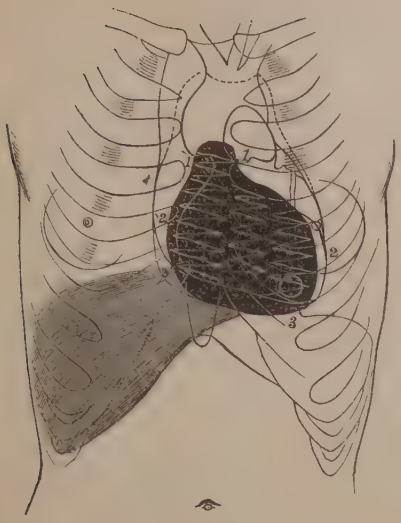


Fig. 91.

Figure 90, from a housemaid aged 20, affected with rheumatic pericarditis.
Early period of the increase of the pericardial effusion.

First day of the friction sound; third day after admission. The sounds of the heart were natural when she was admitted.

The pericardial effusion probably already occupies to some extent the space between the under surface of the heart and the floor of the pericardium, and elevates the heart to a slight degree, and, to a moderate extent, displaces the lungs upwards and to each side; and the centre of the diaphragm, where it forms the base of the pericardium, and the subjacent portions of the liver and stomach downwards. Owing to the displacement of the lungs upwards and to each side from before the heart, the whole of the right ventricle except the upper portion of the conus arteriosus, the inner or left border of the right auricle, and the apex and a portion of the front of the left ventricle are exposed.

Probable region of pericardial dulness on percussion (see the black space). The outlines of the region of pericardial dulness, which is increased in extent, are not described on this occasion, but the extent of the friction sound and the position of the impulse are given; and I have assigned to the region of dulness an outline corresponding to the region of friction sound and the position of the impulse. The region of pericardial dulness has not yet acquired the pyramidal or pear-shaped form that it presents during the acme of the pericardial effusion, but still retains the general form of the healthy region of cardiac dulness, but its outline is considerably enlarged in all directions, and is higher behind the sternum than over the cartilages. It extends across from the right edge of the sternum to the left nipple; its upper boundary probably crosses the sternum on a level with the upper edges of the third costal cartilages, and occupies the third space; and its lower boundary is probably situated a little above the middle of the ensiform cartilage, and the upper edge of the sixth cartilage.

Third day. *The impulse of the heart* is felt at the fifth space below the nipple. (See the circle in that space.)

Fourth day. The impulse is feeble, being slightly perceptible below the nipple.

Friction sound (see the zigzag lines, systolic thick, diastolic thin). Third day. A loud but soft to-and-fro friction sound is heard over the sternum from below the manubrium to its lower end, and up to but not beyond its right border; and over the fourth and fifth cartilages and intermediate spaces, where it extends almost, but not quite to the nipple, where it is feebler than it is over the sternum. The friction sound is rendered much harsher by pressure. Fourth day. The friction sound is nearly the same in extent, character, and area as it was yesterday, but it is now audible over the manubrium during expiration; it is lower and louder below during inspiration than expiration; and it is louder generally during the systole than the diastole.

Figure 91 from the same patient as figure 92, affected with rheumatic pericarditis.

Period of the first acme of pericardial effusion. Third day of the friction sound and of the increase of pericardial dulness, fifth day after admission.

The explanations of pericardial effusion and dulness given with figure 82, page 544, apply also to this figure.

The pericardial effusion completely distends the sac, which is pyramidal or pear-shaped, as in figures 80, p. 540; 82, 83, p. 544; 86, p. 551; 88, p. 553. The extent of the effusion,

and of the displacement upwards and to each side of the lungs, and downwards of the diaphragm, liver, and stomach may be inferred from the description given below of the extent of the region of pericardial dulness on percussion. The whole front of the heart is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the pulmonary artery, and the ascending aorta within the pericardium, owing to the extensive displacement of the lungs from before those parts.

The region of pericardial dulness (see the black space) on percussion is pyramidal or pear-shaped, like the distended pericardium. The upper and narrower region of dulness over the great vessels (1, 1) is situated behind and below the lower half of the manubrium and extends a little way into the adjoining first and second spaces; the larger portion of pericardial dulness, which includes the heart itself and the volume of fluid effused into the space between its under surface and the floor of the pericardium (2, 2; 3, 3), extends from the second space down to the lower border of the sixth cartilage, and almost to the end of the ensiform cartilage, and from an inch to the right of the sternum to about half an inch to the left of the nipple. The lower boundary of the heart (2, 2) is probably situated behind the lower border of the fifth cartilage; and the heart (2, 2) extends from this boundary up to the third cartilages: and the volume of effused fluid between the under surface of the heart and the floor of the epicardium extends from the lower boundary of the heart down into the epigastric space, almost to the end of the ensiform cartilage, and the lower edge of the sixth left cartilage.

The impulse has been elevated from the fifth to the third space, and extends outwards to the nipple line. (See the concentric curves in that space.)

The friction sound (see the zigzag lines—systolic thick, diastolic thin) is double, and extends from the nipple to the lower end of the sternum. It is most harsh about the middle of the sternum, and is louder at the upper end of that bone during expiration, and at its lower end during inspiration; and is more intense during the systole than the diastole. The frottement is also audible over the left second, third, and fourth cartilages; and is soft without pressure, but with pressure it is creaking over the fourth cartilage.

A mitral murmur is audible at the apex.

For the later views of this case see figures 92, 93, 94, pp. 575, 576.

When the effusion has increased to its utmost limits, the heart is elevated, its impulse being raised from the fifth to the fourth or third space; the increased effusion displaces the lungs and so exposes the whole surface of the heart and great vessels; and depresses the central tendon of the diaphragm downwards towards the abdomen, fluid being alone present below the fourth space. The whole region of actual friction is shifted upwards, and with it the whole region of the friction sound; which is no longer audible below the fourth or fifth cartilage, but spreads over the right auricle and the left ventricle, as well as the right ventricle; and upwards over the great vessels and to the top of the sternum. The friction sound silenced below is intensified and extended above; so that there is a transfer upwards of the friction sound; while the dulness on percussion increases in all directions, upwards as well as downwards.

Four cases differed from the rest in this, that while the friction sound spread upwards at the time of the acme, it also either increased downwards, or, retaining its hold below, increased extensively to the left side.

The comparative relative Area and Intensity of the Friction Sound just before,[1] *and during the Acme of the Effusion into the Pericardium.*—In twenty-nine cases the comparative area and intensity of the friction sound were observed both before, and at the time when, the effusion into the pericardium was at its height.

Area.—When the effusion into the pericardium increases, the heart is raised, and the lungs are displaced upwards, and to the left and right by the increased fulness of the sac and the greater elevation of the heart itself; for the organ is then pushed upwards from a wider into a narrower space. It is natural to expect that, under these circumstances, the area of the friction sound should steadily increase upwards and to each side with the increase of the area of pericardial dulness. This was found to be so in the great majority of instances. Thus the area of friction sound was greater at the time of the acme than before it in twenty out of the twenty-nine cases; while it was less under the same circumstances in only two of them. In six patients, the area of the friction sound was equal before and during the acme; and in one case the friction sound was absent before, but present at the time of the height of the disease.

These clinical facts show that when the curtain of lung in front of the heart and great vessels is displaced by the distended pericardium and the elevated heart, the friction sound spreads upwards, and to the right and left; so as to be audible over the whole front of the right ventricle, the great vessels, the right auricle, and the apex.

The lower boundary of the friction sound, while it retains its place, at the time of the height of the effusion, becomes softened in character. The focus of intensity of the rubbing sound is shifted

[1] At the time of the last observation, made before the effusion had reached its height.

upwards, with the upward shifting of the heart and its impulse; and the intensity of the sound is toned and graduated downwards, from the seat of its focus to that of its inferior limit.

Intensity.—In nearly three-fifths (16 in 29) of the cases, the friction sound was more intense; and in fully one-third of them (10 in 29), it was less intense, when the effusion into the pericardium was at its height, than just before that time. The tendency, then, is for the friction sound to increase both in intensity and area, during the acme. The exceptions to this rule are, however, much more frequent as regards intensity than area; for the area lessened at the time of the acme, in only two instances, while the intensity did so at that time, in ten instances out of twenty-nine.

The area of the friction sound, then, is, as a rule, larger, and its intensity greater at the time of the acme of the pericardial effusion, than at that of the last previous observation, made from one to two days before the acme. The exceptions to this rule are rare as regards the area, but rather frequent as regards the intensity of the friction sound, which is greater in one-third of the cases on the day before, than at the time of the acme. The change, both in area and intensity, is often notably rapid and great; the character of the friction sound being sometimes altogether altered, and its area remarkably enlarged in the course of one or two days.

The Character and Area of the Friction Sound at the Time of the Acme of the Effusion into the Pericardium.—The friction sound, audible over the region of the heart and arteries and the pericardium during the acme of the pericardial effusion, presented great variety of character, intensity, and area in the forty-four cases under examination. I. In nine of those cases the friction sound was accompanied during the acme by a thrill over the region of the heart and great vessels; and II. in thirty-five of them the presence of a thrill was not observed. I. Of the nine cases with a thrill, (1) in five a sound resembling the creaking of new leather; (2) in one a grating sound; and (3) in three a harsh friction sound was respectively audible over the region of the pericardium. II. Of the thirty-five cases in which a thrill was not observed, (1) in seven a creaking sound was heard; (2) in two the sound was grating in character; (3) in fifteen a definite friction sound, intensified by pressure, which in two instances excited a creaking noise, usually harsh, but sometimes not so, was audible; (4) in five the friction sound was soft in character, but was rendered harsh or more intense by pressure, except in one instance, in which pressure was not employed; (5) in four a friction sound, previously absent, came into play when pressure was made over the region of the heart; (6) in one friction sound, present during one, was absent during two of the three days during which the acme lasted; and finally (7) in the remaining case a double friction murmur, intensified by pressure, was audible over the region of the pericardium during the acme.

I.—*Cases with Thrill and* (1) *a Creaking*, (2) *Grating*, *or* (3) *Harsh Friction Sound over the Heart.*—In nine of the forty-three cases under review, a systolic thrill was felt over the heart, and (1) in five of those cases a creaking; (2) in one of them a grating; and (3) in three of them a harsh friction sound was audible at the seat of thrill at the time of the height or acme of the disease. In six of these cases the thrill was present over the right ventricle, and, in some of those, but not in all, it was probably situated over the left ventricle also; in another of them it was present over the apex and the second space, but not over the right ventricle; in one of the two remaining cases it was felt over the apex; and in the other one over the second space alone.

(1) *Creaking Friction Sound.*—In three of the cases with a thrill over the right ventricle, and in one of those with a thrill over the apex alone, a creaking sound was audible over the seat of thrill.

One of these patients, a man aged 27, came in with extensive pericardial dulness; a thrill over the right ventricle extending from the fourth left cartilage to the sixth; a loud systolic creaking friction sound consisting of five vibrations, the diastolic sound being much smoother than the systolic, over the seat of the thrill; and a double *frottement* extending widely over the front of the chest from the second cartilage down to the ninth on both sides, and audible at the epigastric space. The pericardial dulness on that day extended upwards to the third space, and on the following day to the third cartilage, when it reached its greatest height. The region of thrill had increased upwards, and extended from the third cartilage to the sixth. A creaking sound was audible apparently over the whole seat of the thrill, but over the fifth cartilage there was a vibrating, grating, systolic friction sound of a churning character, which was creaking towards the end of the systole, the diastolic sound being short and smooth.

(2) *Grating Friction Sound.*—A grating friction sound without a creak was present on the presumed day of the acme in one case.

(3) *Harsh Friction Sound.*—A harsh friction sound was present with a thrill in three cases. One of these cases, a girl, aged 17, came in with an extensive impulse, a double thrill, and a loud, double

scraping sound over, but not below, the heart. On the second day, there was less dulness, and no note of thrill, and the friction sound was less harsh and extensive: but, on the third day, there was less effusion, the impulse was lower and more diffused, and the friction sound was much more intense and extensive.

We may, I think say, on reviewing these cases, that at the time of the acme of the disease, when a thrill is present over the right ventricle, a creaking noise is audible over the seat of the thrill; and that from this noise, as from a focus, a to-and-fro sound radiates in all directions over the front of the chest, reaching far beyond the limits of the region of actual friction, becoming more feeble towards its outlying margins, and spreading almost up to the clavicles, out to or beyond the nipples, and down to the eighth or ninth cartilages; and that when the effusion lessens and the thrill disappears, the creak vanishes, and the friction sound softens, and limits its area to the region of actual friction, being bounded below by the sixth cartilage. The reason for the great extension during the acme of the friction sound upwards, outwards, and downwards beyond the region of actual friction in these cases is obvious. The heart, surrounded by the distended pericardial sac, is displaced upwards into the higher and narrower portion of the cone of the chest. It works in a confined space, and rubs with its roughened surface against the roughened surface of the pericardium; and the lungs being pushed aside, it presses against the sternum and cartilages, and excites vibrations and a creaking or grating friction sound over the walls of the chest in front of the heart. The play of the two roughened surfaces of the pericardium upon each other induces vibrations, sensible to the hand, that excite consonant vibrations in the superimposed sternum and cartilages; and these parts, acting as a sounding-board, transmit the sound to a distance over the front of the cage of the chest in all directions, and especially downwards. When the thrill is limited during the acme to the second space, over the pulmonary artery, or to the apex of the heart, or is felt both over the apex and the second space, the creaking or grating noise is limited to the seat of the thrill; and the friction sound does not extend beyond the region of actual friction, excepting perhaps to a small extent over the circuit of the apex. When in such a case the effusion lessens, the heart descends, and the thrill disappears, the friction sound may spread downwards, so as to reach the eighth cartilage.

II.—*Cases in which a Thrill was not observed over the Region of the Heart or Great Vessels.*

(1) *Cases in which a Sound like the Creaking of New Leather was audible at the time of the Acme of the Effusion, no Thrill being present.*—In seven of the forty-four cases under examination, a creaking noise, usually systolic, was heard without a thrill at the seat of the impulse of the heart at the time of the acme.

In all of these cases, and in several of those in which a thrill over the heart was accompanied by a creaking or grating noise, as soon as the fluid in the pericardium lessened and the heart descended, the creaking noise was replaced by a comparatively smooth friction-sound. This occurred on the day after the acme of the effusion in four of the seven cases. This sudden disappearance of the creaking noise with the diminution of the fluid and the descent of the heart, appears to me to show that the presence or absence of the creaking noise depended more on the position of the heart and on the degree and kind of pressure exerted by it during its contraction; than on the character of the roughened coat of lymph covering the heart and lining the pericardial sac, since that lining cannot have changed materially in one day when the disease was at its height. At the time of the acme of the effusion into the pericardium, the heart is elevated so as to occupy the upper and narrower part of the cone of the chest; and beats with force in its contracted space against the cartilages and sternum which confine its movements. When the heart pulsates thus against the walls of the chest, the movements of the former are resisted by the pressure of the latter. The accumulated force of the heart overcomes the resistance of the walls of the chest, and the accumulated resistance of those walls then overcomes the force of the heart; these two opposite forces by turns arrest and overcome each other and give rise to a series of fine jerks or vibrations that may give birth to a thrill, and a vibrating creaking noise. In one case, this creaking noise consisted of five distinct vibrations; and such a succession of vibrations forms, indeed, the essential nature of the thrill and its attendant creaking sound.

The creaking sound, and the main varieties of friction sound, may be imitated by rubbing the forefinger on the thumb with varying degrees of force when the back of the thumb rests upon the ear. When the finger and thumb rub gently or with moderate force upon each other, to and fro, the rubbing sound is smooth or harsh in proportion to the gentleness or force employed. When, however, the pressure exerted by the finger on the thumb is great, the resistance to their onward movement on each other causes them to stop in a succession of jerks, which produce a creaking noise.

When the fluid decreases, the heart descends into the ampler space of the chest; the organ moves with freedom; and, as it no longer presses with a resisted force against the walls of the chest, the thrill, vibrations, and creak give place to a moderated friction sound; which may be so harsh as to sound like the rubbing of sand-paper; or so soft as to resemble a murmur.

(2) *Vibrating, Grating Friction Sound.*—The grating, vibrating friction sound ranks next to the creaking noise in intensity. It is, in fact, a sister-sound to the creaking noise, with which it is closely allied. Thus, it may be audible when there is a thrill, when it may be heard alone, or associated with a creak; or it may by pressure be converted into a creak; or it may precede or follow, displace or be displaced, by that sound; or it may, like it, be produced by pressure. The grating sound, like the creaking sound, is the combined effect of pressure and friction, but the pressure is usually less, while the rubbing surfaces are, I believe, more invariably rough, when the sound is grating than when it is creaking. A grating sound was audible during the acme of effusion in two or three cases in which there was a thrill, and in two in which there was no thrill; and it was excited by pressure in two. It was, therefore, observed in one-seventh of the cases (6 or 7 in 44).

We have already seen that the creaking sound is usually single, but it is the reverse with the grating sound, which is usually double. The grating friction sound is a jarring, grating, vibrating noise, rough and to-and-fro in character, made in a succession of jerks, each jerk being separately audible, and varying slightly, and the whole series not combining to form one note like the creaking sound, but, as I have just said, a jarring, grating, vibrating noise. I made out, as I have already stated, that in one case the creak was composed of five vibrations, or at the rate of twenty-two vibrations in a second; but, as I took no special note of it, I do not know what number of vibrations were made in a second by the grating noise. I believe, as I have already hinted, that the grating noise is always associated with the rubbing of the two harsh and roughened surfaces of the heart and pericardium upon each other, but I have no direct proof of this at present.

(3) *Harsh To-and-Fro Friction Sound, intensified by Pressure, at the Time of the Acme of the Pericardial Effusion.*—Résumé, *including the whole of the preceding cases, whether with or without a thrill.*—We have just seen that a creaking noise, usually systolic, was present over the heart at the time of the acme of the disease in one-fourth of the cases in which the dulness was observed at or about the period when the effusion was at its height (12 in 44); while in four other cases it was then excited by pressure, and in two it was heard just before the acme of the effusion. Creaking, therefore, was present as a primary sound in twelve cases; as a secondary sound, or from pressure, in four cases; and in two others it was audible just before the acme. We have also seen that a grating friction sound, usually double, was present over the heart when the effusion was at or about its height, as a primary sound in three cases in which there was no creaking, and in one or more in which there was creaking; and as a secondary sound in two in which it was excited by pressure; while in four others it was present just before or after the period of the acme of the disease.

If we combine the two sounds, we find that during the acme the creaking and grating sounds were primary in fifteen cases, and secondary, or excited by pressure, in six; while they were associated with each other in one or more. Besides these fifteen cases, in which creaking or grating sounds were primary, there were nineteen cases in which there was a definite friction sound, which was usually harsh; in all of these it was double, or to-and-fro in character, being audible both during the systole and the diastole of the ventricle, and in all but two it was intensified by pressure. Three-fourths, therefore, of the patients (34 in 44) in whom the pericardial dulness was observed when at or near its height presented either a systolic creaking noise, or a double grating, or a definite to-and-fro friction sound, usually harsh in character.

Besides the nineteen cases in which there was a double *frottement*, usually harsh, at the time of the acme; there were seven cases in which that sound was associated with a creaking noise; and in one it accompanied a grating noise. In these cases the creaking or grating noise was limited to that part of the right ventricle, or the apex, that was pressing with the greatest force upon the costal cartilages or sternum, while the double *frottement* pervaded and often overstepped the rest of the heart and the great vessels.

If we group together the eight cases with harsh double *frottement*, in seven of which the *frottement* was associated with a creaking sound and in one with a grating noise, and the nineteen cases not so associated, we find that in one-half of those twenty-seven cases the character of the sound is definitely specified (13 in 27); while in twelve it is described as a harsh double friction sound; and in two as a to-and-fro sound.

Of the thirteen cases in which the character of the double sound was specified, in four it was described as being like that made by rubbing with sand-paper; in

seven as being either rasping, or musical planing, scraping, scratching, grazing or rustling, the latter sound being a genuine *frou-frou;* while in the remaining two the sound resembled that made by sharpening a scythe.

In the whole of the twenty-seven cases except two, pressure with the stethoscope intensified the double *frottement;* it sometimes altered or modified the character of the sound; and in five instances it transformed the double *frottement* into a creaking sound. When the creaking sound was thus brought into birth by pressure, or secondary, it was usually double; but when the creak was always present, or primary, it was, as I have already shown, usually and essentially single or systolic.

In all these cases the double *frottement* was essentially a to-and-fro sound. The character and volume of the sound, and the relative intensity of the to-and-fro, or the systolic and diastolic friction sounds, varied over the different parts of the heart. As a leading principle, the greater the pressure exerted by the heart, or any portion of it, during its action upon the cartilages or sternum against which it beat, the more intense was the friction sound.

The friction sound in the remaining cases of this group was limited to a comparatively small area.

In two of the nineteen cases, in both of which there was a thrill over the right ventricle, the rubbing noise, as I have already stated, extended over the front of the chest, far beyond the region of actual friction. These two cases, however, stand apart, for in the remaining seventeen the area of the friction sound was limited to the region of actual friction; with, however, this slight exception, that in six of the patients the to-and-fro sound spread upwards to the top of the sternum, and in one of them it was diffused outwards as far as the left armpit. The upper limit of the distended pericardial sac and of actual friction is rarely higher than the transverse centre of the manubrium, which is about an inch below the top of the sternum; therefore in the six patients just spoken of, the friction sound extended itself upwards for from an inch to fully two inches above the actual seat of friction over the great vessels, which, at their higher portion, are partly covered by lung.

The explanation of this extension of the friction sound upwards beyond the immediate seat of friction is the same as that of the diffusion of the friction sound over the front of the chest far beyond the region of the distended pericardium and of actual friction, when a thrill and a corresponding creaking noise are present over the heart. The to-and-fro movements of the heart upon the pericardial sac, both being covered with lymph, excite a to-and-fro sound which is audible over the region of those movements. The vibrations that produce the sound are communicated to the sternum, which is played upon by the rubbing surfaces; and the sternum, which acts as a sounding-board, propagates the sound to its own upper end, which is at some distance from the seat of the parent vibrations. The extension of the friction sound beyond the region of actual friction depends on the loudness and intensity of the rubbing noise, and the force with which the heart when it is rubbing to-and-fro, presses against the sternum and cartilages. Of the six cases in which the to-and-fro sound mounted to the top of the sternum, in three there was a creaking sound over the heart, with a thrill also in two of those; in two others a creaking sound was excited by pressure; and in the remaining one a loud, harsh, double friction sound was present over the region of the pericardium. Although a creaking friction sound was audible over the apex in four instances, in only one of them did the to-and-fro sound spread to the left beyond the apex, but in that one the rubbing sound extended outwards into the left armpit. In that case there was dulness over the left lower lobe, and bronchial breathing between the left axilla and the spine. It is, therefore, evident that the heart and pericardium were displaced towards the left side owing to the condensation of the left lung, and that this circumstance facilitated the extension of the friction sound to the left axilla.

With these few exceptions, the region of friction sound coincided in these cases with the actual region of friction at the time when the effusion into the pericardium was at its height.

(4) *Cases in which a Soft Friction Sound, audible over the Heart at the Time of the Acme of the Effusion into the Pericardium, was converted by Pressure into a Harsh Rubbing Noise.*—Four cases with a soft friction sound, in which pressure rendered the sound harsh, come under this heading, and in one of these the friction sound elicited by pressure resembled the noise made by sharpening a scythe. In a fifth case, with a similar friction sound, the pressure test was not employed.

In these four cases a comparatively soft double friction sound was intensified and altered by pressure, becoming converted in one instance into a sound like that made by sharpening a scythe, and in one into a rasping, grating noise. Here pressure expelled any interposed fluid; brought the opposite roughened surfaces of the pericardium more closely into contact; and aroused counter-pressure on the part of the heart against the cartilages and sternum during its to-and-fro rubbing movements. These effects spoke

out not only in a louder and more diffused, but also in an altogether altered sound; so that the soft sounds, sometimes so murmur-like as to be almost doubtful in quality, became instantly transformed into a loud double and broken noise, like that made by sharpening a scythe, or into a rasping, grating, almost creaking sound.

(5) *Cases in which a Double Friction Sound, not otherwise audible, came into play when Pressure was made over the Heart during the time of the Acme of Pericardial Effusion.*—In four cases during the acme, on listening without making pressure, the healthy sounds of the heart were alone audible; but on making pressure those sounds were either replaced or accompanied by a double rubbing noise.

In three of these cases, at the time the effusion into the pericardium was at its height, when the stethoscope was applied lightly over the heart, the natural heart sounds were alone heard, friction sounds being everywhere inaudible. When, however, pressure was made with the stethoscope, a double friction sound was immediately brought into play, which could be suspended or renewed at will by withdrawing or replacing the pressure. In one case the friction sound thus generated was limited to the region of the right ventricle, and in another to the base of that ventricle; but in a third case it was diffused over the whole space occupied by the heart and great vessels. The impulse was feeble in one of these patients, and was felt over the right ventricle in another. It is difficult to say why friction sound was absent without pressure over the seat of the impulse; but it is self-evident that if we press the cartilages or sternum inwards upon the walls of the heart moving to and fro, those walls will work with increased counter pressure against the resisting walls of the chest; and may thus elicit a friction sound when previously absent, or intensify a friction sound already existing, owing to the increased friction of the two roughened surfaces. In two of the cases a to-and-fro sound was audible without pressure over the apex, and in one of them over the lower border of the right ventricle also; but it was brought into play by pressure over the whole region of the heart and great vessels.

The subsequent history of these cases illustrates with great clearness the cause of the absence of friction sound without pressure, and its presence with pressure during the acme of the disease. In three of them, as soon as the effusion into the pericardium lessened, the heart descended, and its impulse became stronger and lower; the fluid interposed between the front of the heart at its lower border and the pericardial sac disappeared; and the friction sound came into spontaneous play where it was before absent without pressure. That sound, indeed, gradually augmented in loudness and intensity, and increased in area upwards, sideways, and especially downwards.

(6) *Case in which Friction Sound was Absent for Two of the Three Days during which the Acme of Pericardial Effusion lasted.*—This patient, a woman aged 21, came in with great pain and a double friction sound all over the region of the heart. The pain was relieved by leeches. Next morning the effusion was at its height, but the friction sound had vanished and could not be brought back even by pressure. That evening there was a return of pain, and a renewal of the friction sound which lasted until next day, but again vanished on the fourth day, when the effusion was still at its acme. She was in great distress from pain over the heart, but the impulse was faint in the third and fourth spaces. Next day there was less effusion, a lowered impulse, and no distress, and friction sound was rendered audible by moderate pressure over the right ventricle. Why was the friction sound absent in this case of pericarditis? When we consider that the impulse was perceptible, it must be allowed that the answer is difficult. The loss of blood on the second day and the great distress on the fourth day may in some measure, however, account for the exit of the friction sound.

(7) *Case in which a Friction Murmur was audible over the Heart at the Time of the Acme of the Disease.*—This case of a youth æt. 17, presented a long history, and proved fatal on the forty-eighth day. On examination after death, the heart was found to be universally adherent by means of recent lymph. Throughout the whole period, with rare and doubtful exceptions, the inflammation of the pericardium was made evident, not by the ordinary friction sound, but by a true friction murmur.

The Area of the Friction Sound during the Acme of the Effusion.—The area of the friction sound when the effusion into the pericardium is at its height may, on the one hand, be so extensive as to cover the whole front of the chest, extending from the clavicles down to the ninth right and left costal cartilages; or, on the other, be so limited as to be confined to the middle or lower portion of the sternum. This great diffusion, or narrow limitation of the friction sound at the period of the acme of the disease, is, however, comparatively rare; and, as a rule, the area of the friction sound corresponds either with the area of actual friction, or with that of dulness on percussion over the pericardium.

The friction sound was audible over a great extent in all those cases, amounting

to nine, in which a thrill was felt over the heart or great vessels, and especially in those in which it was perceptible over the front of the right ventricle.

In all the cases with thrill the friction sound was audible over the right auricle and ventricle, the outlying portion and apex of the left ventricle, and the great vessels; in all but one of them, also, it extended to the top of the sternum, beyond the region of the distended pericardium over the great vessels. In these cases, as I have already explained, the friction sound was most intense over the region of the thrill, and it radiated thence over a wide area, becoming gradually less intense from its focus to its extreme limits, being conducted by the sternum and cartilages acting as a sounding-board.

In six of the twelve cases in which a creaking sound was heard over the heart, the area of the friction sound extended down to the seventh, eighth, or ninth costal cartilages; but in five of these the creak accompanied a thrill. In the remaining six cases the frottement extended to the sixth cartilage, or occupied an unspecified space to the right and left of the sternum. It is evident, therefore, that the great diffusion of the sound in these cases was due more to the thrill, than to the creaking sound that was audible at the seat of the parent thrill.

I need not here specify the exact limits of the friction sound in the remaining cases.

These clinical facts show that, when the effusion into the pericardium is at its height, if we put out of view those cases in which a thrill is felt over the right ventricle, the friction sound is, with a slight exception, practically limited to the region of pericardial dulness, or rather of the heart and great arteries. This exception applies to the presence of the friction sound over the upper end of the sternum, which is fully an inch higher than the uppermost limit of that region. This was observed in nineteen cases, and in ten of these no thrill was noticed over the region of the heart or great vessels. In all these cases the friction sound was conducted to the top of the manubrium, from the actual seat of friction by the sternum itself, acting as a sounding-board.

When the lower boundary of the friction sound reaches to the lower end of the sternum and the sixth cartilage, that limit is still within the lower boundary of the region of pericardial dulness, which is situated, when the pericardium is completely distended, behind the ensiform cartilage and along the lower margin of the sixth cartilage. As I have already shown, however, the lower boundary of the heart, and consequently of the region of actual friction, is, in the great majority of cases, above the lower end of the sternum and the sixth cartilage; for the fluid in the pericardium presses the heart upwards, and interposes itself between the lower border of the heart and the walls of the chest in front of that border. The position of the impulse is a good practical test of the position of the actual seat of friction. In three of the seven cases in which the friction sound was audible as low as from the seventh to the ninth cartilages, the impulse was felt in the fifth space, and in one of them, a case of established valvular disease with enlarged heart, in the sixth space. But with one single exception, in which the beat of the heart was felt in the fifth space, in all the rest of the cases the impulse was not present below the fourth space, and in nine instances its lowest position was in the third space. In the nature of things, the seat of the actual friction behind the sternum, except at its upper portion, corresponded, as a rule, pretty closely with its seat at the intercostal spaces.

In all the cases save one the friction sound was audible down to the lower end of the sternum at the time of the height of the effusion, and in twelve of them it was heard over the sixth cartilage. In all these cases, therefore, it is evident that the friction sound was audible below the actual seat of friction. The sternum is an excellent sounding-board, and the conduction of the friction sound to the lower end of that bone, by its own resonant vibrations, at once explains the presence of the sound at its lower end. The presence of the *frottement* over the sixth cartilage, an inch below the actual seat of friction, appears to me to call for a different explanation. The observed facts are, indeed, different in these two cases. The sound heard at the lower end of the sternum is, like that at its upper end, usually of the same harsh to-and-fro quality, and of about the same intensity as that audible over the two rubbing surfaces at the middle of the bone. But this, as a rule, is not so with regard to the friction sound audible over the sixth cartilage, for that is softer, smoother, and less loud than the sound over the seat of the impulse, from one to two spaces higher up. The presence of the soft muscular space cuts off the direct connection between the fifth cartilage and the sixth. The sixth cartilage is, however, directly attached to the sternum, and that bone, acting as a sounding-board, doubtless conveys some of its own resonant vibrations to the cartilage. But it is to be noted that the sound over the fifth space, though softer and feebler than that over the fourth space, is harsher and louder than that over the sixth cartilage. It is self-evident that the sound over the space can scarcely be conducted from the sternum; and I think, therefore, that we

must look to the fluid within the pericardium, and to the inner surface and structure of the roughened and thickened pericardium itself, as the principal media by which the sound is conducted in these cases to the sixth cartilage.

If we except those cases in which a thrill is felt over the right ventricle or at the apex, we find that when the sac is filled with fluid the friction sound stops quite suddenly along the left and right margins of the region of dulness over the pericardium. This sudden arrest of the rubbing sound at its outer border is less marked along the right than the left margin. This is, I consider, explained, firstly, by the softer, smoother, and more equal character of the to-and-fro sound over the right auricle than over the right and left ventricles; and, secondly, by the presence of fluid between the compressed right auricle and the walls of the chest in front of it, along its outer border. If, on the other hand, we look at the left border of the distended pericardium, we find that there the solid ventricles by their own pressure and action against the ribs and spaces, displace the fluid and completely occupy the ground. Here we pass suddenly from the loud double *frottement* made by the two rubbing solid surfaces of the ventricle and the rib lined with roughened pericardium, to the silent, soft, non-conducting surface of the lung.

We may, I think, conclude, with the qualifications just stated, that when the effusion is at its height, as well as when it is increasing in quantity, the friction sound is limited to the region of pericardial dulness; and, though with less rigor, to the region of actual friction; and that the law originally stated by Dr. Stokes, that the area of the friction sound is usually limited to the region of the heart, is correct in the great majority of cases, during the period of the acme of the effusion.

Before concluding what I have to state with regard to the area of the friction sound, I would here estimate, as nearly as I can, the extent to which the sound was heard over the various chambers of the heart and the great vessels during the acme of the pericardial effusion in the forty-four cases now under examination.

In one-half of the cases (21 in 44) the friction sound was audible over the whole front of the heart, including the right auricle and ventricle, the apex and a portion of the left ventricle, and the great vessels. In seven or eight other cases it was heard over the right auricle and ventricle, in four or five of which it was also present over the apex, and in one over the great vessels. In fifteen other cases the *frottement* was audible over the right ventricle, in nine of which it was also heard over the apex, and in four or five over the great vessels. In six of these cases the friction sound was limited to the right ventricle. If, upon this estimate, we take each portion of the heart separately, we find that the friction sound was present during the acme over the right ventricle in the whole of the forty-four cases under notice; over the apex of the left ventricle in thirty-four or perhaps thirty-five of those cases; over the right auricle in twenty-eight or twenty-nine of them; and over the great vessels in twenty-six or twenty-seven of them.

Intensity and Character of the Friction Sound over the different parts of the Heart and Great Vessels during the Acme of the Effusion.—When inquiring into the relative intensity and character of the friction sound over the different cavities of the heart, except the right ventricle, and the great vessels at the time of the acme of the effusion, I shall take into account the forty-four cases now under examination; but as regards the right ventricle I shall limit myself to the twelve cases with primary creaking sound, the two with grating friction sound, and the nineteen cases in which there was a harsh friction sound intensified by pressure, which form a total of thirty-three cases. Although the left ventricle forms the pivot of the heart's action, and does its work with threefold more power than the right ventricle, I shall first examine the friction sound as it presented itself over the right ventricle, because it forms the front of the heart; covers the left ventricle except at its left border and apex; and is the main seat of actual friction.

Right Ventricle.—As the right ventricle forms the front of the heart, it is always in contact to a greater or less degree with the anterior walls of the chest. Owing to the distension of the pericardium during the acme, and the elevation of the heart into the contracted space at the upper part of the chest, the heart and great vessels are stripped of the lung that covered them, and press directly forward upon the middle and upper part of the sternum and the higher costal cartilages and intercostal spaces, from the second to the fifth.

The to-and-fro movements of the right ventricle, by rubbing against the opposed surface of the sac, give birth to the to-and-fro friction sound audible in front of the ventricle. Those movements play from right to left during the contraction of the ventricle, and from left to right during its dilatation (see Figs. 62, 63, p. 401). The sweep of the walls is very extensive behind the sternum, at the junction of the auricle to the ventricle; thence it gradually lessens; and comes to a stand-still near the septum. The friction movements are therefore greater, and the friction sounds are louder, at the sternal than the costal halves of the cartilages. As the

position of the ventricle is raised from the fourth and fifth spaces to the third and fourth spaces, the *frottement* is usually louder over the sternal portions of those spaces, and the adjoining portion of the sternum, than elsewhere.

As the movements made during the emptying of the ventricles are active, and those made during the filling of the ventricle are passive, the increased pressure made by it upon the cartilage and sternum during the systole often intensifies the *frottement*, and, as I have already shown, may even transform it into a creaking noise. Thus, of the thirty-three cases under examination, in six there was a systolic creak over the right ventricle; in thirteen the systolic friction sound was louder than the diastolic; in two the systolic and diastolic sounds were equal; and in twelve it is not stated whether there was any difference between the two sounds.

From these clinical facts it is evident that the active friction sound made during the contraction of the ventricle is, as a rule, louder than the passive friction sound made during its dilatation. In a small minority of cases, however, the two sounds are equal, and a true to-and-fro sound is produced, the diastolic portion of which speaks with the same intensity, length, and continuousness as the systolic portion. In these cases I believe that the impulse is feeble, and that the systolic friction sound, like the diastolic, is, so to speak, passive, and is not intensified by the greater pressure from within of the anterior wall of the ventricle upon the walls of the chest.

The *conus arteriosus* of the right ventricle calls for special notice. It is situated behind the third space and the two adjoining cartilages, and as it enjoys extensive play during the systole, when its movements are twofold, from above downwards, and from left to right, the friction sound is often notably harsh, loud, and to-and-fro in that situation. Sometimes it is there creaking or grating, when it may be accompanied by a thrill. It sometimes resembles the sound made by rubbing together two opposite surfaces of emery paper, of stuff or of silk; or it is rasping, or scratching, or rustling when it may present a true *frou-frou;* or it may, though less frequently, be soft in character. A friction murmur is, however, rarely or never present in this situation. Pressure readily intensifies and alters the friction sound over the conus arteriosus, and sometimes converts it into a creaking sound. As the conus arteriosus is covered in health by a thin layer of lung, it is not usually the early seat of friction sound; but as the lung, when once displaced from before it, does not readily replace itself, the rubbing sound is often heard in this position up to a late period in the history of the case. The friction sound is notably double or to-and-fro over the conus arteriosus, and this may be accounted for by the ready completeness with which the right ventricle spontaneously fills itself during the ventricular diastole.

The Apex and Outlying Portion of the Left Ventricle.—The apex and outlying portion of the left ventricle are in health covered by the lung. The extent to which the lung thus affords a protection for the apex depends upon the vigor of the individual, the size of the chest, and the amplitude of the lungs. The portion of left lung immediately covering the apex is a thin tongue, the lowermost protruding angle of its upper lobe, which laps round the apex of the organ, and interposes itself between that part and the ribs. During the diastole, when the ventricle is inactive, the covering of lung is complete; but when the ventricle contracts, owing to the combined muscular rigidity of the organ, and the outward pressure of the blood that is compressed by the contracting cavity, it pushes aside the tongue of lung in front of it, so that the apex sweeps against the ribs and their interspaces. It is thus in young persons and those who are not robust; but in strong adults, inured to exercise, the average size of the lung is increased, and the apex is so embedded in the lung, that its proper beat cannot be felt, except perhaps at the end of a forcible expiration, or when they lie on the left side. In one instance, and in one only, an obscure friction sound was heard over and limited to the apex before it was audible elsewhere. This was on the day of admission, but on the following day it had left the apex, and transferred itself to the whole right ventricle and right auricle. I can offer no explanation of this exceptional sign.

As a rule, the friction sound was, as I have said, limited at first to the right ventricle; but as the disease advanced, the increased fluid in the pericardium displaced the left lung and laid bare the apex, so that the friction sound spread itself from the right ventricle to the left.

When the effusion was at its height the heart was raised, and the apex-beat was felt in the fourth, or even the third space, at or just above and beyond the nipple. Friction sound was probably audible over the apex during the acme in thirty-four of the forty-four cases now under notice; it was absent from that point in nine; and its presence there was doubtful in one case.

The movement of the apex is, in its nature, the reverse of that of the right ventricle at its junction with the right auricle; for while, during the systole, that part moves from right to left, the apex moves from left to right, and from below

upwards. As the active sweep of the apex takes place during the contraction of the ventricles, it is natural to expect that the friction sound at the apex should be mainly systolic, and the examination of my cases shows that this is so. Of the thirty-four cases in which a friction sound was audible over the apex, in six it was heard during the systole only; in ten the *frottement* was double, but was more intense and prolonged during the systole than the diastole; and in none was it stated that the two sounds were of equal intensity during the two periods. In six cases there was a creaking friction sound, usually systolic, at the apex.

When the lower lobe of the left lung shrinks under the double effect of pulmonary apoplexy within the lung, and pleurisy on its exterior, on the one hand; and of compression of that portion of the lung and of the left bronchus, by the great distension of the pericardium, on the other, the apex becomes completely exposed, and extends far to the left. In one such case, a youth, aged 17, a systolic creaking sound was audible over and beyond the apex, and the friction sound extended far to the left, ceasing suddenly in the axilla.

Right Auricle.—The right auricle is in health completely screened from the anterior wall of the chest by the middle lobe of the right lung, which separates it from the middle of the sternum and the costal cartilages to the right of the lower half of that bone. Friction sound is therefore never audible over the right auricle until the portion of lung that is interposed between it and the right cartilages is pushed aside by the advancing tide of effusion, so as to lay bare the auricle. When the effusion into the pericardium was at its height, a friction sound was audible over the right auricle in three-fifths of the cases (28 or 29 in 44).

The expansion of the right auricle is quite passive, and its contraction is made with so little exercise of force, that its movement to the right during its period of filling, and its movement to the left during its period of emptying, are made so quietly, that it exerts no pressure on the cartilages during its to-and-fro movements. It is natural to expect that the to-and-fro *frottement*, the *frou-frou* produced by the passive double friction of the right auricle, should be made up of two equal sounds, and as a rule those two sounds were equal over that cavity.

In twelve of the twenty-nine cases in which friction sound was audible over the right auricle, the systolic and diastolic sounds were equal; in eleven the *frottement* was double, but the relative intensity of the two sounds was not described; and in one a systolic sound, almost creaking in character, was audible over the right auricle. In this last exceptional case a similar almost creaking noise was heard over the base of the right ventricle at the lower portion of the sternum, and that was evidently the source of the rubbing sound over the auricle.

The two sounds made respectively over the right auricle during the two alternate movements of its dilatation, with contraction of the ventricle, and its contraction with dilatation of the ventricle, are not only equal in character, intensity, and continuousness; but they are also more soft and smooth in tone than they are over the ventricle; this contrast being most remarkable in some of those cases that present a thrill and a creaking sound over the right ventricle, and the diffusion of a harsh double friction sound over the whole front of the chest extending downwards even to the eighth or ninth right and left cartilages.

The question here arises whether under these circumstances the soft double friction sound audible over the cartilages to the right of the lower sternum is due to the immediate friction of the subjacent right auricle, or to that of the right ventricle, transmitted through the fluid and softened in its transmission? I think that we must infer that the latter is the usual source of this sound, when we consider that the yielding right auricle is compressed by the fluid in the pericardium at the time of the acme. Why under these circumstances, the two sounds are usually equal, I cannot say.

The Ascending Aorta and Pulmonary Artery.—In health the two great arteries lie behind the upper half of the sternum and the spaces to the left of it, above the level of the third cartilages. They not only have the bony protection thus afforded them, but they are additionally sheltered by a thin covering of lung that is interposed between them and the bony shield in front, and is made up of the inner adjoining margins of both lungs. The aorta is guarded by the strongest portion of the sternum, and the pulmonary artery lies behind the second space and cartilage, and the adjoining margin of the sternum. In the early stages, therefore, of pericarditis, friction sound is never audible over the great vessels. When the fluid increases, the distended pericardium and the elevated heart and great vessels push the double curtain of lungs to each side, so as to bring the great arteries into contact with the sternum and the first and second spaces and cartilages. The heart and great vessels then, as I have already said, occupy the narrower space in the upper portion of the cone of the chest, and there is now no longer room both for them and for the portion of lung superficial to them in health, which is therefore displaced.

In considering the character of the friction sound over the two great arteries, we must distinguish the aorta from the pulmonary artery. The roots of those arteries, including under that term their apertures, valves, and sinuses, descend and ascend fully half an inch during the successive periods of the systole and diastole of the ventricles; the movement of the systole being more active than that of the diastole.

The root of the pulmonary artery is situated at the front of the heart, and when the lung is displaced from before it, the artery lies immediately behind the second, and sometimes also the first, left intercostal space, the second costal cartilage, and the adjoining border of the sternum. The movement of the pulmonary artery, like that of the conus arteriosus from which it springs, is downwards and from left to right during the systole, and the reverse during the diastole. The friction sound over the pulmonary artery, is not, therefore, so far as I know to be distinguished from that over the conus arteriosus. The two-and-fro sound caused by those two adjoining and connected parts must resemble and blend with each other; but while that of the pulmonary artery is situated over and above the second space and the adjoining border of the sternum, that of the conus arteriosus extends downwards from that point to the fourth cartilage, but widening to the right, so as to occupy the whole breadth of the centre of the sternum.

A peculiar systolic scratching noise, that somewhat resembles a friction sound, is sometimes audible over the pulmonary artery during the course of acute rheumatism, and is generally associated with endocarditis. This sound is evidently caused by the vibration of the blood advancing during the systole along the artery when not in a state of tension; and is to be distinguished from a friction sound by its limited area, the sound being confined to the second space, and not accompanied by friction sound elsewhere over the heart; its restriction to the period of the systole and its consequent total want of a two-and-fro character; its freedom from change when pressure is made over it; its unaltering character on successive days; and the absence of pain over the heart or other symptoms or signs of pericarditis.

The root of the aorta instead of being exposed in front, like that of the pulmonary artery, is buried deeply in the centre of the heart, being covered by that artery, the conus arteriosus, and the left border of the right auricle. The root of the aorta cannot therefore cause a friction sound. The ascending aorta, where it comes into view above the right auricle and behind the lower half of the manubrium, is in health deep in situation, being covered by the adjoining margins of the opposite lungs. When, however, the heart and great vessels are lifted upwards by the advancing invasion of the fluid in the pericardium, the lungs are displaced from before the ascending aorta, which may possibly be pressed against the back of the manubrium. Even then, however, it can only excite a partial friction sound, for its movements, which are downwards and upwards, are very slight.

Friction sound was audible at the manubrium over the ascending aorta and the adjoining portion of the pulmonary artery at the time of the acme of the effusion into the pericardium, and especially during expiration, in twenty-six or perhaps twenty-seven of the forty-four cases under review; but this friction sound was evidently not generated by the double movement of those vessels, but was conducted upwards by the sternum, acting as a sounding-board, from the harsh double friction sound over the right ventricle. This was shown by that sound reaching with full intensity to the top of the sternum, which is a little above the transverse aorta, in twenty-six or perhaps twenty-seven of the forty-four cases; and by the close correspondence between the character of the double *frottement* over and above the great vessels at the upper half of the sternum, and that over the right ventricles, at the lower half of the sternum.

At the time of the acme of the effusion into the pericardium the whole heart is raised, and the lungs are separated from each other in front, so that the pulmonary artery, the conus arteriosus and the rest of the right ventricle, the apex and outlying portion of the left ventricle, and the right auricle are uncovered, and brought into immediate contact with the walls of the chest in front of them.

The whole front of the right ventricle bears upon the sternum and left cartilages with varying force. Sometimes it produces a thrill during its contraction, which may extend over its surface from the third to the sixth cartilages, and is often accompanied by a systolic creaking sound. At other times, sometimes with, but generally without a thrill, a double grating sound or a harsh friction sound of various tones, the systolic sound being usually louder than the diastolic, springs up over the whole right ventricle. In rare instances the two sounds are equal. More rarely a soft friction sound, rendered harsh by pressure, or a to-and-fro sound, excited by pressure but absent without it, is present over the right ventricle.

A friction sound is heard over the apex during the acme in about three-fourths of the cases. The apex may, like the right

ventricle, present a thrill and a creaking sound during the systole; or a loud, prolonged systolic friction sound, and a short, feeble diastolic one. In no instance are the systolic and diastolic friction sounds equal over the apex.

During the acme the right auricle in two-thirds of the cases presents over its surface, to the right of the lower half of the sternum, a double, smooth, to-and-fro murmur or friction sound, equally loud during its dilatation and contraction. This double smooth *frottement* over the right auricle is probably transmitted, softened in its transit, through the fluid, from the noisy and active right ventricle.

The friction sound, if any, that may be made during the acme by the ascending aorta and the adjoining portion of the pulmonary artery behind the manubrium, is almost always masked by the friction sound of the right ventricle, which is conducted by the sternum acting as a sounding-board, the sound being thus conducted in more than half of the cases to the upper end of the bone.

The double *frottement* proper to the pulmonary artery when covered with lymph is undoubtedly audible during the acme over the second space, where it must resemble and blend with the double *frottement* proper to the conus arteriosus.

In every instance pressure intensifies the two friction sounds; and it sometimes transforms an ordinary *frottement* into a creaking or grating sound; or a soft friction sound into a harsh rubbing noise; or it excites a friction sound when one was previously absent.

Second Acme. — *Renewed Increase of Effusion into the Pericardium owing to Relapse.*—In eleven cases the effusion into the pericardium, after it had reached its height and commenced to decline, again increased in quantity, and attained to a second acme. Another case that had a relapse and a second acme, that was admitted during the period of the first acme, has not been included in the inquiry that is about to follow. In five of those eleven patients under consideration the fluid, after declining for a second time, again increased so as to present a third acme of pericardial effusion, and in one of the five there was a fourth wave of increase.

I shall examine in these cases with relapse and renewed acme, the comparative height of the pericardial effusion; the extent of the heart's impulse; the area and intensity of the friction sound; the severity of the general illness; and the intensity of the accompanying endocarditis, and its permanent effect on the functions of the valves of the heart during the period of the later acme.

Extent of Effusion into the Pericardium.—In five of the cases the effusion into the pericardium was equal in extent during the first and the second acme; while in five it was greater, and in one it was less, during the first than the second acme. In six of the cases, from two to five days, and in five of them from six to eight days, elapsed between the end of the first period and the beginning of the second period of the height of the effusion.

Position of the Impulse.—In six of the cases, and probably in a seventh, the impulse at its inferior boundary occupied a lower position by from one to two intercostal spaces during the second acme of the effusion than the first (compare Figs. 94, p. 576; and 88, p. 553, respectively with Figs. 91, p. 559; and 86, p. 551; in two cases the impulse occupied the same position during the two periods; in one instance it was imperceptible throughout; and in one it was very feeble.

We thus find that in the great majority of the cases the impulse of the heart was lower during the second acme of effusion, or the period of relapse, than during the first acme. The reason is, I think, evident. When the fluid in the pericardium begins to increase during the early period of pericarditis, the heart, which is then yielding in structure and usually of the natural size, is steadily floated upwards by the increasing tide of effusion into the pericardium, which may indeed compress the auricle, and lessen the size of the heart. The heart, under the double influence of the inflammation on its exterior, and the resulting thick coating of lymph, on the one hand; and the inflammation on its interior, and the resulting crippling of valves, enlargement of cavities, and thickening of walls, on the other, becomes increased in size. The whole organ is, in fact, enlarged, and it is often unyielding in its position owing to its tough new covering, and perhaps to partial adhesions that may have already connected the double surfaces of the thickened pericardium and the heart, especially along and near the septum; and although the renewed increase of fluid elevates the heart to a certain extent, this second elevation of the impulse is not usually so great as the first elevation.

Thrill.—A thrill was felt over the heart for the first time during the second acme in three of the cases. In two of them the thrill was present over the apex, and this was the natural effect of the lowered position, greater prominence, and increased force of that portion of the heart during the second acme than the first. In the other case the thrill was present over the second left space, but in this patient the second acme was the true one, for the effusion was considerably higher during the second than the first acme. A thrill is, in fact, more frequently present over the second space during the first acme

than the second, and over the apex during the second acme than the first, for the reasons that I have just stated.

Area and Intensity of the Friction Sound during the Second Acme of Increased Effusion into the Pericardium.—During the second Acme of the pericardial effusion a creaking friction sound was audible over the heart in four cases, and a grating noise in one; a to-and-fro sound in five patients, and a double friction murmur increased by pressure in one.

In five of the cases the area of the friction sound was greater, and in four it was less during the second than the first acme of the effusion into the pericardium, and in two it was of equal extent during both periods. In five of them the friction sound was audible over a lower position during the second acme than the first, and in none of them was the friction sound lower during the first acme than the second.

In like manner, the friction sound was more intense in six cases, and less intense in four, during the second acme than the first; and in one it was of equal intensity during both periods. In four of the patients both the area and the intensity of the *frottement* were greater, and in three they were both less, during the second than the first acme.

The following agencies, on the one hand, tend to increase the area and intensity of the friction sound during the second acme as compared with the first: The greater size of the heart; the increased thickness and force of its walls; the lowered position of the organ and its impulse; and the greater roughness and toughness of the lymph covering the heart and lining the pericardium.

The following, on the other hand, tend to lessen the area and intensity of the friction sound during the second acme as compared with the first: The greater extent to which the lungs sometimes cover the heart; the restraint placed on the movements of the heart, and especially of the right auricle, by the thickness and toughness of its envelope of lymph; and the adhesions that have often already taken place between the pericardium and the heart; and especially along the septum, between the ventricles, and at the apex. Vital influences blend with and counteract these physical influences in producing the result.

My analysis of the cases does not enable me to assign to each of those causes its proper share in the production of these effects.

In the one fatal case the heart was universally adherent, and in that patient the friction sound was less in extent and intensity during the second acme than the first, owing, I consider, to adhesions that had already begun to form between the heart and the pericardium.

The friction sound, as we have seen, was lower in extent during the second acme than the first in one-half of the cases (5 in 11), owing to the lower position of the heart and its impulse during that period.

In five of the cases the friction sound maintained the same character during the second acme as during the first, but in six others it was altered. Thus, one that had a friction sound on pressure, one that had a smooth friction sound harsher on pressure, and one that had a harsh friction sound creaking on pressure during the first acme, presented, all of them, a creaking friction sound during the second acme; while one with a to-and-fro sound during the first, gave a grating noise during the second acme.

From what I have just said, it is evident that the influences tending to increase the area and intensity of the friction sound during the second acme were in greater force than the influences tending to lessen them; and that the friction sound was usually more intense and more extensive, especially downwards, during the second acme than the first.

Comparative Area and Intensity of the Friction Sound just before, during, and soon after the Second Acme of Effusion into the Pericardium.—The friction sound is, as a rule, louder and more extensive during the second acme of pericardial effusion than either just before or soon after that period. At this stage, therefore, the *frottement* increases with the advance, and decreases with the decline of the fluid.

Degree of the General Illness during the Second Period of Increased Pericardial Effusion.—In five of the cases the illness was extreme, in three it was severe, and in three it was slight or probably so during the second acme.

Of the five cases in which the illness was extreme, the face was anxious in four; there were choreal movements and rigidity, chiefly of the left arm, in one; breathing was laborious in one and quick in four, the respirations ranging from 32 to 48; pain was present over the heart in one, and in another pain was felt, apparently in the side, on a deep breath; swallowing was difficult in two; one had diphtheria; and one raised phlegm tinted with blood.

The patients who were thus affected with relapse of the inflammation of the pericardium suffered more frequently with symptoms of great severity during the first than the second period of the increase of the effusion into the sac. Thus during the first acme in seven patients the illness was extreme, including the five in which it was so during the second acme, and in three it was severe. In one case the symptoms were not described.

Of the seven cases in which the illness

was extreme during the first acme, perspiration was very profuse in three; the face was anxious, pallid, livid, or of a leaden hue, in four; there were slight choreal movements in one; breathing was quick in five, the respirations ranging from 40 to 48; pain was present over the heart in four of those seven patients and in two others in whom the symptoms were less severe; and swallowing was difficult or painful in three.

We thus see that pain attacked the region of the heart in six cases during the first acme, and in only one case during the second acme. The breathing also was more urgently affected during the first acme, when they numbered from 40 to 48 in the minute, than during the second acme, when they ranged from 32 to 48.

On the other hand, depression and anxiety were more marked during the second acme than the first.

The general illness was much more often extremely severe during the first acme in those cases that suffered from a relapse, than during the single acme in those that had no relapse. Thus of those patients in whom there was a renewal of the acme, the illness was extreme during the first acme of the effusion in two-thirds (7 in 10 or 11), and severe in one-third (3 in 10 or 11); while of those who had no renewal of attack, the illness was extreme in only one-third of the cases (10 in 30 or 32), severe in one-half (14 in 30 or 32), and not severe in one-fifth (6 in 30 or 32). In one case of the series with a relapse, and in two cases of the series without a relapse, the symptoms were not recorded.

Intensity of the Endocarditis accompanying the Pericarditis during the Second Acme of the Effusion; and Permanent Effect of the Endocarditis on the Valves. — All the cases that had a relapse of pericarditis were affected with endocarditis in an intense degree. One of the patients had old-standing disease of the mitral and aortic valves; and in seven of them valvular disease was established when they left the hospital, the mitral valve being affected in all of them, and the aortic valve also in three. In three cases the mitral valve, which was incompetent during the attack, owing to inflammation of the valve, completely regained its function.

The proportion of cases in which the valves were permanently crippled among those who were affected with relapse of the pericarditis was much greater than among those who were not so affected. Thus the valvular incompetence became permanent in two-thirds of the patients with relapse of the affection (7 in 10), three of them being affected with both aortic and mitral disease; and in only about one-fourth or one-third of those who had no relapse (11 in 52 and 7 others who left with lessening murmur).

These clinical facts tend to make it probable that when there is a relapse of the inflammation of the exterior of the heart, there is a relapse also of the inflammation of the interior of the heart and its valves; and that the inflammation when thus prolonged tends to cripple the valves for life.

Second Relapse of Pericarditis with a Third Acme *of Pericardial Effusion.*—In five of the eleven cases with relapse and a renewed increase of effusion into the pericardium, after the fluid began to decline, there was a second relapse, and the fluid increased in quantity for a third time. In one of those cases there was indeed a third relapse followed by a fourth acme of pericardial effusion.

In one of the cases the effusion into the pericardium was equal in amount during the first acme, the second, and the third, the wave of increase rising on each occasion to the same height. In two of them the fluid was equal in quantity during the first acme and the third, but was less during the intermediate period of renewed increase; and in the two remaining cases the wave of increased effusion lessened on each repetition, the effusion being less during the third acme than the second, and less during the second acme than the first.

In those five cases from three to five days elapsed between the second acme and the third.

The impulse, at its inferior boundary, was lower during the third acme than the first in three of the cases; and it was lower in one case and higher in another during the third acme than the second; while its position was unchanged during those two periods in a third. In one of the cases the impulse was imperceptible throughout, and in another it became so at the period of the third acme.

The presence of a thrill was not observed in any of the cases during the third acme.

The friction sound is in a declining state during the third acme. The *frottement* was of a definite rubbing to-and-fro character, intensified by pressure, in only one of the four cases during the final acme. In one of those patients the friction sound was double and smooth in character; in another it was single and systolic; in a third it was almost like a bellows murmur; and in the remaining case it was absent with light pressure, but firm pressure brought a to-and-fro sound into existence.

Four of the five patients belonging to this group were affected with great general illness during the final acme; their breathing was distressed and rapid, numbering respectively from 36 to 60 in the minute; while two of them had pain in the chest,

and the remaining two pain in the region of the heart.

The Area and Intensity of the Friction Sound during the Decline *of the Pericardial Effusion.*—In forty-three cases the comparative area and intensity of the friction sound were observed both when the effusion into the pericardium was at its height, and during the period of its decline.

(1) The friction sound spread downwards to a greater extent during the decline than the acme of the effusion into the pericardium, in three-fifths of the cases (26 in 43). (2) In less than one-fourth of the cases (10 in 43) the reverse took place, the friction sound being more extensive, and especially downwards, during the acme of the effusion than when the fluid diminished. (3) The area of the friction sound extended downwards to an equal extent during the acme and the decline of the effusion in a still smaller proportion of the cases (7 in 43).

(1) *Cases in which the Friction Sound spread downwards to a greater extent during the Decline than the Height of the Effusion into the Pericardium.*—I shall consider (1) the time of occurrence; and (2) the duration of the downward extension of the friction sound in these cases; (3) the area; and (4) the character of the sound; and the position of the heart and its impulse and thrill; and (5) the degree of the general illness during the period in question.

1. *Time of the Occurrence of the Downward Extension of the Friction Sound.*—The friction sound spread rapidly downwards soon after the fluid in the pericardium began to decline in all but a very small proportion of these cases. Thus the rubbing sound had already extended downwards to its lowest position in four-fifths of the patients (21 in 26) during the first three days after the acme. During the three following days the descent of the rubbing sound appeared in four more of the cases; but in the last of these this condition came into play quite suddenly on the twelfth, and still more on the fourteenth day after the fluid began to lessen.

2. *Duration of the Extreme Downward Extension of the Friction Sound.*—The downward extension of the friction sound lasted in these cases for a very short period. In two-thirds of them (17 in 26) it was observed during only one day, and in but two cases, or rather one, did it last over three days. This extension downwards of the friction sound during the decline of the fluid was therefore short and transitory.

3. *Area of the Downward Extension of the Friction Sound.*—When the fluid in the pericardium, after having reached its height, lessens in quantity, the heart descends, its impulse is lowered by from one to two intercostal spaces, and the friction sound extends downwards. The area of the rubbing noise is, as a rule, by no means limited to the area occupied by the heart itself; but spreads downwards to an extent varying from one to four inches below the lower boundary of the heart. The friction sound does not, in these cases, diffuse itself downwards over the whole breadth of the region below the heart; for it is usually silent over the front of the abdomen in the epigastric space; while it is present along the right and left seventh and even eighth costal cartilages that bound that space to the right and left; and over the ensiform cartilage that dips downwards from the lower end of the sternum at the centre of that space.

The rubbing noise is usually heard with equal intensity over the right and the left seventh and eighth cartilages. Sometimes, indeed, the sound was louder and more extensive over the right seventh and eighth cartilages than the left; and it appeared as if in those cases the cartilages that rested on the liver conducted the sound better than the cartilages that covered the stomach. The contrast between the harsh rubbing noise heard in some cases over those cartilages, and the complete silence present over the intervening epigastric space was very remarkable.

The friction sound, besides travelling downwards, extended also upwards in one-half of these cases (14 in 26) when the fluid in the pericardium lessened, and the heart and its impulse descended. In one-third of the patients (8 in 26) the area of the friction sound was equally high over the front of the chest during the period of the acme of the effusion into the pericardium, and that of its decline.

In four instances the whole area of the friction sound shifted bodily downwards when the pericardial effusion lessened, and the heart and its impulse descended; so that the upper and lower borders of that area were then simultaneously lowered. In these four cases while the lower boundary of the region of friction sound descended from the sixth to the seventh cartilages, its upper boundary descended in two of them from the second left cartilage to the third, and in the other two from the third cartilage to the fifth.

In two-thirds of those cases (16 in 26) in which the friction sound extended much downwards after the acme, it was also audible up to the top of the sternum. In three of those cases the friction sound so covered the front of the chest as to be audible up to the clavicles, while in one of them it reached the first cartilage. In the whole of these cases the friction sound extended from two to nearly four inches above the actual seat of friction. The

Fig. 92

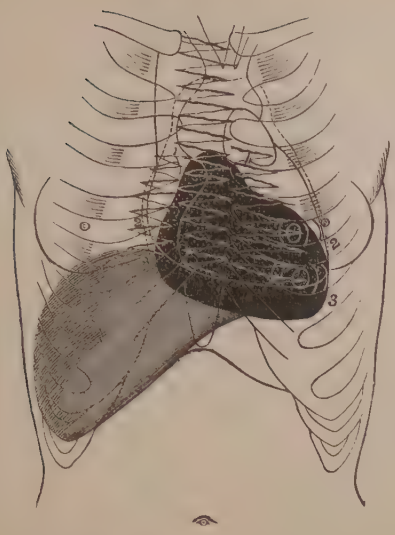
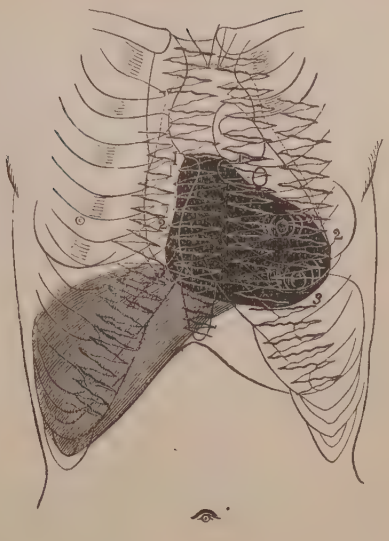


Fig. 93.



For previous views of this case, see Figs. 90, 91, page 559.

Fig. 92, from a housemaid aged 20, affected with rheumatic pericarditis.

Period of the decline of the pericardial effusion after the first acme.

Fifth day after the acme of pericardial effusion, seventh day after the first observation of the friction sound and increased pericardial dulness, and ninth day after admission.

The pericardial effusion has lessened much since the period of the acme, its upper boundary (1, 1) having descended from the middle of the manubrium to the middle of the sternum; and its lower boundary having probably ascended from a little above the end to about the middle of the ensiform cartilage. The heart (2, 2) is lower, and the amount of fluid between the under surface of the heart and the floor of the pericardium (3, 3), though still considerable, has evidently lessened by at least one half. The right ventricle, the inner or left half of the right auricle, and the apex and front of the left ventricle are exposed; but the great arteries are covered with lung.

The region of pericardial dulness (see the black space) probably extends from the middle of the sternum between the third cartilages, and from the fourth left cartilage, down to the middle of the ensiform cartilage, and the lower third of the sixth cartilage; and from a little to the right of the lower half of the sternum to the nipple line. The lower boundary of the heart is behind the upper edge of the sixth rib, and the top of the ensiform cartilage.

The impulse has descended from the third space during the acme, to the fourth and fifth spaces. (See the circular and curved lines in those spaces.)

A double friction sound (see the zigzag lines—systolic lines thick, diastolic thin), which is more harsh on making pressure, is heard over the whole length of the sternum; which is most intense at the middle of the bone, and is louder at its lower end during inspiration, and over the manubrium during expiration; a creaking sound is audible during systole over the third, fourth, and fifth left spaces; and a friction sound is heard to the right of the lower portion of the sternum. (The nipple is too far to the left in this figure.)

Fig. 93, from the same patient as Figs. 90, 91 (page 559), 92, and 94.

Period of the decline of the pericardial effusion; second view, taken the day after a slight and transient second acme, when the fluid was again declining.

Remarkable extension of the friction sound over the greater part of the front of the chest, and especially downwards.

Tenth day after the first acme of pericardial effusion; twelfth day after the first observation of the friction sound and of pericardial dulness; fourteenth day after admission; and four days before the occurrence of a second acme.

The pericardial effusion has diminished. There is therefore less fluid between the under surface of the heart and the floor of the pericardium (3, 3); the roughened front of the heart is more dry, and is closer to the corresponding roughened surface of the pericardial sac; the heart (2, 2), which is somewhat enlarged, is lower in position; and the upper boundary of pericardial effusion (1, 1) is lower, and its inferior boundary is somewhat higher than when Fig. 92 was taken five days previously. The whole right ventricle, the left border of the right auricle, and the apex and front of the left ventricle are exposed; while the great arteries and part of the conus arteriosus are covered with lung.

The region of pericardial dulness (see the black space), which is bounded above by the

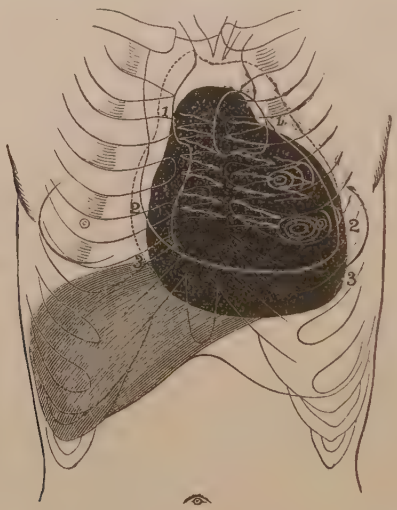
fourth cartilage, and below by the sixth cartilage, is probably rather less extensive above, below, and to the right, than in Fig. 92, taken on the ninth day after admission.

The impulse is lower, stronger, and more extensive than in Fig. 92, and is present from the third to the fifth spaces, and up to or beyond the mammary line (see the circular and curved line in those spaces); and gives therefore direct evidence that the heart is lower in position, and that the effusion has lessened.

The friction sound (see the zigzag lines—systolic lines thick, diastolic thin) has gained a very great extension, being audible over a great part of the front of the chest, from the first costal cartilage to the seventh left and the eighth right cartilages; and from the top of the sternum to the bottom of the ensiform cartilage. This extension of the friction sound is especially marked downwards, where it extends for about four inches below the heart, and is lower on the right than on the left side, reaching over the right eighth cartilage in front of the liver, and the seventh left cartilage in front of the sternum. This is the reverse above, when the rubbing sound extends four inches to the left, and only about two inches to the right of the sternum.

The friction sound is harsher than it was yesterday; over the midsternum it is louder during the systole than the diastole; and it is intensified by pressure; over the manubrium, the two-and-fro sounds are equal; over the ensiform cartilage, friction sound is present during inspiration; it is creaking during systole over the second and first spaces; and it becomes louder below the mamma during inspiration.

Fig. 94.



For previous views of this case, see Figs. 90, 91, page 559, and 92, 93 on the preceding page.

Fig. 94, from a housemaid affected with rheumatic pericarditis.

Third acme of pericardial effusion (the second acme was very slight and transitory).

Thirteenth day after the first acme; eighteenth day after admission.

The pericardial effusion is greatly increased, but its extent and limits are not definitely described. If we compare this third acme with the first acme (Fig. 92, page 575), we find that the distended pericardium, though it contains less fluid, is wider in relation to its length; that the heart is larger; and that the lower boundary of the heart is lower, in this the later and renewed, than in that the earlier and original acme. In the first acme the heart was not yet enlarged, or, being compressed by the fluid in the distended sac, was possibly lessened in size; and the walls of the pericardium were still unyielding, so that the swollen sac took the form that it would naturally take if artificially distended with fluid (see Figs. 79, 80, page 540). In this, the third acme, the heart has become enlarged both by pericarditis and by mitral endocarditis; the lower boundary of the heart, although elevated by the accumulated fluid, is lower than in the first acme; and the walls of the pericardium have become thicker, softer, and more yielding than in health, so that the distended sac yields to the right and left, where it meets with no resistance, to a greater extent than it does upwards and downwards, where it meets with much resistance; and is therefore wider in relation to its length than it was during the first acme, when its form was more purely pear-shaped. The whole front of the heart and great vessels is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the pulmonary artery, and the ascending aorta within the pericardium. The fluid has evidently interposed

itself to a greater extent between the surface of the lower portion of the front of the heart and the walls of the chest during this, the third acme, than during the first acme.

The region of pericardial dulness (see the black space), the limits of which are not described, corresponds in general form and outline to the pericardial effusion, and evidently extends more to the right and left, and less upwards and downwards than it did during the first acme.

The impulse has again been elevated at its lower boundary, and this time from the fifth space, as in Fig. 93, page 575, to the fourth space, where it is feeble; and it is felt over the third space during expiration. (See the concentric circles in those spaces.) The lower boundary of the impulse is therefore lower by one space than it was during the first acme, when it occupied the third space (see Fig. 91, page 559).

The friction sound (see the zigzag lines—systolic lines thick, diastolic thin) is softened, and is limited in area, being heard over the middle region of the sternum, where it is double, and although frictional in character is almost like a bellows murmur; and is audible over the second and third spaces during the systole.

Later progress of this case.—On the following day, the nineteenth after admission, the friction sound was almost creaking, or like the sound made by rubbing with sand-paper, over the second and third left spaces. On the twenty-first day, or the fourth after the third acme, the extent of dulness over the pericardial region had lessened; and a double friction murmur, which was not rhythmical with the sounds of the heart, was audible over the base of the right ventricle, and became harsh on pressure. The friction murmur was still heard on the following day, but after this it was scarcely audible.

region of pericardial dulness was limited above in all but three of the patients by the third intercostal space or the fourth cartilage; and the space between this limit and the top of the sternum nearly measures the extent to which the *frottement* extended above the seat of the friction.

When the fluid in the sac declines, the roughened heart rubs against the roughened pericardium, and in doing so bears directly upon the lower half of the sternum with which it is almost in contact; owing to the removal of the anterior layer of the fluid, and the descent of the heart and its impulse. The sonorous vibrations excited by the movements of the heart are directly conveyed to the sternum, and that bone and the costal cartilages attached to it act as a sounding-board and transmit the rubbing noise in all directions.

In three of the cases the sound was audible over the whole front of the chest. Usually, however, it extended only slightly to the right, and over a greater extent to the left of the lower half of that bone. As a rule, therefore, the rubbing noise extended in a straight line from the top to the bottom of the sternum, and there it divided into two diverging lines, one along the right, the other along the left seventh cartilage, where they form the boundaries of the intervening epigastric space. The area of friction sound thus extending along the sternum and the right and left seventh cartilages closely resembles in shape the inverted letter Λ. Since however the friction sound also extends downwards over the ensiform cartilage, its area is somewhat like a trident with a short central prong.

In one-fifth of the cases (5 in 26) the area of the friction sound dwindled during a short period after the time of the acme, and then suddenly expanded, and especially downwards, at a later date during the decline of the effusion.

In one case the friction sound alternately lessened and increased in area and intensity during the ten days that intervened between the termination of the acme, and the time at which the frottement had a remarkable downward development.

4. *Character and Intensity of the Friction Sound; and Position of the Heart and of its Impulse and Thrill.*—At the time that the friction sound spread downwards when the effusion lessened, the sound was intense, loud, and of a marked character in all but three or four of the twenty-six cases that belong to the group under consideration.

In nine of those cases the friction sound was creaking (6), or grating (3); in thirteen it was harsh and loud; and in four its intensity was not specified.

The friction sound in the twenty-six cases under review, as a rule, gained in intensity as it gained in area; and lost in intensity as it lost in area. Thus in all but six of the cases, the rubbing noise became more harsh when it increased in extent; and in all but two of them it became softer when it lessened in extent.

When the effusion lessened, the impulse, while it descended at its lower boundary, was still felt beating in the higher spaces into which it had been forced during the acme in fully one-fourth of the cases (5 in 19); while, curiously, the impulse ascended to a higher space than it had occupied during the acme in six other patients.

A thrill was felt over the heart in five of these twenty-six cases during the acme of the effusion. In four of these the thrill disappeared when the effusion lessened, and in one it remained, though with lessened intensity. In three other patients a fresh thrill came into play during

the decline of the fluid; in two of them over the apex, and in the other case at the second space.

In these twenty-six cases, when the effusion into the pericardium lessened, the heart, relieved from the pressure of the fluid, descended into its natural space, and even below and beyond it. The heart thus relieved, beat with increased force; its right cavities were enlarged, owing to the increased supply of blood from the system, and the continued resistance offered to the flow of blood through the compressed lung and the incompetent mitral valves; and, as the general result, its anterior walls played with greater power and noise upon the sternum and cartilages, and the friction sound was heard over a largely increased area.

5. *Degree of the General Illness.*—At the time that the area of the friction sound was most extensive, especially downwards, when the fluid in the pericardium lessened, twenty of these twenty-six cases were less ill or in better health, three of them were probably better, and three were worse in health than they were during the acme.

In a large proportion of the cases under review, when the fluid in the pericardium lessened, the heart descended and gained freedom of movement and power, and the general health improved; and as a natural result the friction sound increased in extent, and especially downwards. The comparatively dry roughened surface of the heart rasped two and fro upon the roughened surface of the pericardium. These influences combined to cause the increased harshness and extension of the friction sound; which, starting from its focus of greatest intensity over the right ventricle, radiated in all directions over and beyond the region of the heart and the great vessels, outwards to the right and left, upwards to the summit of the sternum, and especially downwards over the ensiform cartilage and the diverging right and left seventh and eighth cartilages.

(2) *Cases in which the Friction Sound was audible Downwards to a greater extent during than after the Acme of the Effusion.*—In ten cases the friction sound was audible to a greater extent downwards when the effusion was at its height than during its decline.

Two series of influences are at work in these cases, acting at different times, to enlarge the area of friction sound during the acme, and to lessen it during the decline of the effusion.

1. When, during the acme, the friction sound is creaking or grating, being sometimes associated with a thrill, over the right ventricle, and when it radiates thence in all directions, softened in character, beyond the region of actual friction, the heart, raised by the increased effusion into the narrower space at the upper part of the cone of the chest, beats with increased force directly against the sternum, the higher cartilages, and their spaces, and so excites an intense and widely diffused friction sound.

When the fluid lessens the heart descends and is again partially covered with lung; and as it beats over a smaller extent, and with less pressure against the sternum and cartilages, the friction sound lessens in intensity and area.

2. When the friction sound is of moderate intensity and extent during the acme, it sometimes lessens during the decline of the effusion. In these cases the impulse at its inferior boundary is not notably lowered, while it disappears from the upper spaces. In some of these cases the action of the heart is throughout feeble; and probably in others of them slight adhesions take place at the apex and septum which restrain and lessen the descent of the heart, the rubbing movements of the right ventricle, and the area and intensity of the friction sound over the higher intercostal spaces.

(3) *Cases in which the Friction Sound extended Downwards to an equal extent during and after the Acme of the Effusion.*—In seven cases the friction sound was of equal extent during the two periods, when the fluid in the pericardium was at its height and was declining.

Character and Intensity of the Friction Sound during the Decline of the Effusion, and the Relation of the Intensity to the Area of the Friction Sound.—I shall examine these conditions during three periods in the order of time of the decline of the effusion, (1) the beginning of the decline of the effusion; (2) the gradual and the interrupted progress of the decline of the effusion; and (3) the final dying away of the friction sound; and (4) shall then inquire into cases in which the ordinary friction sound gave place to a friction murmur towards the end of the attack.

1. *Character and Intensity of the Friction Sound at the Beginning of the Decline of the Effusion.*—When the amount of fluid in the pericardium began to lessen, if the friction sound increased or diminished in intensity, it usually increased or diminished also in area.

As a rule, the friction sound increased in intensity and area in those cases in which the *frottement* extended further downwards after than during the acme; while it lessened in intensity and area in those in which the friction sound spread more downwards during the acme than after it.

When the friction sound spread downwards during the decline of the effusion,

the sound gained in area in nearly every case (25 cases in 26), and in intensity in two-thirds of the cases (18 in 26). We thus see that while an increase in the intensity of the *frottement* almost invariably leads to an extension of its area—for I find only one exception to this rule—and while a diminution of its intensity likewise generally causes a diminution of its area; yet, in certain cases, the friction sound gains in extent, though it lessens (4 cases in 43) or remains unchanged (3 cases in 43) in intensity. This is explained by the lowering of the heart, and the consequent descent of its impulse during the decline of the effusion in all the cases—the surface of the roughened organ being thus brought into more extensive contact with the sternum at its lower half, and with the corresponding costal cartilages: while in the small number of cases in which, although the friction sound gains in area, it is lessened or not increased in intensity, the heart, released from its confinement in the contracted space of the chest above, where it rubbed with force and noise against the sternum and cartilages in front of it, finds itself moving with ease in its proper place in the lower and wider part of the chest, and so presses with less force and less noise than before on the sternum and cartilages in front of it. The causes of the increased intensity as well as area during the decline of the effusion, which, as we have just seen, occur in the great majority of the cases under examination, have been already considered at page 578.

2. *The Gradual and Interrupted Progress of the Decline of the Effusion.*—In thirty-one of the forty-three cases now being examined, the effusion in the pericardium steadily and gradually declined, and, as we have already seen, in twelve of them, owing to relapse, the effusion after beginning to decline, again increased in quantity generally once, sometimes twice, and on one occasion even a third time.

The progress of the friction sound during the decline of the effusion was rarely uniform. It was in several of the cases silenced and suspended for a time (6 in 43); it more frequently, however, when in full play, became feebler during a short period, and then again louder (13 in 43). In a larger number of the cases the *frottement*, after attaining to its greatest intensity, more or less steadily lessened in loudness and extent until it finally disappeared (23 in 43).

In one case the friction sound suddenly and permanently disappeared after an attack of syncope. In this patient, a girl, the friction sound vanished when the action of the heart became enfeebled; and she died in a second attack of syncope a few hours after the first attack.

Cases in which the Friction Sound vanished and reappeared during the Decline of the Effusion.—In six of the forty-three cases under review and in one other patient the friction sound disappeared and reappeared during the decline of the effusion. In five of these cases the *frottement* was absent for from two to three days, and in one of them for about seven days.

In three of the patients the friction sound, as in the case just referred to, vanished for a time after the application of leeches for the relief of pain.

If we view these cases as a whole, and take into the survey the case of the female servant who died from a second attack of syncope, the first attack having permanently quenched a loud and pervading friction sound, we shall, I think, see that when the force of the heart's action and the volume of the blood in circulation are lessened—either by immediate syncope, by loss of blood from leeching, by diarrhœa, sickness, or other exhausting influences, by pain in or over the organ, by extreme distress in breathing, or more often by a combination of several of these lowering agencies—then the rubbing sound, when in full play, may gradually or suddenly vanish, and may suddenly rekindle into full volume after a longer or shorter period of silence.

Cases in which the Friction Sound lessened and then increased in Area and Intensity during the Decline of the Effusion.—In thirteen of the forty-three cases under examination, and in three other cases, the friction sound, when in full play, lessened in extent and intensity, and after a longer or shorter interval again resumed more or less nearly its full sway.

In one of these sixteen cases the diminution of the *frottement* was associated with sudden faintness; in two with loss of blood from leeching; in eight with increase of the general illness—in seven of which as the health improved the friction sound resumed its extent and intensity—in two with an amelioration of the symptoms; in two with irregularity and intermission of the pulse and the action of the heart; and in two the state of the health is not described.

In eight cases the diminution of the friction sound corresponded with an increase of the general illness, which showed itself generally by an anxious expression, accelerated and difficult breathing, and pain over the heart; sometimes with cough and rusty phlegm; and sometimes with abundant perspiration. With the renewed increase of the rubbing sound there was in all these cases; save perhaps one, a marked improvement in the health; manifested usually by a comparatively cheerful expression, more easy respiration, lessening or absent pain over the

heart, and assuaging of cough with diminution of phlegm.

(1) *Duration and* (2) *Progress of the Friction Sound during the Decline of the Effusion.*—(1) *Duration.*—The friction sound lasted for a very variable period during the decline of the disease.

In the group of thirty-one cases that had no relapse and no return of the effusion into the pericardium, the friction sound lasted from three to nineteen days, its average duration being ten days.

In the group of twelve cases that suffered from relapse with return of the effusion into the pericardium, the friction sound lasted from eleven to twenty-two days, its average duration being fifteen days.

(2) *Progress.—Cases in which the Maximum Development of the Friction Sound took place during the Decline of the Effusion.—Period between the Maximum Development and the Cessation of the Friction Sound.*—In thirteen of the nineteen cases under examination the area of the friction sound steadily lessened from the day of its maximum extension to that of its final disappearance. It contracted gradually from right to left and from left to right, from above downwards and from below upwards, towards the centre or focus of actual friction. It thus died away from beyond and over the great vessels, the right auricle, and the apex, and from the region that it had previously occupied below the lower boundary of the heart. Towards and over the region of actual friction it step by step concentrated itself, and after lingering over the right ventricle with softening tones for a shorter or longer period, it quietly died away. In about one-half of the cases (6 in 13) this subdued sound outlived the period of its greatest intensity and extent for from one to two days; in the remainder, for from three to seven days; and in one only did it exist for nine days.

The front of the right ventricle was, as I have just said, the last home of the friction sound, as it had been indeed the seat of its birthplace. As the position of that ventricle varied in different patients accordingly as the heart was larger or smaller in size, higher or lower in situation, the final seat of the softened friction sound varied in different cases, from the left third and fourth cartilages to the fifth or sixth; and from the middle third of the sternum to the ensiform cartilage.

There was a general but by no means invariable correspondence between the area of the friction sound on its last observation, and the position of the impulse.

In only three of the nineteen cases now under review did the impulse occupy the same position when the friction sound was heard for the last time, as when it was most extensive. In four cases it had descended at its lower boundary from the fourth space to the fifth; and in four cases it had disappeared from the upper space at the time of the last observation of the friction sound, when compared with the time at which it was predominant. There was therefore in these patients a tendency for the heart and its impulse to take up a lower position, and to be covered to a greater extent with lung as the friction sound was about to disappear, and the case advanced towards its termination. On the other hand, in two other cases the impulse gained ground above, and appeared in the second space for the first time when the *frottement* was heard for the last time.

The descent of the impulse both above and below when the case advances to recovery and the friction sound is dying out, appears to me to be the natural bias in these cases when the heart is not adherent, and descends into its natural situation; when the right ventricle and pulmonary artery are not greatly enlarged; and when the upper lobe of the left lung expands in front so as to cover the pulmonary artery and the upper portion of the right ventricle.

When, however, the heart becomes more or less adherent; when the pulmonary artery and right ventricle become enlarged owing to mitral regurgitation; when mitral incompetence is combined with adherent pericardium; when the walls of the pericardium are thickened; or when the left lung does not expand in front of the upper border of the heart so as to cover the pulmonary artery and the conus arteriosus; and notably when two or more of these conditions combine their influence, then the impulse tends to remain in or attain to the higher intercostal spaces, and especially the second space.

In one remarkable case belonging to the group of nineteen now under review, the friction sound was lost on the fifth day after the acme, and reappeared on the twelfth day with greater intensity and over a larger area than at any previous time. In three other cases the friction sound, after gradually diminishing in intensity and area, became suddenly reinforced; and in two others a similar diminution and increase of the *frottement* took place but to a comparatively slight degree.

3. *The final dying away of the Friction Sound.*—The friction sound offered greater variety in different cases just before the time of its extinction than at any other period of its existence.

(1) In a very small number of the cases (4 in 43) the friction sound, when in full play, suddenly disappeared; (2) in two-fifths of them (16 in 43) the *frottement*, after being more or less loud up to a certain date, rapidly declined, and vanished

in one or two days; (3) in a fifth of them (8 in 43) the decline of the friction sound was gradual; (4) and in two-fifths of them (16 in 43) the ordinary rubbing sound gave place towards the end of the case to a friction murmur sometimes double, and increased by pressure (8), sometimes double and excited by pressure (5), sometimes single and systolic and intensified by pressure (2), and in one case a single friction murmur was excited by pressure.

4. *Cases in which the ordinary Friction Sound gave place to a Friction Murmur towards the end of the attack.* — In fifteen patients, and possibly in a sixteenth, a friction murmur was audible in lieu of the ordinary friction sound towards the end of the attack of pericarditis.

We have already seen that in a certain number of cases, at the beginning of the attack, the ordinary friction sound was preceded by a friction murmur: and that in one remarkable case a friction murmur prevailed throughout the whole course of the disease to the exclusion of the usual rubbing sound. I would here refer to what has already been said as to the friction murmur as it was observed during the beginning of the attack, at pages 556–557.

In one case a systolic friction murmur audible on making pressure, and in another case a systolic friction murmur increased by pressure, was respectively the final sign of pericarditis.

In six cases a double friction murmur was audible on pressure towards the close of the affection. One of the cases of this group, a servant girl aged twenty, presented on the seventh day, when the effusion was at its height, an extension of the *frottement*, when there was a double grating friction sound. On the eleventh, when the effusion was declining there was a feeble murmur-like friction sound over the right auricle, to the right of the lower sternum; and later in the day the heart sounds were natural over the lower sternum, but pressure brought out a double friction murmur not quite rhythmical with the sounds of the heart. A systolic friction sound was audible over the left fifth cartilage. On the fourteenth day a faint double murmur was still excited by pressure over the lower sternum. This was the last day of undoubted pericardial friction sound, but on the eighteenth day a double grating friction sound burst out on pressure at the end of a deep breath, that was probably pleuritic.

In several of these cases a friction murmur either prevailed over the right ventricle during the early stages, or was limited to certain favorite spots, such as the right auricle, when the friction sound was at its height. Later, the friction murmur gradually again developed itself as the harsher friction sounds became softened, and at length spread itself over the heart. Soon, however, this disappeared as a constant sound, but for one or two final days of the disease it could be again awakened by making pressure over the right ventricle. Several of these cases ended with a double friction murmur that was intensified by pressure.

In addition to these cases in which the friction murmur prevailed exclusively towards the termination of the disease, there were others in which, while the friction sound was harsh, and even creaking or grating over the focus of its greatest intensity, it was yet so toned down towards the lower margins of the area of rubbing sound, especially at and below the ensiform cartilage, that a double friction murmur was audible there, when a loud double grating noise was heard over the right ventricle. In some of the cases also, when a creaking, or grating, or rasping sound prevailed with a thrill over the right ventricle, a double friction murmur was audible over the right auricle. Here the stormy noises prevailed over the forcible ventricles, and the soft murmuring sounds over the passive auricle.

The occurrence of a creaking, grating, or harsh friction sound depends on the force with which the heart contracts and presses against the cartilages and sternum, and on the roughness of the lymph-covered rubbing surfaces; the creaking sound being mainly excited by pressure, the grating noise by the roughness of the two surfaces when the one rubs actively upon the other. The friction murmur, on the other hand, is due to the gentle or restrained movements of the heart, and the comparative smoothness of the rubbing surfaces all over the heart, that occur towards the end of the attack. It may also be present in its softest and most murmur-like tones over the comparatively smooth and feeble right auricle, and below the heart over the epigastrium, when the attack is at its height, and is speaking with the greatest harshness and noise over the more vigorous parts of the organ; and when the harsh friction sound is evidently softened and rendered murmur-like during its transmission through the fluid intervening between the seat of active friction, and the comparatively distant surface of the chest over the right auricle or the epigastrium.

I have already given the distinctions between the friction murmur and the valvular murmur when inquiring into the occurrence of the former during the first blush of the affection. The rules that apply to the distinction of the friction murmur during the early period of the attack apply also to its distinction during the later period. These rules have been already given at pages 556–557, but the following is a *résumé* of the more important

distinctions between the friction murmur and the valvular murmur :—

The friction murmur is not rhythmical with the natural heart-sounds, but the two sounds are heard side by side ; the valve murmur is rhythmical with the natural heart-sounds, and the two sounds are in perfect unison. The friction murmur does not begin with an accent or shock, but is of equal tone throughout ; the valvular murmur begins with an accent or shock, the accent or shock of the corresponding first or second sound which serves as the starting-point for the murmur. The friction murmur is greatly intensified, and is often altered in tone on pressure ; the valvular murmur is brought nearer to the ear by pressure, but is not altered in tone.

There are certain differences between the early and the late friction murmur, although their characters in the main correspond.

In situation the early and late friction murmurs for the most part correspond, being generally seated over the base or body of the right ventricle. The early friction murmur was situated to the left of the sternum in six cases (6 in 8), in four of which it was also heard over the sternum ; and it was present over the sternum alone in two cases (2 in 8). The late friction murmur was audible over the sternum alone in four cases ; over that bone and to the left of it in five ; to the left of the sternum alone in four ; and to the right of the sternum in three cases, including one case in which it was also audible to the left of the sternum. From these figures it would appear that the early friction murmur is always situated over the right ventricle ; but that while the late friction murmur is present over the right ventricle in seven-eighths of the cases, it is audible over the right auricle in one-fifth of the cases.

The late friction murmur is smoother and more equal in tone ; more prolonged ; less rustling and more murmur-like ; more alike in tone and intensity during the systole and the diastole ; varies less from day to day ; and lasts much longer than the early friction murmur. Pressure intensifies both of them and often modifies their tone, but I think that the early friction murmur is more frequently converted by pressure into a true rubbing sound than the late friction murmur.

The complication of a coexisting aortic murmur with the friction murmur is more frequent during the late than the early period of the affection.

The Character and Tests of Pericardial Friction Sound.

I shall, before concluding the subject of pericardial friction sound, briefly consider the characteristic nature and tests of that sound, including its character and rhythm ; its position and extent ; the influence exercised over it by respiration ; its variation from day to day in character, intensity, rhythm, position and extent ; and finally, the effect upon it of external pressure over the region of the pericardium during pericarditis, or the *pressure-test* of friction sound.

Character of the Pericardial Friction Sound.—The friction sound when in full play, and of its usual to-and-fro character, speaks for itself. I have already illustrated, in the preceding pages, the clinical history of the forms and variations, the growth, ripening, and decline of the friction sound. When the friction sound is smooth and soft, almost resembling a murmur, or when a friction murmur is present, the sound no longer declares itself, from its very nature, to be of a rubbing quality, and requires for its distinction that other points shall be considered besides the tone, nature, and to-and-fro quality of the sound. The clinical history and distinguishing characters of the friction murmur during the early advance and the late decline of the attack of pericarditis have been given respectively at pages 556 and 581.

The Rhythm of the Friction Sound.—In a large proportion of my cases it was noticed that when the friction sound was not of its completely developed to-and-fro and rubbing character, that is, during both the advance and the decline of the pericarditis, the healthy sounds of the heart were heard along with the double or single friction sound. The natural sounds of the heart and the friction sounds were never welded or incorporated together, but were each of them heard separately, and, so to speak, side by side. They did not seem to begin or end together ; and although they were both sounding at the same time, they yet appeared to be completely separate and apart. They were not, therefore, rhythmical with each other. That the natural heart sounds are in play within the period of the to-and-fro friction sound is evident, for when that sound becomes sufficiently loud and continuous, whether by the natural advance of the disease, or by pressure made from without, the sounds of the heart are overwhelmed, being masked by the predominant rubbing noises.

When the to-and-fro friction sound is loud, harsh, and in full play, the systolic and diastolic sounds being equal in duration—though rarely in loudness, the systolic sound being the louder—each sound seems almost to fill up its respective space, leaving two very short intervals of silence between the two sounds. These two friction sounds never begin with an accent or shock, but they commence, continue, and

end as a rule with the same tone throughout. In these respects they differ from the natural heart sounds. The first sound always ends in a shock, followed by a short but definite space between itself and the second sound; and the second sound consists in a short shock, followed by a prolonged space between itself and the first sound. The mitral murmur always begins with a shock or accent, the shock of the first sound, and the murmur fills up the space more or less completely between that shock and the second sound. The diastolic aortic murmur also commences with a shock or accent, the shock of the second sound, and it usually fills up the space but not always completely, between that shock and the first sound. The absence of a commencing shock or accent from the friction sound or friction murmur and the presence of a commencing shock or accent with the valve murmurs distinguish those two classes of sounds from each other.

The first contraction of the ventricles precedes by an appreciable period the flow of blood from them into the great arteries; and after that flow has ceased, the exterior of the heart is still in motion. The play of the surface of the heart against that of the pericardium therefore precedes, accompanies, and follows the natural first sound of the heart, and precedes and accompanies the coinciding valvular murmur if present. The closure of the aortic valve precedes the second sound by the tenth of a revolution of the heart's action. The diastolic frottement therefore both precedes and follows the second sound; and accompanies a diastolic murmur, if present, throughout its whole period. The friction sound being made by the moving exterior of the heart, is in relation to the healthy heart sounds and the valvular murmur, which spring from the interior of the heart, as if it were made, so to speak, by an instrument playing outside the room, while they are made as if by an instrument playing inside the room. The friction sound is therefore a surface noise, working apart from, and often over-riding the healthy heart sounds and the valvular murmurs. The healthy heart sounds and the valvular murmurs are, on the other hand, internal noises made simultaneously and by the same parts, and playing together inseparably and in unison.

When listening to the two sounds, the frictional and the natural heart sounds, playing together but not in concert or unison, I have found it very difficult to say whether the systolic friction sound commenced before the first sound of the heart or not. For the reasons just given, however, and that a considerable space of time intervenes between the beginning of the systole and its final shock, amounting to about two-fifths of the healthy revolution of the heart's action, it is evident that the commencement of the systolic friction sound must precede the final shock of the first sound. In one case I heard a short brush at the beginning of the systole, and this no doubt represents the natural beginning of the prolonged systolic friction sound. As a rule the systolic friction sound is of equal tone throughout, whether it is creaking, grating, rubbing, or rustling; but in one instance that sound became suddenly less loud about the middle of its course, and remained so to the end of the systole, the second half of the sound being weaker than its first half.

In one instance a systolic brush, excited by pressure, occupied the latter two-thirds of the systole; in another a systolic whiff, excited by pressure, extended into the diastolic period; and in a third, a double brush was excited by pressure, the systolic being the longer, and each brush occupied a part of the systole and a part of the diastole. I state these signs as I heard them, but cannot account for them.

The diastolic friction sound presents much greater variety in character and rhythm than the systolic friction sound. While the systolic sound is usually continuous through the whole of its proper period, the diastolic friction sound is often of short duration; when it is, I believe, usually present about the beginning of the diastole, and when it accompanies but is separate from the natural second sound; in one instance, however, the natural second sound was followed by a diastolic graze. Sometimes there was a double graze or rub during the diastole; when the entire friction sound resembled the noise made by sharpening a scythe, having one forward or systolic, and two backward or diastolic strokes. When the friction sound was to-and-fro, the second sound appeared generally to be equal in duration, but not in loudness, to the first. When a creaking sound was present it was mostly limited to the systole; this was not so, however, with the grating noise, which was usually a double sound.

The diastolic sound was usually equal in intensity and length to the systolic over the right auricle, both sounds being in all but one instance soft in character. This double soft to-and-fro sound over the right auricle was evidently transmitted, softened during its transit, from the loud speaking right ventricle, through the fluid, to the cartilages in front of the right auricle.

The diastolic friction sound was often absent, and, relatively to the systolic friction sound, was always short and feeble over the apex. In more than one instance, in adults, the diastolic friction sound at the apex appeared to have in it a peculiar twist.

Respiration exercised in many of my cases a definite and speaking influence

upon the area, and in a few of them upon the intensity of the friction sound. The friction sound became more loud or harsh in three cases during expiration, and in four during inspiration; and in one the frottement disappeared at the end of a deep breath.

The area of the friction sound increased below during inspiration in a large number of cases, or thirty-one, while in a much smaller number of instances, or eight, it increased above during expiration.

The Friction Sound varied in character, intensity, rhythm, and position from day to day. The clinical history contained in the previous pages of the friction sounds during pericarditis is pervaded throughout with instances of the great daily variability of the friction sound in all its relations. This changing condition of the friction sound during the successive phases of the disease is one of the important characteristic features of that sound. This feature has been already abundantly illustrated.

Position and Extent of the Pericardial Friction Sound. — Dr. Stokes[1] in 1834 stated that the friction sounds in pericarditis are audible generally only over the region of the heart. I stated independently, in 1843, that I had never heard the friction sounds beyond the region of the heart.[2] We have seen in the previous pages that during the advance of the effusion, and usually during its acme, the friction sound is limited to the region of the heart, but that in certain cases with a thrill, the friction sounds spread during the acme from the seat of the thrill as from a focus, in all directions, over the front of the chest, and especially downwards.

During the period of the decline of the effusion, the friction sound, as we have seen, also often extends beyond the region of the heart, over the front of the chest, and especially downwards to the seventh and eighth, and even the ninth cartilages (see pp. 574, 577). The various changes in the area of the friction sound are given in the previous pages, and to those I refer for the more extended study of this subject.

The position, limitation, and extension of the pericardial friction sound supply characteristic differences between pericardial friction sounds and endocardial murmurs.

The Effect of Pressure with the Stethoscope *over the region of the Pericardium during Pericarditis on the Friction Sound; or the* Pressure Test *of Pericarditis.*—I called attention in 1843, in my paper on the situation of the internal organs,[1] to the effect of pressure made with the stethoscope over the region of the pericardium in rheumatic pericarditis, in intensifying or even bringing into play a pericardial friction sound. Since then Dr. Walshe—who, in the *British and Foreign Medical Review*, very kindly reviewed my paper just referred to, soon after its publication, and Dr. Stokes, independently observed this sign. This effect of pressure is thus spoken of by Friedrich. "Sehr brauchbar is das von Sibson, Walshe, und Stokes, angegebene Zeichen, das nämlich Reibungs geräusche bei Druck mit dem stethoskop stärker werden, was allerdings Endocardiale Geräusche nicht thun."[2]

The pressure test shows itself in two ways, (I.), when pressure over the region of the heart elicits a friction sound that was previously absent; and the other, (II.), when pressure made over the seat of a friction sound intensifies, changes, or modifies that sound.

I. *Influence of Pressure over the region of the Heart in exciting a Friction Sound not previously audible.*—Pressure made with the stethoscope over the region of the heart elicited a friction sound not otherwise audible in twenty-nine of the forty-four cases that are included in the tables of cases of pericarditis given, in all of which cases the acme of the pericardial effusion was observed. As might be expected, it was usually (1) during the period of the commencement of the attack or (2) that of its decline that this sign was observed; and a friction sound otherwise latent was also thus brought into play by pressure (3) at the time of the acme of the effusion in four patients whose cases have already been touched upon at page 564, and in one case during a second acme of the effusion.

1. *Friction Sound excited by Pressure during the onset and early period of the attack of Pericarditis.*—In eight cases, as has just been stated, the attack of pericarditis first declared itself by a friction sound induced by pressure over the region of the heart. As a rule this sound, so awakened, was smooth in character. In three instances it appeared as a single or double friction murmur, in one as a whiff, and in one as a soft to-and-fro sound. In the other three cases, however, the rubbing sound was more marked, being harsh and systolic in one, of a winnowing character in another, and creaking, in the third of those cases. In three of these eight cases, pressure was required to bring out the friction sound over the right ventricle during the advance of the effu-

[1] Dublin Journal, iv. 60.

[2] Situation of the Internal Organs. Prov. Med. Times., xii. 52.

[1] Prov. Med. Trans., xii. 540.

[2] Friedrich, Die Krankheiten des Herzens, page 229.

sion. The friction sound was excited by pressure made, in six cases over the sternum, in one over the fourth cartilage, and in one over the heart. As a rule the spontaneous friction sound partook somewhat of the character of the friction sound previously generated by pressure. Thus it was creaking in the case in which it was originally creaking; harsh in one of those in which it was harsh; to-and-fro in that in which it was to-and-fro; rather smooth in the patient with a systolic friction murmur; and a double friction murmur prevailed through the long history of the fatal case, in which a double friction murmur was originally aroused by pressure.

The acme of the pericardial effusion usually occurred in these cases very soon after the first appearance of the excited friction sound, or from the first to the third day, in six of the eight cases.

2. The four cases in which a friction sound, otherwise absent, was elicited by pressure during the acme of the disease have been already considered under their proper heading at page 564.

3. *Friction sound excited by Pressure during the decline of the effusion into the pericardium; and during the dying away of the attack.*—In the great majority of the cases in which pressure was required to elicit the friction sound during the period of the decline of the pericardial effusion, this sign was a prelude to the dying away of the friction sound. Thus in nineteen of the twenty-four cases that belong to this class the frottement never again appeared as an independent sound; and the attack of pericarditis was coming to an end. In three of the cases the friction sound, after being for a time only audible when excited by pressure, reappeared for from five to ten days as an independent to-and-fro sound. There was a complete suspension of the friction sound in connection with extreme general illness in two of these cases, and the return of the spontaneous friction sound was in both of them associated with improvement of health, and was preceded by the appearance of a pressure friction sound.

The friction sound became inaudible except on pressure in nearly one-half of the cases under examination during the first four days after the acme of pericardial effusion (11 in 24); and in more than one-half of them this sign came into play from five to twenty-one days after the occurrence of the acme (13 in 24).

The character of the spontaneous friction sound last observed before the pressure friction sound was called forth was, with few exceptions, decidedly of a subdued tone.

The lower two-thirds of the sternum was the favorite seat of the pressure friction sound which was heard in eleven of the cases over that bone, including two in which it was heard over the ensiform cartilage. In seven of the cases the rubbing sound was excited by pressure over the cartilages from the third to the fifth, in one other instance over the second space, and in one over the fourth space. Besides these cases the pressure friction sound was heard over the heart in one case, the right ventricle in three, and the apex in three.

II. *Influence of Pressure over the Region of the Heart in intensifying a Friction Sound already present.*—Pressure exercised a marked influence on the friction sound in all but one of the forty-four cases under inquiry, and in that single exception there is no mention of the employment of pressure over the region of the heart during the attack of pericarditis. Pressure, therefore, as a means of diagnosis, and of illustrating the clinical conditions of the friction sound in pericarditis, is essentially interwoven into every part of what has gone before in relation to friction sound in that affection: and one part has been devoted to the study of cases in which a soft friction sound audible over the heart at the time of the acme of the effusion into the pericardium, was converted by pressure into a harsh rubbing noise (see page 564). It is not, therefore, needful to give here again in a detached form what has already appeared distributed naturally through the preceding pages.

In four instances or observations, an endocardial murmur was masked on pressure by the occurrence of a friction murmur or friction sound. A friction murmur was modified by pressure in fifteen instances; a systolic murmur being intensified (in 3), rendered double (in 1), or transformed into a double friction sound (in 1), by the employment of pressure; and by the same means a double friction murmur was intensified in five and converted into a double friction sound in four instances. In a few instances (3) a friction sound resembling a murmur acquired its complete frictional character by pressure; and in a greater number a systolic friction sound was thus intensified (in 4), or rendered double (in 5). An ordinary friction sound usually double, sometimes soft or grazing (in 18), sometimes of the usual to-and-fro character (in 38), sometimes harsh (in 18), was intensified, or altered in tone, or rendered more harsh in seventy-four instances or observations. As a rule a succession of observations was made upon each case, and the same patient often reappears again and again under the varying phases of the friction sound, and of the influence of pressure upon that sound.

I have not, as a rule, illustrated in this summary the various transformations that the friction sound may undergo

under the touch of pressure; but those two remarkable noises, the grating and the creaking friction noises, have been separately analyzed, and all the instances in which either of those sounds replaced another character of friction sound, or was strengthened by pressure, are given in the previous summary.

A friction sound of indefinite quality was rendered grating by pressure in six instances, and in two a grating friction sound was intensified or rendered more harsh by pressure. A creaking friction sound was in an especial manner the offspring of pressure when applied over the seat of an ordinary friction sound, since in six instances a friction sound, double in all but one, was rendered almost creaking by pressure, and in twelve instances, various kinds of friction noise, grating, harsh, smooth, and murmuring, were transformed by pressure into a creaking sound; while in two others, pressure converted a systolic creaking sound into a double creaking sound. These eighteen instances occurred in fourteen different cases. In each of two of these patients a creaking sound was excited by pressure four different times in the course of the clinical history of the case; showing a strong tendency to the repeated recurrence of this sign when it has been once excited. In six cases a creaking friction sound was rendered more intense by pressure, and only one of these cases appears also among those just spoken of in which an ordinary friction sound was converted by pressure into a creaking sound.

Although I have only noticed in the summary those two more striking noises, the grating and the creaking, as being excited by pressure, yet there are many other friction sounds of a definitely individual character that are thus brought into existence. These sounds differ in no essential respect from those that are spontaneously excited from within by the simple rubbing of the heart against the pericardium, when their opposing surfaces are covered with roughened lymph. Pressure over the heart affected with pericarditis excited—either originally or by transformation, among my various cases—a single and a double friction murmur; a whiff; a single, and more often a double brush; rustling, grazing, scraping, scratching, and sawing friction sounds; a double sound like that made by rubbing with sand-paper; and a peculiar double sound, broken during the diastole, that brings to my ear a noise like that made by sharpening a scythe. A to-and-fro sound was not unfrequently excited by pressure. I again and again noticed that under the influence of pressure the two friction sounds, and especially the diastolic one, became more continuous.

Owing to the increased intensity and continuousness of the friction sound causd by pressure over the heart in pericarditis, the natural sounds of the heart which were previously audible side by side with the friction sound, but were not strictly rhythmical with it, were frequently silenced under the influence of pressure.

The Movements of Respiration in Pericarditis.

In the Cases included in the following Table the movements of respiration were observed with the aid of the chest measurer.

TABLE SHOWING THE MOVEMENTS OF RESPIRATION IN PERICARDITIS.

I.—Cases in which the Respiratory Movements of both the Chest and Abdomen were observed.

* Explanation.—These figures indicate the movements of respiration in hundredths of an inch.

Female, æt. 16. Mitral murmur.

1st day. Friction whiff on pressure, pain left side.	*Rib.*	*Rt.*	*Lft.*	4th day. Acme of pericardial effusion.	*Rib.*	*Rt.*	*Lft.*	5th day. Pain in epigastrium.	*Rib.*	*Rt.*	*Lft.*
	2d	15*	8*		2d	25*	15*		2d	9*	12*
	5th	5	2		6th	6	5		6th	3	3
	9th	9	7		9th	14	6		9th	5	5
	abdom.	5	9		abdom.	6	5		abdom.	3	3
	abd. below ens. cartil.		−10		abd. below ens. cartil.		−8		abd. below ens. cartil.		−6

7th day. Better, pericardial effusion less.	*Rib.*	*Rt.*	*Lft.*	19th day. No friction sound; better.	*Rib.*	*Rt.*	*Lft.*	26th day.	
	2d	15	12		2d	9	7		
	5th	6	5		6th	3	2		
	9th	9	7		abd. below ens. cartil.		−4		abd. below ensiform cartil. 10
	abdom.	10	3		abdomen at navel		7		abd. at navel 15
	abd. below ens. cartil		−3						

Female, æt. 20. Mitral murmur, disappear on recovery.

9th day. Acme of pericardial effusion, less pain heart, respirat'n 52.	*Rib.*	*Rt.*	*Lft.*	10th day. Resp. 48.	*Rib.*	*Rt.*	*Lft.*	12th day. Feels better. Resp. 50.	*Rib.*	*Rt.*	*Lft.*
	2d	5	4		2d	8	4		2d	18	12
	5th	2	2		6th	1	1		6th	5	3
	6th	3	3		7th	7	3		abdm.	3	2
	9th	7	5		9th	7	4		abd. below ens. cartil.		0
	abdm.	6	0		abdm.	4	4		abd. at navel		0
	abd below ens. cartil.		−3		bel. ens. car.		−6				
					abd. at navel		7				

15th day. Friction sound more limited; feels better.

Rib.	*Rt.*	*Lft.*
2d	15	15
6th	4	3
9th	12	8
abd. below ens. cartil.		0
abd. at navel		5

18th day. Lying on right side, friction sound on pressure.

Rib.	*Rt.*	*Lft.*
2d	14	12
6th	7	4
9th	10	7
abdm.	3	5
abd. below ens. cartil.		0
abd. at navel		4

21st day. Better, aspect good, resp. 30, no friction sound.

Rib.	*Rt.*	*Lft.*
2d	12–20	10–15
6th	5	?
9th	10	"
abdm.	8	10
abd. below ens. cartil.		4
do. deep br'th		20
abd. at navel		5

22d day. Weak.

below ens. cartil.	5
ditto, deep breath	25

33d day.

Below ensiform cartilage, deep breath	50

37th day.

Below ensiform cartilage, deep breath	90

Female, æt. 15. Mitral valve disease.

1st day. Pain left side, ill a week. No friction sound.

Rib.	*Rt.*	*Lft.*
2d	20	15–20
6th	6	3
9th	4	7
abdm.	6	6

8th day. Acme. resp. 54, pain side.

Rib.	*Rt.*	*Lft.*
6th	3	3
9th	9	4
abd. below ens. cartil.		–1

12th day. Better, but resp. 55, no friction sound.

Rib.	*Rt.*	*Lft.*
2d	18	15
6th	6	2
9th	9	2
abd.	1	–3
abd. b. ens. c.		–2

Male, æt. 27. Mitral murmur.

2d day. Acme, very ill, resp. 36.

Rib.	*Rt.*	*Lft.*
2d	7–9	7–10
6th	4	4
9th	9	5
abdm.	7	12

3d day. Better.

Rib.	*Rt.*	*Lft.*
2d	6	7
6th	6	3
9th	6	9
abd. below ens. cartil.		12

7th day.

Rib.	*Rt.*	*Lft.*
2d	6	7
6th	4	2
9th	4	7
abd. at navel		20

Female, æt. 18. Mitral murmur.

5th day. Acme, pain over heart.

Rib.	*Rt.*	*Lft.*
2d	30	20
6th	7	6
9th	10	10
abd. below ens. cartil.		–2

24th day. Slight friction sound

8th day. Pain in chest, friction sound.

Rib.	*Rt*	*Lft.*
2d	30	20
6th	6	5
9th	15	7
abdm.	–3	–2
abd. below ens. cartil.		–4
abd. at navel		0

11th day. Acme.

Rib.	*Rt.*	*Lft.*
2d	30	20
6th	6	3
9th	10	7
abdm.	6	3
abd. below ens. cartil.		–4
abd. at navel		0

Male, æt. 23. Mitral murmur, established on recovery.

7th day. 3 days after acme, very extensive friction sound.

Rib.	*Rt.*	*Lft.*
2d	20	20
6th	6	5
9th	5	

10th day. Improving, less friction sound; left pleurisy.

Rib.	*Rt.*	*Lft.*
2d	25	20
6th	9	10
9th	13	10
abdm.	12	5
abd. below ens. cartil.		–1

13th day. Better, very slight friction sound.

Rib.	*Rt.*	*Lft.*
2d	15	12
6th	6	4
9th	13	12
abdm.	6	10
ab. b. ens. car.		2
abd. at navel		6

Male, æt. 25. Mitral murmur.

6th day, Acme, lessening pain over hear..

Rib.	*Rt.*	*Lft.*
2d	20	20
4th	10	10
7th	9	10
9th	9	10

10th day, Resp. 22, better friction sound.

Rib.	*Rt.*	*Lft.*
2d	8	7
6th	16	8
10th	9	9
abdm.	25	20
abd. below ens. cartil.		20
abd. at navel		40

13th day. Better, sits up in bed, friction sound on pressure.

Rib.	*Rt.*	*Lft.*
2d	15	15
6th	10	10
abdm.	30	35
abd. below ens. cartil.		30
abd. at navel		40

Male, æt. 17. Mitral murmur, established on recovery.

8th day. Before acme pain in heart.

Rib.	*Rt.*	*Lft.*
7th	10	4
abdm.	12	7

12th day. Acme.—abdomen below ensiform cartilage, –7.

II.—Cases in which the Respiratory Movements of the Centre of the Abdomen were observed.

A.—*Cases observed* (1) *below the Ensiform Cartilage, and* (2) *at the Navel.*

Male, æt. 17. Aortic mitral murmur	12th day, second acme.	Below ensiform cartilage,	–3,	at navel,	4		
	21st day, no friction sound	"	"	"	0,	"	12
	35th day, ...	"	"	"	18,	"	20
Male, æt. 15. Mitral aortic murmur	4th day, acme, pain epigast.	"	"	"	6,	"	10
	5th day, after acme	"	"	"	5,	"	
	6th day, "	"	"	"	4,	"	
Female, æt. 17. Mitral murmur.....	2d day, acme?	"	"	"	0,	"	10
Male, æt. 17. Mitral murmur.....	1st day, acme	"	"	"	1,	"	12
	7th day, after acme	"	"	"	6,	"	
	15th day, friction sound	"	"	"	3,	"	12

B.—*Cases observed below Ensiform Cartilage.*

Male, æt. 22. Mitral murmur.....	4th day, acme	Movement below ens. cartil.	-2
	7th day, decline of fld.	" " "	6
	9th day, second acme	" " "	9
	29th day, well. Deep breath ..	" " "	110–170
Female, æt. 21. Mitral murmur.....	5th day, after acme	" " "	2
	11th day, improving	" " "	3
	32d day, clothes on. Deep breath	" " "	50
Male, æt. 15. Mitral aortic murmur	1st day, before acme	" " "	16
	3d day, acme	" " "	6
	11th day, well	" " "	20
Female, æt. 22. Mitral murmur.....	13th day, second acme	" " "	-2
	22d day, third acme	" " "	0
Female, æt. 19. Mitral murmur.....	9th day, after acme	" " "	1
	11th day, ...	" " "	-7
Female, æt. 24. Mitral murmur.....	17th day. acme	" " "	-3
	25th day, ...	" " "	17
Male, æt. 14. Mitral murmur..... ..	6th day, after first acme	" " "	3
Female, æt. 25. Mitral murmur..... ..	4th day, acme, or after	Mvt. bel. ens. cart. or lower,	4
Male, æt. 26. Mitral murmur..... ..	5th day, before friction sound..	Movement below ens. cartil.	5
Male, æt. 35. Mitral murmur..... ..	8th day, no friction sound ..	" " "	20

The movements of respiration were affected in pericarditis in three different relations: (1) those of the ribs; (2) those of the abdomen on each side, just below the eighth cartilage; and (3) those of the centre of the abdomen.

(1) The respiratory play of the upper ribs was more than doubled in extent in three-fourths of the cases observed (5 in 7), so that respiration was as a rule high. This was due to the arrest or restraint of the action of the diaphragm caused by the extensive inflammation of the central tendon of the diaphragm, where it forms the floor of the pericardium.

In one of the two exceptional cases, the movements of the second ribs were not at all or only slightly augmented throughout the whole period of the illness; but in the other case, in which the respiration was greatly accelerated, the action of those ribs, which was slight during the acme of the affection, was much increased during the decline of the effusion.

The respiratory movement of the ribs on the left side of the chest was less than that of those on its right side, as might naturally be expected, in more than one-half of the cases (5 in 8); but in the remaining three patients the action of the two sides was nearly equal both during the acme and the decline of the pericarditis. The difference in the movement of the two sides of the chest was not, as a rule, limited to the ribs adjoining the pericardium, but extended along their whole range, from the second to the ninth. The study of the Table will show, however, that there were some exceptions to the rule that the play of the ribs was restrained throughout on the left side; since in two of the three cases in which the two sides of the chest moved with equal freedom, the ninth left rib was greatly restrained in its movements.

(2) The lateral movements of the abdomen below the eighth cartilages were greatly restrained in three-fourths of the cases (6 in 8); and the respiratory play of the left side of the abdomen was much less than that of its right side in the same proportion of cases (6 in 8).

(3) The inspiratory movement of the abdomen below the ensiform cartilage was either reversed (in 12), arrested (in 1), or restrained (in 6) in every case of pericarditis in which that sign was observed. This is at once accounted for by the inflammation, in that disease, of the central tendon of the diaphragm where it forms the floor of the pericardium, which leads to the virtual paralysis of the central portion of the diaphragm. This fact, that the anterior wall of the epigastric space, instead of advancing, recedes during inspiration, gives us a physical side of great value in the diagnosis of pericarditis, and of the advance and decline of that disease. Thus in the first case in the Table a girl, aged 16, the anterior wall of the abdomen below the ensiform cartilage fell backwards during inspiration for the tenth of an inch during the three early days, when the disease was at its acme; then, as the tide turned and the effusion diminished, the abdomen receded less and less up to the seventh day, when it did so for only the fiftieth of an inch; after this it regained its natural forward movement, and on the twenty-sixth day the abdomen at the epigastric space advanced (the tenth of an inch) as it had receded on the day of admission. In the other case the front of the abdomen advanced the sixth of an inch on the day of admission, when the pericarditis had scarcely pronounced itself; the sixteenth of an inch on the third day, when it had reached its acme; and the fifth of an inch on the eleventh day, when it had declined and disappeared. In my paper on the movements of respiration I showed that in health the abdomen at the navel advanced during inspiration a quarter of an inch or a little more, but I did not ascertain the respiratory movement at the epigastric space. A short time ago I observed, with Mr. Rossiter, the respiratory movements of the abdomen in eleven patients in St. Thomas's Hospital, several of whom were convalescent, and one had pericarditis;

when we found that the inspiratory advance at the epigastric space varied from the sixth to the fifth of an inch. The latter was also the extent of the advance in two healthy men. I consider that this forward movement fairly represents the healthy respiratory play of the part in question; that in pericarditis, as a rule, the whole of this advance is lost; and that in addition the play is reversed to the extent of from the fiftieth to the tenth of an inch. It is worth noting, in conclusion, that in the case of pericarditis observed by Mr. Rossiter and myself in St. Thomas's Hospital, a boy, aged 12, in whom the disease was at its height, the wall of the abdomen receded during inspiration at the epigastric space from the sixteenth to the twentieth of an inch, and at the navel from the thirty-fifth to the fiftieth of an inch.

Pericarditis in Bright's Disease of the Kidneys.

Dr. Bright, in the first volume of Guy's Hospital Reports, gives 100 cases of albuminuria, seven of which, according to the tables, and eight according to his description, had pericarditis. Subsequently Dr. Gregory and Sir James Christison, in Edinburgh; Martin Solon, Becquerel, and Rayer, in France; and Malmsten, in Germany, gave each of them a series or summary of cases of Bright's disease, in all of which cases, except those communicated by Malmsten, pericarditis was either infrequent or absent.

Dr. Taylor called attention, in 1845, to the large proportion in which cases of pericarditis are affected with Bright's disease, and to the frequency with which pericarditis occurs in cases of Bright's disease. He found that out of thirty-one patients with pericarditis, nine, if not eleven, had Bright's disease; and that of fifty post-mortem inspections of cases with Bright's disease, five, or one in ten, had pericarditis.

Several years later, or in 1851, Frerichs published his important work on Bright's disease, which contains a valuable table showing various conditions that existed in 292 cases collected by him from various sources, and including 21 observed by himself. He states that in 13 of those collected cases there was pericarditis; that is in only 4½ per cent., or 1 in 22 of the cases. This return, which has been, and still is, much quoted, gives a lower proportion of attacks of pericarditis in Bright's disease than in the cases given or enumerated by Dr. Bright (7 or 8 per cent., or 1 in 14 or 12), Dr. Taylor (10 per cent., or 1 in 10), M. Rayer (5·4 per cent., or 1 in 18), and Dr. Gregory (5 per cent., or 1 in 20); and a higher proportion than in the cases observed by Becquerel (4·6 per cent., or 1 in 62). Frerichs appears to have overlooked some of the cases of pericarditis in his analysis. Te test his figures, I examined as nearly as I could the same cases or tables given by the observers quoted by him, and I find that in a total of 326 cases, 17 or 19 had pericarditis, or about 5·5 per cent., or 1 in 18.[1]

During the nineteen years, ending in 1869, 285 cases of Bright's disease were examined after death in St. Mary's Hospital, and of these 25 or 1 in 11·3 or 8·8 per cent. were affected with pericarditis; which was present therefore somewhat more frequently in those cases than in 1691 collected cases of Bright's disease, 136 of which, or 1 in 12·3 or 8·17 per cent. had pericarditis.

Besides the twenty-five cases of pericarditis noted in the records of St. Mary's Hospital, there were fifteen of partial or doubtful pericarditis; but these cases ought not, I think, to be taken into the general account.

If we separate the various forms of Bright's disease occurring in St. Mary's Hospital from each other we shall see the proportion in which each form was affected with pericarditis.

SUMMARY.

Acute Bright's disease, from scarlet fever, total number, 6; affected with pericarditis, 0; with partial pericarditis, 1.

Acute Bright's disease, *not* from scarlet fever, total number, 15; affected with pericarditis, 2, or 1 in 7·5, or 13·3 per cent.; with partial pericarditis, 0.

Fatty or large white Kidney, total number, 62; affected with pericarditis, 1, or 1 in 62, or 1·6 per cent.; with partial pericarditis, 5.

Contracted Granular Kidney, total number, 128; affected with pericarditis, 13, or 1 in 10, or 10 per cent.; with partial pericarditis, 7.

Granular Kidney of natural or large size, total number, 34; affected with pericarditis, 3, or 1 in 11·3, or 8·8 per cent.

Granular Kidney, grand total number, 162; affected with pericarditis, 16, or 1 in 10, or 10 per cent.; with partial pericarditis, 7.

Lardaceous disease of Kidney, actual and

[1] Frerichs. Dr. Bright, 100 cases; Sir James Christison, 14; Dr. Gregory, 37; Martin Solon, 8; Rayer, 48; Becquerel, 45; Bright and Barlow, 10; Malmsten, 9; Frerichs, 21; Total, 292. Author. The same authorities respectively; 100, 14, 39, 10, 55, 45, 9, 33, 21; Total, 326.

Cases of pericarditis in the above, Frerichs, 13; Author, 17 or 19.

probable, total number, 22; affected with pericarditis, 2, or 1 in 11, or 9 per cent.

Nature of Kidney disease doubtful, 11; affected with pericarditis, 4, or 1 in 2·7, at 36 per cent.; partial pericarditis, 2.

Total number of cases of Bright's disease, 285; affected with pericarditis, 25, or 1 in 11·3 or 8·8 per cent.; with partial pericarditis, 15.[1]

Calculus in kidney, pelvis, or ureter, or dilated pelvis (hydronephrosis), total number, 12; affected with pericarditis, 0.

Suppurative Nephritis from stricture, &c., total number, 13; affected with pericarditis, 1, or 1 in 13, or 7·7 per cent.

That I might enlarge the area of observation, I have brought together from various sources, including the returns from St. Mary's Hospital, the number of attacks of pericarditis in 1681 cases of Bright's disease; and the number of attacks of pleurisy, peritonitis, and pneumonia, in 1228 cases.

I have also given the number of cases with pericarditis, pleurisy, and peritonitis, pneumonia, pulmonary apoplexy, and purulent deposit or abscess of the lung; and certain conditions of the heart and aorta in the various forms of Bright's disease among the 285 cases examined at St. Mary's Hospital; distinguishing also those cases in which the heart was small, of natural size, rather large, and large or very large, giving separately those various conditions as they appeared in the cases affected with pericarditis.

Among the cases of Bright's disease collected from various sources, 8·1 per cent. or 1 in 12·3 were attacked with pericarditis.

These cases are arranged in three sections devoted respectively to England, Germany, and France; and the occurrence of pericarditis in Bright's disease is here shown to be most frequent in Germany (1 in 9·5, or 10·4 per cent.), and least frequent in France (1 in 33, or 3 per cent.), while it is of medium or average frequency in England (1 in 11·9, or 8·4 per cent.).

Comparative frequency of Pericarditis in the various Forms of Bright's disease.—I shall here inquire into the frequency of pericarditis in the different forms of that disease.

Pericarditis is not frequent in cases of acute Bright's disease from scarlet fever in the young, since it only occurred in 1 in 14, or 7 per cent. of the patients under 16 years of age. The tendency to pericarditis in children in such cases is slight, as was pointed out to me by Dr. Dickinson, who kindly supplied me with the valuable tables of his cases of that class, amounting to 21. Pericarditis is on the other hand frequent in acute Bright's disease in the adult, since it was present in 1 in 6½ or 15·4 per cent. of those cases. The value of these returns has been greatly added to by the cases of acute Bright's disease kindly communicated to me by Dr. Greenfield.

During the transitional period, when acute Bright's disease slowly gives place to the fatty or large white kidney, pericarditis is probably frequent, since it occurred in one of Dr. Dickinson's four transitional cases.

When, however, acute Bright's disease instead of recovering passes into the second or chronic stage, in the form of large white kidney, the tendency to general pericarditis disappears, since it only occurred in 1 in 27 or 3·7 per cent. of the collected cases, and one in 62, or 1·6 per cent. of the St. Mary's Hospital cases, and the kidney in that single case was in the third or contracted stage of fatty disease. Five, however, of the St. Mary's Hospital cases with fatty kidney had partial pericarditis, showing that this affection, although still inherent, does not tend to develop itself in that form of the disease.

The two great and opposite forms of Bright's disease, the fatty kidney, or the chronic stage of acute Bright's disease, and the contracted granular kidney, show a marked difference in the proportion with which they were respectively affected with pericarditis; which attacked those with contracted granular kidney from six to four times as often (1 in 10[1] and 1 in 6[2]) as those with fatty kidney (1 in 62[1] and 1 in 26·6[2]).

Cases of lardaceous disease of the kidney have pericarditis with a moderate or average frequency (1 in 11, or 9 per cent.,[2] and 1 in 13·3, or 7·5 per cent.[2]).

Inquiry into the influence respectively of the fatty kidney, and the contracted granular kidney, in the production of pericarditis.—When inquiring into the influence of these two forms of Bright's disease in the production of pericarditis it may be well to consider two points which appear to be associated with the production of pericarditis, though for different reasons; (1) the proportion in which cases with fatty and contracted granular kidney were affected respectively with pleurisy, peritonitis, and pneumonia: and (2) the relative proportion in which the heart was enlarged and its left ventricle was hypertrophied in those two forms of disease; and the im-

[1] For details of the cases of partial pericarditis, see pages 592, 593.

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

mediate relation, if any, that the enlarged heart may have had to the production of pericarditis.

1. Pleurisy attacked 60 of the 285 cases with Bright's disease occurring in St. Mary's Hospital (1 in 4·8 or 21 per cent.[1] and 1 in 6 or 16·4 per cent.[2]). It will thus be seen that in these cases of Bright's disease pleurisy was twice as frequent as pericarditis (1 in 11·3[1] and one in 12·3[2]). We have here a marked difference between the pericarditis of acute rheumatism and the pericarditis of Bright's disease, since while in the former disease, or acute rheumatism, the inflammation of the pericardium is much more common than that of the pleura; the pleurisy when present, being usually either due (1) to the spreading of the inflammation of the pericardium to the pleura, or (2) to pulmonary apoplexy which is the consecutive effect of the double inflammation of the heart, inside and out; in the latter affection, or Bright's disease, the pleurisy is an independent affection, and is, as we have just seen, twice as frequent as pericarditis in the cases under inquiry.

The same in principle may be said of peritonitis, which is practically unknown in acute rheumatism; while it occurs nearly as often as pericarditis in Bright's disease; the numbers being 93, or 1 in 13,[2] and 19, or 1 in 15[1] of peritonitis against 100 or 1 in 12·3[2] and 25 or 1 in 11·3[1] of pericarditis.

Two-fifths of the cases of pericarditis were also affected with pleurisy (10 in 25) and three-fifths were free from that affection (15 in 25); while only 2 in 25 of those cases had peritonitis.

The relative frequency of pleurisy and peritonitis on the one hand, and pericarditis on the other, varied much in the different forms of Bright's disease.

In acute Bright's disease from scarlet fever in the young, pleurisy occurs three times (1 in 5) and peritonitis twice (1 in 7) as often as pericarditis (1 in 14); but it is otherwise in acute Bright's disease in the adult, not from scarlet fever, since in such cases pericarditis is as frequent as pleurisy (each 1 in 6·5), while it is twice as frequent as peritonitis (1 in 11·5).

Pleurisy attacks many more cases (1 in 4[1] and 1 in 4·5[2]) with fatty kidney than pericarditis (1 in 62[1] and 1 in 27[2]); while in those with contracted granular kidney, pericarditis (1 in 10[1] and 1 in 6[2]) occurs, judging by the collected cases, nearly as often as pleurisy (1 in 4·3[1] and 1 in 4·8[2]). Although pleurisy is rather more frequent, pericarditis, as we have seen, is much less so in cases with fatty than in those with contracted granular kidney; and it is therefore evident that the causes producing the two inflammations have but little in common, and that the one rarely excites the other. Peritonitis occurred twice as often (1 in 31[1] and 1 in 15[2]) as pericarditis in cases with fatty kidney, while pericarditis attacked three times as many as peritonitis (1 in 21) in those with contracted granular kidney.

Pleurisy and peritonitis (each 1 in 10·8[2]) were both of them more frequent than pericarditis (1 in 13·3[2]) in cases of lardaceous disease of the kidney.

Pneumonia, which when it occurs by itself is an occasional cause of pericarditis, while it is less common (1 in 6·4[1] and 1 in 7·6[2]) than pleurisy (1 in 4·8[1] and 1 in 6[2]) is more common than pericarditis in cases of Bright's disease. Those two secondary affections, pneumonia and pleurisy, were of exactly equal frequency in cases of acute Bright's disease, whether from scarlet fever or not; so that what has been said with regard to the latter of those affections applies to the former.

Pneumonia was common (1 in 4[1] and 1 in 6[2]) and pericarditis was rare (1 in 62[1] and 1 in 27[2]) in cases with fatty kidney. It was almost the reverse in those with contracted granular kidney, in which pneumonia (1 in 10[1] and 1 in 9[2]) scarcely equalled pericarditis in number (1 in 10[1] and 1 in 6[2]). The proportion of pneumonia was, therefore, about twice as great in cases with fatty, as in those with contracted granular kidney, while pericarditis, rare in the former, was frequent in the latter form of the disease, making it evident that there was little in common between the production of pneumonia and that of pericarditis in these cases. Pneumonia was present in only one-third of the cases of Bright's disease that were affected with pericarditis (8 in 25).

2. Enlargement of the heart, usually with hypertrophy of the left ventricle, was present in one-half of the cases of Bright's disease under review (129 in 259) in which the size of the heart was described. The heart was large in more than half of the cases of pericarditis in which the size of the heart was defined (10 in 19[3]); or 10 in 129 of the total number of cases of Bright's disease with enlargement of the heart. Pericarditis occurred in six cases in which the heart was of natural size (or 6 in 61). It would thus appear that 1 in 10·1 of the latter in which the heart was natural in size, and 1 in 12·9 of the former, with hypertrophy of the heart, had pericarditis. This would seem to say that hypertrophy of the heart had no apparent influence in the production of pericarditis in these

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

[3] The size of the heart was doubtful in six cases with Pericarditis.

cases. If, however, we add the cases in which the heart was small (23), none of which had general pericarditis, to those in which it was natural in size (61), we find that 6 in 84, or 1 in 14 of those combined cases, had that affection. If to these we join the cases in which the heart was rather large (45) 3 of which had pericarditis, the result is that 9 in 129, or 1 in 14·3, were thus attacked. From this analysis it would appear that enlargement of the heart exercised a definite but not a predominant influence over the production of pericarditis in cases of Bright's disease.

Although hypertrophy of the heart is absent in almost one-half of the cases of Bright's disease with pericarditis, we know that in every form and case of that disease, whether acute or chronic, fatty or granular, the action of the left ventricle is unduly strong; for it has to send the poisoned blood through vessels of great tension that oppose resistance to the onflow of the blood. The result is that in every case of Bright's disease, the left ventricle, whether hypertrophied or not, is beating with undue force; and thus tends, by the pressure of its walls with undue force against the pericardium, to induce pericarditis. The heart is prevented from becoming enlarged in many cases of Bright's disease by the exhausting loss of albumen, the general waste, and the lowering character of the disease. This especially applies to cases of fatty, lardaceous, and suppurative kidney. The left ventricle, notwithstanding the great waste of tissue that goes on in those cases, is actually hypertrophied in a certain proportion of them; and it is so in the greater number of those with acute Bright's disease, in spite of the waste of tissue entailed by the great loss of albumen and blood in such cases. We have already seen that in acute rheumatism, over-action of the heart tends to induce pericarditis. It is, therefore, consistent with analogy, reason, and the clinical facts, that in Bright's disease over-action of the heart should increase the tendency to pericarditis, that tendency being already resident in the disease. May it not be that on the one hand, the lessened force of the heart, induced by the weeping of albumen, dropsy, and other secondary wasting diseases in cases with fatty disease of the kidneys, explains to some extent the rarity of general pericarditis (1 in 62[1] and 1 in 27[2]), and the comparative frequency of partial and undeveloped pericarditis (1 in 12·4), in that disease? and that on the other hand, the increased size and action of the heart in cases with granular kidney, which usually lose little albumen, are not dropsical, and are free from exhausting secondary disease, tend to increase the frequency of general pericarditis in that affection (1 in 10[1] and 1 in 6[2])?

Although the cases of partial pericarditis, which amounted to fifteen, cannot be classed rightly with those of general pericarditis; for the partial variety appears to have a tendency to remain partial, and those cases are not usually included among those with pericarditis, yet those cases ought to be studied. One of the fifteen cases of partial pericarditis had acute Bright's disease from scarlet fever (1 in 6, or 16·6 per cent.); five of them had fatty kidney (5 in 62, or 1 in 12·4, or 8 per cent.); seven of them had contracted granular kidney (7 in 129, or 1 in 18·3, or 5·5 per cent.); and in two the state of the kidney was not specified.

The proportion in which partial and general pericarditis respectively attacked the different forms of Bright's disease somewhat correspond.

In four of the cases of partial pericarditis the heart was very large (1 in 32·2), and in three it was rather large (1 in 15); while in five of them the heart was of natural size or small (1 in 16·8), and in three the size of the heart was not described,

It thus seems that great enlargement of the heart does not favor the persistence of partial pericarditis, but rather tends to develop it into general pericarditis.

Amount of Fluid in the Pericardial Sac in Pericarditis from Bright's Disease.—The amount of fluid in the pericardial sac varied considerably in the twenty-five cases of pericarditis from Bright's disease, the smallest quantity being two drams, and the largest about a pint, in which case the contents of the sac were purulent.

In one-fifth of the cases (5) the contents of the pericardium are not described; and in one-fifth of them (5) there were recent adhesions. The sac contained only a small quantity of serum, or not more than one ounce in one-third (5) of the remaining cases (15); a moderate amount, or a few ounces, in another third of them (6); and much fluid, eight ounces in one instance, a pint in another, in the remaining third (4) of those cases. It is evident that the presence of adhesions, or of a small, a moderate, or an abundant amount of fluid in the pericardium, depends on the stage of the pericarditis at the time of death; and that in the several cases the fluid had either been removed, or was lessening, increasing, or at its height, when the final observation was made. It may, I think, be admitted that in the pericarditis of Bright's disease there is

[1] The cases of Bright's disease examined after death in St. Mary's Hospital.

[2] The collected cases.

[1] The cases of Bright's disease examined after death in St. Mary's Hospital.

[2] The collected cases.

less effusion in the pericardium than in rheumatic pericarditis; but from the evidence here given it would appear that there is no very material difference in the amount of fluid in the sac at the time of death in the two classes of cases.

Character of the Exudation on the Surfaces of the Heart and Pericardial Sac in Pericarditis from Bright's Disease.—In a small proportion of cases the lymph covering the heart and lining the pericardium in case of pericarditis from Bright's disease presents the same pale and rough surface, firm to the finger, with "cat's-tongue"-like projections, so usual in pericarditis from acute rheumatism. It was thus in two of the twenty-five cases that were examined after death at St. Mary's Hospital. In two other cases also, both of acute Bright's disease, a rather firm layer of fibrin easily peeled off from the heart, leaving a finely-injected red surface underneath.

In the majority of cases of pericarditis from Bright's disease the exudation differs from that usual in rheumatic pericarditis. Universal adhesions of the heart, rare in the latter, are common in the former affection; the heart having been completely adherent in three instances, extensively so in one, and doubtfully so in another of those cases. There was pus in the sac in two cases. The lymph—was soft, granular, imperfectly organized, or in patches in six, in two of which the presence of pericarditis was perhaps doubtful; or was bloody or very red on the surface, or mixed with blood in three of the twenty-five cases of pericarditis from Bright's disease. These conditions, which affected nearly two-thirds of those cases, are rare or unknown in rheumatic pericarditis. The remaining cases were less definite in character, the heart in four of them having been covered by recent lymph, while in two the pericardium was affected with "recent pericarditis."

Appearances in Partial Pericarditis.—The cases of partial or doubtful pericarditis varied much in their features. In four of them flakes of lymph floated in the serum contained in the pericardial sac, the surfaces of the heart not being named. Pericarditis was limited, slight, or in traces or patches in seven other cases, and in two more it was highly vascular or congested. One case presented rough lymph easily detached, leaving an apparently healthy surface; and in the last instance there was a red fluid containing flakes of lymph in the sac, and lymph on the heart, the surface of which was healthy. These two cases, and the four in which flakes of lymph floated in the serum, were probably free from actual pericarditis.

Physical Signs of Pericarditis Occurring in Bright's Disease.—Dr. Taylor gives careful reports of nine cases of Bright's disease with pericarditis, in three of which there was a friction sound, while in six of them there was no definite sign of the affection. In three of these six cases there were complete recent adhesions, rendering friction sound impossible. In one of the three cases in which pericarditis was not discovered during life, a layer of soft lymph coated the heart, but there was no lymph on any part of the loose pericardium, and this appears to account for the want of friction sound. In one of the three cases that presented a friction sound, a double creaking noise was heard between the apex of the heart and the sternum; and the heart and sac were covered with soft, slightly rough lymph.

In two of the three cases without friction sound, excluding the three with complete adhesions, and in two of the three with friction sound, there was no adequate explanation, after death, of the absence of that sound in the two former cases, in which the opposed surfaces of the heart and sac were rough and scabrous; nor of its presence in the two latter cases in one of which there were extensive adhesions of the heart; while in the other the surface of the heart was simply red from fine injection, and there were but a few spots of lymph on the anterior coronary artery.

I possess notes of the symptoms during life, and the appearance after death of nine fatal cases of Bright's disease with pericarditis. I cannot find the notes of a tenth case with regard to which I find two lines of an abstract of symptoms. In seven of the cases immediate signs of pericarditis were observed, and in three of them the signs of pericarditis were not observed.

Cases in which the Signs of Pericarditis were not Observed.—In one patient, a man, aged 61, with granular kidneys, the heart, which was very fat, was covered and the sac was lined with recent lymph. On the third day after his admission, on which day he died, the heart's action to the left of the ensiform cartilage was loud; and loud mucous rattles were audible all over his chest. In the second case, a man, aged 47, the opposite surfaces of the pericardium, and the heart, at its base, and along the great vessels were rough with a deposit of fibrin. This patient was in the hospital fifty-two days, but there is only one note of the state of his heart, which was on the fifth day after his admission, when the sounds were rather loud.

I cannot find the notes of the remaining case with Bright's disease and pericarditis; but the following is the brief abstract preceding the notes of the examination after death. "At first, dou-

bling of the first sound, afterwards systolic murmur after epistaxis" so that friction sound was evidently not observed in this case.

Cases in which the Signs of Pericarditis were Observed.—(1) A creaking noise with a thrill was present in three of the seven cases of pericarditis with friction sound; (2) a creaking sound without a thrill in two of them, and (3) in the remaining two there was a "friction sound."

(1) *Cases with Thrill and a Creaking Friction Sound over the Seat of the Impulse, and Frottement extending far beyond and especially below the Region of the Pericardium.*—There were three cases of this class. One of them a woman, aged 32, who was in the hospital for a week, presented after death some fluid in the pericardium, and a rough deposit of recent lymph of a bright red color, which covered the heart and lined the sac. On the day after her admission a systolic murmur was audible over the cardiac region. Two days later, when she complained of pain going across the chest, the upper border of cardiac dulness was situated at the third space; and a rasping, creaking friction sound, chiefly systolic, was heard all over the front of the chest, and down to the eighth and ninth costal cartilages, its maximum intensity being at the centre of the sternum, and during the middle of the systole. Next day a strong thrill extended over the heart from the right of the sternum to the nipple, and as high as the third cartilage: and the creaking sound was triple, being exactly like that made by the rise and fall and rise in the saddle. On the following day, the fifth, the thrill was less intense, and there was a triple creak at the apex, the friction sound being still audible over the lower cartilage; and two days later she died.

The second patient, a woman, aged 27, with contracted granular kidney, and pericarditis, had several patches of recent lymph on the surfaces of the heart and the free pericardium, and presented a double thrill, a double creak, and an extensive friction sound, which were all absolutely suspended for one day, under the influence of flooding.

The third case, a man, aged 33, had mitral-aortic incompetence, and highly albuminous urine. The heart and pericardium were greatly increased in size, and the right ventricle was covered with a white fibrinous structure, rough to the finger, like a cat's tongue. On admission he had pain over the heart; and for two days, mitral and double aortic murmurs were audible. He became worse, and on the fourth day the diastolic murmur disappeared. On the ninth day he was drowsy, a strong thrill was felt with each impulse from the third cartilage to the fifth; a loud grating double friction sound was present over the seat of the thrill, the rubbing noise radiating thence up to the top of the sternum, down to the eighth cartilages, and to the left and right; a leather creak was audible at the apex; and a sound of a friction character was heard behind, over the dorsal spine. On the next day, when he died, the vibration had increased, and extended from the third to the seventh cartilages; it lessened in extent above, on inspiration, below, on expiration; and was accompanied by a loud creak during systole, and a fainter creak during diastole, the sound spreading from the seat of the vibration over the front of the chest, and the upper third of the belly.

(2) *Cases with a Creaking Friction Sound, no Thrill being Observed, over the Seat of the Impulse, and a Frottement extending beyond, and especially below the Region of Pericardial Dulness.*—One of the two cases of this class was a young married woman, with granular disease of the kidney. A firm coating partly in ridges and partly like a cat's tongue covered the heart and lined the sac. On her admission a creaking systolic friction sound was audible at the apex, in the fifth space. Four days later, when the pericardial dulness was at its acme, reaching up to the third cartilage, her respirations being fifty, the friction sound was no longer creaking but presented itself as an occasional brush; but three days after this, or on the eighth day, there was a loud leather creak over the whole region of the pericardium. After this the friction sound almost disappeared; but on the twelfth and preceding days it had again burst into full play as an extensive leather creaking noise, covering the whole pericardium, and extending down to the seventh cartilage; and eight days later she died.

In the second case, a man, aged 30, with small, probably granular, kidneys, recent, bloody, honeycombed lymph lined the pericardium and covered the heart. On the day of his admission the two sounds of the heart were indistinct. Next day the impulse was extensive, and a loud double creaking sound, more intense during systole, occupied the whole region of the heart, extending downwards to the seventh and eighth cartilages, and into the epigastrium. During the next few days the frottement was much smoother and more restricted in area. On the eighth day he was weak and in distress; the friction sound was audible over the whole pericardium, and beyond it, from the top of the sternum to the lower cartilage; and he could scarcely swallow or speak: and in the evening he died.

(3) *Cases with "Friction Sound."*—One of the two cases of this class, a man aged 38, with granular kidney of full size, had recent lymph over the whole surface

of the heart, and in some places the heart and pericardium were adherent by cord-like prolongations of lymph. On the fifty-seventh day there were doubling of the second sound, and a murmur over the third cartilage. On the seventy-fifth day, which was eight days before his death, "double friction sound over the pericardium," was noted for the first time. Three days later the pericardial friction sounds, which were scarcely audible without making pressure, were mingled with pleuritic friction sounds; but after this he was too ill for examination.

The other patient, an old woman, with contracted granular kidney and pericarditis, the whole surfaces of the heart and sac being covered by recent soft granular lymph, complained, on the twenty-first day after her admission, of great pain at the region of the heart. Next day there was pericardial dulness, and friction sound was present between the sternum and the left nipple; and three days later she died.

Several of these seven cases of Bright's disease and pericarditis presented certain broad features in common. In three of them a thrill or tactile vibration could be felt over the region of the heart's impulse, extending from the third to the fifth, the sixth, and in one instance the seventh cartilages. In one of those cases the thrill extended from the right border of the sternum across the chest to the nipple. In these three cases, and in two others in which a thrill was not observed, a loud sound like the creaking of new leather, usually double, but more intense and prolonged with the systole, was audible over the whole seat of the thrill, or when that was absent, over the region of the heart's impulse. The friction sound was, however, in none of the five instances restricted to the area of the thrill or impulse, or even of the distended pericardium; but extended upwards to the top of the sternum, downwards to the right and left along the seventh and eighth costal cartilages, and over and even below the ensiform cartilage. In these cases the widespread friction sound became softer in tone, and especially downwards, as it widened away from the focus of its greatest intensity. In two of these five cases with creaking and extended friction sound, the deposit of fibrin or lymph on the surface of the heart was firm and like a cat's tongue, in one of them it was rough, in one it was bloody and honeycombed, and in the fifth, patches of recent lymph were present on the heart.

In three of these cases there was a period of complete or partial suspension of the creaking and extensive friction sound; which after spreading with great intensity and over a large area, became silent or feeble and contracted in area for a time, and then suddenly burst forth again with full intensity, and over a wide space. It was evident that under these circumstances, some influences were at work exciting the heart at the time of the creaking and widespread friction sound, and depressing the heart when that sound ceased or became feeble. In one instance, the suspension of the thrill and creak was traced to the influence of flooding.

In the two other patients the surface of the heart is described as being covered with recent, and in one of them with soft, lymph. In neither of them is it noted that the coating of lymph was rough. In both of these cases it is simply stated that a "friction sound" was present over the region of the heart.

In all of these patients pressure intensified the friction sound.

Cases with a Friction Sound that were not Fatal, or not Examined after Death.—Besides these seven fatal cases of Bright's disease with pericarditis in which friction sound was observed during life, I find three other cases in which the signs of pericarditis were observed when the patients were in the wards.

One of these cases, probably fatal, admitted during the recess, very imperfectly recorded, presented a pericardial friction sound, which was chiefly present at and below the left nipple.

Another patient, a carpenter, aged 35, had Bright's disease and aortic regurgitation of some standing. On the eighty-second day he had great pain in the heart, and four days later a rough double noise resembling a friction sound was audible over the cardiac region. Four days after this there was dulness over the pericardium from the third space downwards, and pain over the heart, relieved by leeches; and next day a to-and-fro friction sound was audible over the heart, which continued for six days; after which, when he was in distress from aching over the heart, and sickness, the rubbing noise vanished, being replaced by the lost diastolic murmur of aortic regurgitation. This case left the hospital in improved health.

The last case of Bright's disease with friction sound, was one of great interest, a cab-driver, aged 45. His urine was loaded with albumen, and contained coarse granular and fatty casts. There was, on the fourth day, an extensive impulse, and a remarkable doubling of the first sound heard all over the region of the impulse, which was heard along with, but apart from, a peculiar pericardial friction sound chiefly systolic, which was audible for two inches below the nipple. This sound which was rasping at first, became creaking two days later, and five days after that, was only audible when pressure was made over the same spot, the sound being like that caused by rub-

bing together two pieces of emery paper. Next day there was great extension of the friction sound, which required no pressure for its production, over the whole region of the pericardium; and four days later, the seventeenth after admission, the friction sound was soft, double, and murmur-like, chiefly heard on pressure, and was accompanied by the natural heart sounds, with which it was not rhythmical. I could not make out which sound had the start of the other. For a few days a systolic friction murmur was audible on passing beyond the nipple line, and a double rustle was heard on pressure down to the twenty-eighth day. The extensive doubling of the first sound held its ground throughout, and on the forty-fourth and fifty-third days a little frottement was again present, produced by pressure. On the sixty-fifth day he felt lighter over the heart, and a tremor or thrill was perceived, extending over the cardiac region from the right to the left nipple. A loud double new-leather creak extended over the whole of this region, but the rubbing noise spread far and wide, being heard from axilla to axilla, and down the ensiform and seventh and eighth cartilages. The thrill and creak retained their intensity and area for five days, but on the 6th day the thrill was feeble, and the creak was replaced by a to-and-fro sound extending from the third to the sixth cartilage. Doubling of the first sound was mixed up with the friction sound, but pressure intensified the latter and extinguished the former. On the seventy-third day there was no thrill, and a systolic friction sound, double on pressure, was present between the fourth and sixth cartilages. Two days later the rubbing sound was no longer audible without pressure, and was quite lost on the seventy-ninth day. In this remarkable case the friction sound was present over a limited region near the apex, from the fourth to the twenty-eighth day; came into play slightly on the forty-fourth and fifty-third days; and on the sixty-fifth day burst out, with a thrill, with great intensity over the region of the impulse, and radiated thence as from a focus, all over the front of the chest, and down to the eighth costal cartilages, being audible with a lessening area and diminishing intensity to the seventy-fifth day. This long and intermittent duration of pericardial friction sound appears to me to be peculiar to the pericarditis of Bright's disease, and is certainly never found in rheumatic pericarditis.

These ten cases—which I have given with some detail, as, with the exception of Dr. Taylor's cases and two related, in this respect briefly, by Traube, I have found no cases of pericarditis from Bright's disease in which the signs are related—presented features that are common in them, but are comparatively rare in rheumatic pericarditis. A thrill was present, as we have just seen, in four of these cases or almost one-half (4 in 10); and a sound like the creaking of new leather was heard in six of those cases, or more than one-half (6 in 10), over the seat of the thrill or impulse; and that radiated thence as a softening sound over the front of the chest, beyond the region of the pericardium, and downwards over the ensiform cartilage and the seventh and eighth costal cartilages. These signs were much less frequent in rheumatic pericarditis, since a thrill was present in only one-fifth of those cases, or 13 in 63, and was distributed over the region of the impulse in only seven, was limited to the second space in two, to the apex in three patients, and to both those regions in one; and a creaking friction sound was present at or near the time of the acme of the pericardial effusion in about one-fourth of those cases, or about 18 in 63. The long duration of the friction sound, and its frequent suspension, observed in several of those cases of pericarditis from Bright's disease, likewise distinguish them from those with rheumatic pericarditis.

Calculus in Kidney, Pelvis, or Ureter; or Dilated Pelvis:—and Suppurative Nephritis from Stricture, &c.—I have added, in the Table of Pericarditis in Bright's disease, two sections of cases that, without ranking under that affection, float upon its borders; and substantially belong to the same disease in this respect, that the blood is poisoned, owing to the retention within it of the debris of the broken-up tissues of the body, owing to the imperfect action of the diseased kidney. In the first series, the secreting structure of the kidney is often atrophied by the backward compression of the organ, owing to the distension of the pelvis from the presence of calculus in the ureter, pelvis, or kidney. None of these cases, amounting to twelve, had pericarditis. In the second series of cases, numbering thirteen, there was suppurative disease of the pelvis or kidney, owing mainly to stricture, or disease of the prostate, or bladder (in 11 cases); in one case, to calculus in the ureter, and in another to pyæmia. One of these cases had pericarditis.

I refer to the table for the general condition of these two sets of cases.

Pericarditis, neither Rheumatic nor from Bright's Disease.

Rheumatic pericarditis, so common in the wards, is rare in the post-mortem room; and pericarditis, as we have seen, occurs in as many as eight or nine per cent. of all fatal cases of Bright's disease.

Although uncomplicated pericarditis is a very rare affection, yet its association with other diseases when fatal, and generally as an effect of those diseases, is by no means rare. There is no single malady that is associated with pericarditis nearly so often as the two just mentioned; yet if we combine all the other fatal cases with that affection, except those with Bright's disease and acute rheumatism, we shall find that pericarditis is found on examination after death nearly twice as often in those combined affections as in Bright's disease, and three or four times as often as in fatal cases of acute rheumatism.

The records of the examinations made after death at St. Mary's Hospital during the nineteen years ending 1869–70 contain forty cases of pericarditis that were neither rheumatic nor from Bright's disease. The accompanying summary shows that thirty-nine of these cases of pericarditis were associated with some other disease, general or local, and that in only one case was the affection uncomplicated.

Besides these forty cases of pericarditis, there were sixteen with partial or slight pericarditis.

In addition to these cases I have analyzed in one view (1) Dr. Chambers' complete and valuable table of the causes of pericarditis in 136 cases observed after death in St. George's Hospital during ten years; (2) thirty-seven cases with pericarditis published in the *Pathological Transactions;* and (3) seventy-nine cases collected from various sources.[1]

A. Three cases of pericarditis and three of slight pericarditis had pyæmia, one had scarlet fever, and in one the affection was associated with tubercular disease of the suprarenal capsule; B. twelve cases of pericarditis were associated with affections of the heart or aorta; C. fifteen with affections of the lungs or pleura; D. one with ulcer, and one with cancer of the œsophagus; E. five with affections of the abdomen; F. and besides these cases of secondary or associated pericarditis, there was, as I have just said, one in which the affection appeared to be primary, or uncomplicated.

A. *General Diseases.*—One of the three cases of pyæmia was a school-boy whose leg was doubled up under him five days before his admission. He came in with hurried breathing, blue lips, and tenderness over the chest and abdomen; on placing the hand over the heart a sense of friction was felt, and a loud pericardial friction sound was heard all over the cardiac region. He had delirium, and died during the night. The surfaces of the heart and sac were covered with recent lymph in ridges, and connected by threads; and the muscular substance of the heart was firm, and contained numerous minute purulent dots scattered through the fibres of the left ventricle. Dr. Trotter observed this patient.

This case is typical of a frequent method in which pyæmia induces pericarditis. In such cases the inflammation does not at once attack the surface of the heart, but spreads to it from the points of suppurative inflammation minutely scattered through the muscular walls of the organ, just as pleurisy is caused by the masses of suppurative inflammation spread through the lungs. Dr. Moxon[1] has seen several cases of pyæmic abscesses of the heart, mostly in youths with suppurative periostitis, or acute necrosis of the long bones, in which pericarditis was often caused by the bursting of small abscesses into the pericardium. This is not however the invariable mode in which pericarditis is caused by pyæmic abscesses of the heart, since in my case, just given, and in Mr. Stanley's,[2] there was evidently no rupture of the minute collections of pus in the walls of the heart. Dr. Moxon finds that in cases with pyæmic inflammation of the lung near its surface the pleura becomes involved, and thus every diseased portion of tissue is covered with a layer of lymph; and that when general pleurisy takes place, the abscess has generally burst into the pleura, and so caused the serous inflammation. This well represents the parallel conditions in cases of pericarditis caused by pyæmic abscesses in the heart.

Another case may be named, a man, who had rigors on the day after being operated upon for perineal fistula, and was seized on the following day with violent pain in the region of the heart, the sounds of which were natural. Next day there was a distinct pericardial friction sound, which was feeble in the evening, and was not again distinctly audible. He died on the twelfth day after the operation, and the pericardium was found to be adherent to the heart by a thick layer of recent lymph. In this case, unlike that related above, the pyæmic inflammation evidently struck directly at the pericardium, since violent pain seized the heart the day after the operation, and next day there was a pericardial friction sound. These two cases show the rapidity with which the processes of inflammation pass through their stages in pyæmia.

Pyæmia, including with it erysipelas,

[1] Corvisart (6); Bertin (5); Andral (9); Bouillaud (16); Dr. Stokes (13, including 4 from Testa); Dr. Law (2); Sir Thomas Watson (3); Tringel (13); Dr. Graves (5); Dr. Mayne (3); Dr. Green (1); Dr. Beattie (2); and Dr. Thwaites (1); Total, 79 cases.

[1] Lectures on Pathological Anatomy, by Dr. Wilks and Dr. Moxon, p. 122.

[2] Medico-Chirurgical Transactions, vii. 323.

PERICARDITIS IN ITS ASSOCIATION WITH OTHER DISEASES.

Cases collected from all sources.

CASES.	A. Dr. Chambers' (Decanium Pathologium). Pericarditis.	B. Post mortem Rec., St Mary's Hospital. Pericarditis.		C. Pathological Transactions. Pericarditis.		D. Various Authors. Pericarditis.
		General.	Partial.	General.	Partial.	
Pericarditis associated with acute rheumatism	19	?	...	8	1	13
With Bright's disease	36	25	15	3	1	...
Dropsy	.....		...	...	...	1
A—With general or Constitutional Diseases:—						
Pyæmia, secondary inflammation	18 or 17	3	3	5	1	3
Erysipelas (included with pyæmia St. Mary's Hosp.)	4	...	...	...	...	1
Smallpox	.....	...	...	...	...	1
Fever	4	...	...	...	...	1
Scarlet fever	.....	1	...	...	...	...
Cutaneous eruption	.....	...	...	...	...	1
Tubercular disease, supra-renal capsule	.....	1	...	...	...	...
Tubercle	.....	...	...	1	...	...
Cancer	.....	...	1?	1	...	...
Syphilis	.....	...	...	...	...	1
A—Total	26 or 27	5	4	7	1	8
B—With Affections of the Heart and Aorta:—						
Wound of the heart	.....	1	...	...	...	...
Blow over the heart (1), fracture of the sternum (1)	1	...	...	...	...	1
Tubercular pericarditis	.....	2	...	1	...	1
Cancer of heart, pericardium or neighborhood	3 or 4	1	...	1	...	1
Neighboring abscess (2 in heart)	2	...	...	...	...	2
Fibroid disease of walls of heart	.....	...	...	2	...	...
Aneurism of heart	.....	1	...	...	...	...
Aneurism of ascending aorta	2	...	1	3	...	2
Enlargement of heart, without assigned complications	.....	1	...	...	...	...
"Diseased heart" and "dropsy"	18	...	...	...	...	...
Valvular disease of heart	.....	6	4	3	1	8
Cyanosis, malformation	.....	...	...	1	...	..
B—Total	26	12	5	11	1	15
C—With Affections of the Lungs and Pleura:—						
Pneumonia (generally with pleurisy)	10	8	3	...	...	11
Pleurisy (including empyema)	5	5	2	1	...	12
Phthisis	8	2	1	...	...	3
Communication bet. pericardium and abscess of lung	1	...	...	...	...	1
Indefinite affection of the chest	.....	...	...	...	...	1
C—Total	23	15	6	1	...	28
D—With wound (1); slough (1); ulcer (1); and cancer (1) of œsophagus	1	2	...	1	...	...
E—With Affections of the Abdomen, including the Diaphragm:—						
Diaphragmatic hernia (1); tumor connected with stomach (1)	.....	2	...	...	...	...
Abscess of liver (3); one communicated with pericardium	.....	2	...	...	...	1
Peritonitis	3	1	1	...	...	...
E—Total	3	5	1	...	...	1
Pericarditis, not associated with other affections	2?	1	...	1	1	13
Grand total	136	40*	16*	32	5	79

* Not including those from Bright's disease.

was a much more frequent cause of pericarditis in Dr. Chambers' cases observed in St. George's Hospital (22 or 23 in 81 or 1 in 3·8 of the cases of pericarditis that had neither acute rheumatism nor Bright's disease) than in those recorded in St. Mary's Hospital (3 in 46 or 1 in 13·6; or including partial pericarditis 6 in 56 or 1 in 9·5).

Fever, in which the serous inflammations are rare, was only associated with pericarditis in six instances among those from every source. This does not include one of smallpox, properly pyæmic, nor one of scarlet fever.

Those constitutional diseases, tubercle, cancer, and syphilis, were very rarely complicated with pericarditis, or in only one each among the whole of the combined cases, not including however tubercular pericarditis or cancer of the heart, in which the action of the disease was strictly local.

One single instance of chorea, which is so closely connected with acute rheumatism, had pericarditis. This occurred among the collected cases.

The case of pericarditis associated with disease of the suprarenal capsules is figured at page 541. This man could not lie down, his chest was universally dull on percussion in front and at the left side, and the sounds and impulse of his heart were absent. Upon these grounds Sir James Alderson, under whose care he was, correctly inferred that he had pericarditis.

B. *Affections of the Heart and Aorta.*—In one case, a man, pericarditis was caused by a wound of the heart. The right ventricle was penetrated by a wound about half an inch long, and the surface of the heart, and that of the pericardial sac were covered with recent lymph, stained red in many places. He survived the injury nearly five days. The left ventricle was penetrated by a wound half an inch long. In another patient who survived nearly two days, fibrinous coagula were found on either side of the wound, but there was no definite note of pericarditis. Pericarditis was caused by an injury inflicted over the region of the heart in two of the collected cases.

Local affections of the pericardium itself, and of the immediately adjoining structures, whether bearing upon it from within, and occupying the walls of the heart or ascending aorta; or from without, and seated in the neighboring tissues, all tend to produce pericarditis. Tubercular pericarditis occurred in two instances; and as tubercular disease of the pericardium is rare, it is evident that this affection has a strong tendency to inflame the surface of the heart.

Among the affections of the structure of the heart that excited pericarditis by bearing outwards upon the pericardial surface of the heart, there were four cases with cancer of the heart; two with fibroid disease of the heart, in which the disease extended to the surface of the organ; and two of abscess of the heart, in one at least of which, described by Dr. Graves, there was no pyæmia, and in which instance the abscess contained two ounces of pus, and did not therefore cause pericarditis by bursting into the sac. These cases are derived from all sources.

Aneurism of the heart was the cause of pericarditis in another patient, a well-formed woman, aged 53. The pericardium was distended with about eight ounces of fluid, and was adherent in front to the right ventricle, and behind to the left ventricle by quite recent attachments. The mitral valve was thickened and incompetent. An aneurism was discovered, on examination, in front of the left ventricle about the size of a small orange. The walls of the left ventricle were thickened, but in the position of the sac there was not a trace left of muscular tissue, and the wall was only formed by the parietal layer.

In all these cases, whether of cancer, fibroid disease, abscess, or aneurism of the heart with pericarditis, the inflammation of the surface of the heart is excited in the same manner. The new mass, projecting into the pericardium, and bearing upon it during the active contraction of the organ with a rude and unaccustomed force, excites inflammation in the opposite surfaces of the heart and the pericardial sac, and so establishes pericarditis.

Aneurism of the ascending aorta excited pericarditis in eight of the cases derived from all sources; and three of the twenty-six cases of that affection observed in St. Mary's Hospital, presented evidence of previous pericarditis in the form of pericardial adhesion. In these cases the pericarditis is excited by the constantly enlarging aneurism bearing upon the pericardium, in the same manner that it is excited by cancer, abscess, fibroid disease, and aneurism of the heart.

Cases with valvular disease of the heart, including all its varieties, without Bright's disease, were attacked with pericarditis in definite, but by no means frequent numbers, since that affection appeared in only 6 of the 117 fatal cases in which the valves of the heart were incompetent (1 in 20). These proportions are increased if we strike out the thirty cases of the class under examination in which there were complete adhesions of the heart, and in which pericarditis was therefore forbidden. Thus corrected, the attacks of pericarditis number 6 in 87 (or 1 in 14·5). It will be interesting to ascertain whether valvular incompetence with Bright's dis-

ease was more frequently visited with pericarditis, than when it existed free from that affection. In 78 cases of Bright's disease with imperfection of the valves, 5 had pericarditis (1 in 15·6), or, deducting nine in which the heart was completely adherent, the numbers stand 5 in 69 or 1 in 14. From these comparative results it would seem that Bright's disease scarcely increases the tendency to pericarditis in valvular disease of the heart, for the proportion is almost identical in the two sets of cases. Partial pericarditis was present in 4 of the 117 cases with valvular insufficiency that were free from Bright's disease; and in three of the 78 cases of that class in which the kidneys were affected.

The six cases of pericarditis have been just distributed over the whole series of cases with valvular disease, the varieties of the affection being merged under one common title. If, however, we distinguish the different affections of the valves from each other, we find a remarkable difference in the proportion in which they were respectively attacked with pericarditis. The cases of mitral incompetence included all but one of those attacks of pericarditis, or 5 in 32; or, deducting 12 with complete adhesions of the heart, 5 in 20 or 1 in 4 of those cases were thus affected. The remaining instance of pericarditis appeared in one of the thirty-one cases of mitral-aortic insufficiency, or deducting fourteen with complete pericardial adhesions, 1 in 17 of those cases. Not one of 32 cases with aortic valve-disease, or of 20 cases with mitral obstruction, had pericarditis.

Pericarditis in cases of valvular disease had a strong but not exclusive preference for mitral incompetence among the collected cases, including those in the *Pathological Transactions*, for among eleven cases in which the affection of the valve was specified, eight had mitral insufficiency, while two had mitral-aortic, and one had aortic valve-disease. May not the comparative frequency of pericarditis in mitral valve-disease be due to the resistance to the flow of blood through the lungs, and the consequent distension of the right ventricle with blood; the powerful action of that ventricle, which presses so strongly upon the walls of the chest in front; and the fulness of the coronary veins—which occur in the final stage of that affection?

The cases of pericarditis in Bright's disease, with valvular insufficiency, were equally distributed over the whole series, two with mitral incompetence, one with mitral contraction, one with aortic, and one with mitral aortic valve-disease being thus affected,

Pericarditis attacked one case in which there was hypertrophy of the heart without valvular disease, or any other complication except pericardial adhesion. There were altogether eleven cases of hypertrophy of the heart thus circumstanced, and so in six of them the heart was adherent, rendering pericarditis impossible, that affection attacked one in five cases of this class.

It will be well to inquire as to the proportion in which pericarditis attacked cases with and without hypertrophy of the heart. The heart was enlarged in 130 out of 655 cases of all the kinds that were free from Bright's disease, and among these 130 cases, 12, or 1 in 11, had pericarditis. The heart was diseased in 86 of those cases in which the organ was enlarged, excluding eleven without other complications except adhesion; and including those cases with adherent pericardium, the heart was not diseased in 45 instances. Of the cases just referred to, 26 of the 86, and 9 of the 45, had pericardial adhesions, and could not therefore have pericarditis. After deducting the cases with adhesions, 7 in 60 (or 1 in 8·6). with disease of the heart, and 5 of the 36 (or 1 in 7), without other affection than hypertrophy of that organ, had pericarditis. Without going into detail it may be briefly stated that of the rest of the cases, after deducting those with adherent pericardium, 6 in 104 (or 1 in 17) of those in which the heart was rather large, 4 in 267 (or 1 in 66) of those in which that organ was natural in size, and 1 of the 26, in which it was small, had pericarditis.

These returns make it evident that enlargement, or hypertrophy of the heart exercises a powerful influence on the production of pericarditis. Besides the cases enumerated, there were 107 (or 1 in 6) in which the size of the heart was not described, and of these sixteen (or 1 in 6·7) had pericardial adhesions, and nineteen (or 1 in 4, excluding those with adhesions) had pericarditis. It thus appears that the size of the heart was not described in nearly one-half of the cases with pericarditis, owing evidently to the mind of the reporter being preoccupied by the morbid anatomy of the inflamed organ. One of the cases in which the size of the heart is not noted had mitral incompetence, and may therefore be ranked with those in which the organ was enlarged; and ten of them had pneumonia (in 6), pleurisy (in 3), or empyema (in 1). In these ten cases the labor of the right ventricle must have been increased and prolonged, with the effect of enlarging the right side of the heart. This would tell more on the cases with pleuro-pneumonia than in those with simple pleurisy or empyema, but in such cases, with much effusion into one side of the chest, the obstacle to the stream of blood through the lungs is often

great. This was well evidenced in a case, already alluded to at page 446, of extensive effusion into the right side of the chest which I saw through the kindness of Dr. Wane. Mr. James Lane drew off a large quantity of fluid from the affected side. Before its removal there was a mitral murmur and doubling of the second sound. The doubling disappeared when the fluid was being extracted, and after a time the murmur vanished. In these cases, therefore, the prime effect of the spreading of inflammation from the pleura to the pericardium was heightened by the added secondary influence of the increased size and labor of the right ventricle.

C. Eight patients with pneumonia (8 in 46), three with pleurisy (3 in 26), and two with empyema (2 in 17) had pericarditis. In all these cases (13 in 89), whether the primary affection was pneumonia or pleurisy, it was the pleurisy affecting the outer surface of the pericardium, and spreading thence to its inner surface, that immediately kindled the pericarditis.

Three of the eight cases with pneumonia and pericarditis were under my care, but in none of them did I detect a friction sound.

Two of the three cases with pleurisy and pericarditis were my patients, and in both of them friction sound was heard. One of these was a little girl, who had been attacked a fortnight before with pain in the left side and over the heart, and was brought to the hospital in the mother's arms, in distress, pale, and breathing hurriedly. There was extensive pleurisy of the left side, and next day there was dulness on percussion, and a double, rather smooth friction sound over the whole pericardium. Chorea soon appeared, and on the seventh day, when there was a mitral murmur, the effusion had reached its acme. Two days later, when the friction sound was limited to the lower sternum, she died. The other case was a man who had been ill six months with pleurisy of the left side. On the eleventh day after admission double pericardial friction sound came into play, and continued to the nineteenth day. After two days it vanished from over the heart, and was only audible at the apex; it was thus ten days later, and on the following day he died. The heart was almost universally adherent by yellow lymph.

Although in these thirteen cases the pleurisy excited inflammation of the exterior of the pericardial sac, which travelled through its fibrous structure to its interior, and then attacked the surface of the heart; yet in many of the seventy-six other cases with pleuro-pneumonia or pleurisy the exterior of the pericardium was inflamed, and yet the sac proved to be a barrier to the inflammation, which did not extend inwards so as to excite pericarditis. We have seen that in rheumatic pericarditis the inflammation habitually travels through the fibrous walls of the sac, and attacks its exterior, or pleural surface, exciting pleurisy; so that pericarditis tends to pass from within outwards much more than pleurisy of the pericardium does so from without inwards.

A case of pleurisy with pericarditis, under my care, that recovered presented a peculiar pericardial friction sound on pressure, to the left of the lower sternum, that lasted about three weeks.

I have just alluded to the important secondary influence which the increased size and force of the right ventricle exercises in reinforcing the primary influence of the extension of the inflammation from the pleura to the pericardium in cases of pneumonia and pleurisy.

Pericarditis attacked two cases of phthisis out of a total number affected with that disease amounting to 12. This does not include the two cases of tubercular pericarditis with phthisis already spoken of. Dr. Stokes gives an important case communicated to him by Dr. McDowell in which pneumo-pericarditis was caused by a fistulous communication between the pericardium and a small cavity at the summit of the right lung; the apices of both lungs were healthy, but the bases of both lungs were solidified from a deposit of miliary tubercle and from pneumonia.[1]

D. Two cases were attaeked with pericarditis owing to disease of the œsophagus where it passes behind the pericardium. In one of these patients, who was under the care of Dr. Chambers, the œsophagus was ulcerated from the bifurcation of the trachea to half an inch above the diaphragm. The ulcer gave way into the pericardium, which was filled with fluid from the stomach, and the interior of the sac was lined, and the heart was covered with recent fibrin.

The other patient, with cancer of the œsophagus behind the pericardium, a woman, aged 47, a cook, under my care, complained of slight difficulty in swallowing, referred to the fauces. A to-and-fro friction sound, louder with the diastole than the systole, was audible over the cardiac region, being most intense over the sixth cartilage, and heard from thence to the ninth cartilage. Pleural friction was also present. This patient died on the fifth day after admission.

E. There was a small and remarkable group of cases, in which pericarditis was

[1] Dr. Stokes, on Diseases of the Heart and Aorta, p. 25.

caused by affections involving the diaphragm. One of them had diaphragmatic hernia; two others had abscess of the liver involving the diaphragm; and another had a tumor connected with the pericardium, and communicating with the stomach.

In the case of diaphragmatic hernia which was under the care of Sir James Alderson, the stomach, omentum, spleen, and transverse colon were forced through an opening into the left side of the chest, which contained six pints of liquid, partly digested blood, partly food. The heart was displaced to the right of the sternum, and there was pericarditis.

In one of two other cases an abscess, with thickened walls, containing several ounces of greenish pus, was situated between the pericardium and the liver, involving the diaphragm, and communicating with a small abscess in the liver. The pericardium contained many ounces of puriform fluid, and its lining membrane and the surface of the heart were "hyperæmic," the latter being very red and velvety. In the other case, the diaphragm was pushed up by the liver in a conical projection, which was formed by an abscess occupying the interior portion of the left lobe of the liver, and the contiguous part of its right lobe. The pericardium contained two or three ounces of turbid fluid, and the surface of the heart was roughened by a recent deposit of lymph. Dr. Graves gives an important case in which pneumo-pericarditis was caused by a hepatic abscess which communicated with the pericardium and the stomach.

In the fourth case the pericardium was full of thick yellow fluid, and there were some nodules on the aorta; a dense white tumor which was interposed between the pericardium and the diaphragm was softened in the middle, and formed a cavity which communicated with the stomach and spleen, and resembled an ulcer.

One case of peritonitis out of a total of 64 had general, and another had partial pericarditis.

F. There remains one fatal case of pericarditis in which there was no evidence that the affection was secondary to, or associated with, any other disease.

In this patient, a woman, aged 44, the pericardium was nearly the eighth of an inch thick, and its sac contained a large quantity of sero-purulent fluid. The surfaces of the heart and the sac were covered with recent layers of plastic deposit, which was arranged at the base in a honeycomb shape, and was lengthened out at the apex into bands. The heart was small, hard, and contracted; the lungs were congested behind; and there was a quarter of a pint of brown fluid in each lateral cavity of the chest.

Two cases of pericarditis, under my care in St. Mary's Hospital, presented no other definite affection. One of these, a schoolboy, aged 12, was attacked, eighteen days before his admission, with pain in both sides of the chest, worse in the left. On admission the impulse of the heart was in the fifth space, there was fulness over the pericardium, dulness from the second cartilage to the sixth, and a loud to-and-fro sound, which was intensified by pressure, over the same region and up to the top of the sternum. Next day the dulness had lessened, but the friction sound was strong and grating, and extended beyond the region of dulness. For several days it was more feeble and limited; on the fourteenth, and two days later, it was again louder, but on the nineteenth day it had vanished. The other patient, a pregnant woman, took cold six weeks before admission. The heart's action was tumultuous, and on the third day the impulse extended from the sternum to two inches and a half beyond the left nipple, a to-and-fro sound appeared over and below the region of the heart, and a mitral murmur at the apex. Next day an impulse of a grating character, almost a thrill, extended over the region of the friction sound. These signs continued with variations, but lessening, and on the fourteenth day the impulse had shrunk inwards for two inches and a half, being bounded by the nipple line. Three days later a systolic murmur was converted by pressure into a friction sound, which disappeared on the eighteenth day.

The Treatment of Pericarditis.

Pericarditis, as we have just seen, is so rarely met with except as a combination of, or associated with, some other disease, that in the treatment of such cases we have to consider mainly the primary affection, and along with this the local management of the secondary inflammation of the pericardium. I shall of course here practically limit myself to this latter and local point. It will be important, however, to touch upon the measures, in the treatment of the main disease, that may tend to prevent the occurrence of pericarditis. I shall briefly consider (1) the preventive treatment of acute rheumatism, in relation to the possible occurrence of pericarditis, and (2) the local treatment that the presence of pericarditis may render desirable in those diseases which are more or less frequently complicated with that affection.

(1) The chief objects to be kept in view in the treatment of *acute rheumatism* are (1) the mitigation of the endocarditis that is the usual and natural effect of that dis-

ease, and (2) the prevention of pericarditis, which, though the frequent, is not the customary complication of that disease. Fortunately the measures that tend to palliate the inflammation of the interior of the heart, tend also to prevent the inflammation of the exterior of that organ. The absolute rest of every limb and joint; and the soothing application of the belladonna and chloroform liniment, sprinkled on cotton wool, to the affected joints, supported by flannel, applied over the seat of pain with uniform and comfortable pressure, are the most important measures in the treatment of acute rheumatism for the prevention of pericarditis. The rest and support of the affected joints should be strictly maintained for several days after the disappearance of the local inflammation; for the too early use of an affected joint or limb, after the relief of pain and swelling, often leads to a relapse, first attacking the joints of the over-used limb, extending to other joints, and often producing endocarditis and pericarditis. I have given, at pages 492, 493, brief notes of six cases, in which a relapse of the joint affection, usually thus occasioned, induced endocarditis and pericarditis.

(2) The employment of a few leeches, and the application of cotton-wool or a poultice, sprinkled with the belladonna and chloroform liniment, over the region of the heart during the early and painful period of an attack of pericarditis, are the means that I have for a long time employed in the treatment of that affection.

I have before me the collected notes of 36 cases of pericarditis, in which several leeches were applied over the region of the heart. In 29 of these cases there was pain over the region of the inflamed pericardium, and in 7 of them there was no note of the presence of pain. In 24 of the cases suffering from pain, marked relief, sometimes complete, followed upon the application of the leeches; and this relief in a fair proportion of the cases so speedily followed the local bleeding that the relief must be attributed to the leeching. Brief notes of cases in which the application of leeches relieved the pain over the region of the inflamed pericardium will be found in the preceding pages (493, 498, 503, 507). The local bleeding, besides assuaging the local pain, lessened the oppression in the chest and the difficulty of respiration in many cases.

In one instance leeches were applied over the seat of pain five times; although on each occasion relief seemed to follow, yet the pain soon again increased.

In five cases leeches gave little or no relief. Although in these cases pain was not materially lessened by the local bleeding, yet in every instance but one, its action on the patient's state seemed to be favorable. In that patient, whose case has been already referred to at pages 497, 504, 506, and 507, there was pain over the heart, the action of which was very tumultuous at the time of admission. Leeches were applied with great relief, but unfortunately the bleeding from one of them could not be stopped, and she lost much blood. After this the action of the heart was irregular and intermittent, and she was evidently weakened by the hemorrhage. She finally died after a long and severe illness, which was closed by an attack of smallpox.

The employment of leeches produced a definite but very variable effect on the friction sound, and tended to lessen the force and extent of the impulse. Sometimes the friction sound was lessened in intensity (in 8), but as often it became more intense (in 8) after the local bleeding. In one patient (p. 565) its effect was to suspend the rubbing sound, which had been previously extensive and rough, for one day; but in the evening pain returned, and with it the frottement over the region of the heart. Another patient on admission had excessive pain across the heart, where there was a double thrill, and a double harsh scraping friction sound; four leeches were applied; and next morning there was scarcely any pain, no friction sound, and no note of thrill. The friction sound returned on pressure that afternoon, and was again present on the following day. In one instance—I speak from memory—I examined a patient with pericarditis immediately after the withdrawal of leeches, and found that the friction sound that had been previously audible was entirely abolished. This disappearance of the friction sound in such a case is evidently not due to any change in the character of the lymph on the surfaces of the heart and sac, although their vascularity may be lessened, but to the diminished force of the action of the organ. In direct confirmation of this, we have already seen that in several cases friction sound was abolished, suspended, or softened, by the weakening of the action of the heart (see pages 506, 565).

The effect of leeching the region of the heart on the amount of effusion in the pericardium in cases of pericarditis was not very marked. The leeches were applied at the time of the acme of the effusion in ten cases, and in all of them but two the amount of effusion had lessened on the following day, and in the remaining two on the third day after the local bleeding, which lessened local pain in eight of these cases. To balance these instances, in eight others the effusion increased after the application of the leeches, and attained its acme in a day or two; at

the same time, however, the pain over the region of the heart was relieved in six of those cases, but was not so in two of them.

Blisters applied over the heart are frequently employed in the treatment of pericarditis. I resorted to them occasionally up to the year 1856. I cannot, however, find any instance in which they appeared to be of service, and they were certainly, in some cases, a source of discomfort. It is evident that a blister over the region of the heart adds a second and outward inflammation to the primary and inward inflammation, and it therefore, unless there is a counterbalancing gain, increases the evil. Blisters were the definite cause of mischief in a case that I shall have occasion to quote when I speak of the removal of the fluid from the distended pericardium. In that instance they were applied seven times in succession over the præcordial region. A blister cannot alter the lymph covering the heart and lining the sac; and cannot directly lessen the amount of fluid in the pericardium, which, as we have again and again seen, tends of itself to diminish rapidly when it has reached its acme. It appears to me that a blister over the distended pericardium would rather increase than lessen the morbid supply of blood to those inflamed parts to which it is so contiguous. Blisters, besides inflicting local injury, taint the blood by increasing its fibrin, and are apt to lead to a secondary and low kind of inflammation in distant parts, and perhaps even to degrade the character of the pericardial inflammation itself, and to prolong its existence.

It may be said that exciting pain at the surface of the chest in these cases lessens the severity of the internal pain. This is true, but this effect may be induced innocuously, by the application of chloroform over the seat of suffering, combined with belladonna liniment, sprinkled on cotton-wool, and covered with oiled silk.

Paracentesis of the Pericardium.—We have seen again and again that when the fluid in the pericardium has reached its acme, it very soon begins to diminish. It is therefore evident that puncture of the pericardium is very seldom called for. In some rare instances, however, the quantity of serum in the sac is so great as to interfere seriously with the action of the heart, breathing, swallowing, and speech; owing to the compression of the auricles and venæ cavæ, the trachea and left bronchus, the œsophagus and the descending aorta; and the inflammation of the recurrent nerve. Generally the fluid of itself lessens so quickly that these threatening symptoms pass by without real danger to life. In some rare instances, however, life is in danger owing to the distension of the pericardium, and then paracentesis of the pericardium may become urgently called for.

Riolan,[1] in 1649, proposed that in dropsy of the pericardium, the sac might be opened by trephining the sternum an inch from the ensiform cartilage. Senac,[2] and Laennec,[3] at long intervals, both gave the same advice, the point selected by Laennec being immediately above the ensiform cartilage. Desault[4] attempted to open the pericardium between the sixth and seventh ribs, and Larrey[5] between the fifth and sixth ribs; but they both evidently failed to enter the pericardium. Romero[6] opened the pericardial sac in three cases of "hydro-pericardium," twice with success, through an incision made in the fifth space, near the junction of the cartilages to the ribs, this wound being made, partly to explore, partly to open the pericardium or the pleura. The first circumstantial account of tapping the pericardium was in a patient of Skoda's, with pericarditis from cancer of the heart, operated upon by Schuh in 1840,[4] who first inserted a trocar by a perpendicular puncture through the third space close to the sternum over the great arteries, and failing to get fluid, penetrated the sac through the fourth space and obtained a certain amount of reddish serum. This patient lived for nearly six months, and died with extensive cancer of the chest.[6] In 1841 Heger performed paracentesis of the pericardium in another patient of Skoda's with pericarditis. He entered the pericardium through the fifth space, two inches from the left border of the sternum. Altogether 1500 grammes (about 48 ounces) of a brownish serum, finely flocculent, escaped, and nineteen days later, the fluid having reaccumulated, he again punctured the pericardium at the same place, and 500 grammes (about 16 ounces) of a reddish troubled fluid escaped in the course of four hours. This patient died 51 days after the second

[1] Encheiridium Anatomicum et pathologicum, p. 213.

[2] Senac, de la Structure du Cœur, ii. 369.

[3] Laennec, Traité de l'Auscultation Mediate.

[4] Trousseau et Laségue, Arch. Gén. de Méd. Nov. 1854.

[5] Dict. des Sc. Médicales, v. xl. p. 370. These cases are given imperfectly.

[6] Trousseau and Laségue publish this case at length in the Archives, but in his Clinique Médicale Trousseau states that Schuh penetrated in his first puncture a mass of cancer, altogether of a thickness of six inches, which had invaded the sternum. It was not, however, until more than a month after the operation that this tumor showed itself. Arch. G. de Méd. 1854, p. 520.

operation. The pericardium was in great part adherent, and there were nine and five pints respectively in the two sides of the chest, and a tubercular cavity of the left lung. These two patients died from the primary diseases, cancer, and tubercle; but both operations were successful.

Behier thought that he punctured the pericardium through the sixth left space in a case related by him in 1854; the patient died twenty-six days afterwards, but there was no pericarditis, and no mark of puncture in the walls of the sac. Jobert,[1] in 1854, after cutting the skin punctured the pericardium with a trocar, in a case of pericarditis, a patient of M. Trousseau's, through the fifth left space, 1·2 inch from the edge of the sternum. The canula was agitated by the beating of the heart—the fluid came at first in drops and then very slowly, and altogether 400 grammes (about 13 ounces) of liquid flowed in the course of an hour and a half. The patient left the hospital eleven weeks after the operation, suffering from phthisis. Trousseau,[2] in 1856, operated on another case, and opened the chest with a bistoury below the nipple through the nearest intercostal space, and penetrated into the pericardium, from which flowed nearly 100 grammes (about three ounces) of a red serosity; and twice as much yellow serum came from the pleura. The patient died five days after the operation. The last of the French operators that I shall name was M. Aran,[3] who in 1855, after cutting through the skin, penetrated the pericardium with a trocar through the fifth space, about an inch from the extreme limit of pericardial dulness, and withdrew about 350 grammes (fully 11 ounces) of reddish transparent fluid, and then injected a solution of iodine. Twelve days later he tapped a second time and withdrew 1350 grammes (about forty ounces) of albuminous liquid. This patient recovered from the operation, but three months later presented signs of phthisis.

I have now to speak of two important cases of pericarditis with symptoms threatening life, in which Dr. Clifford Allbutt resolved with his colleagues on the performance of paracentesis of the pericardium. One of these cases was operated upon by Mr. Wheelhouse, who vividly describes the condition of the patient and the steps of the operation. He found the patient sitting up in bed, his head resting on his hands, his elbows on his knees, struggling for breath. I quote the following from his description, and refer to his paper for the full details of the operation; and the precautions adopted during its performance: "I choose for my purpose a small trocar. This I placed on the upper margin of the fifth rib, half an inch to the left of the sternum; and inclining it upwards and inwards, thrust it steadily forwards through the intercostal space towards what I believed to be the centre of the ventricle. I pushed it onwards until I could distinctly feel the movements of the heart with the instrument; and then, sheathing the point, I advanced the canula well up to the heart, until I could feel and see, and demonstrate to those around, the impulse of the heart as communicated to the instrument. The trocar was then withdrawn, and the fluid allowed to escape. This it did at first in a steady stream, which soon subsided into a saltatory flow coincident with the heart's contractions. The fluid consisted of a pale pink coagulable serum, and upon the whole, about three ounces escaped. During the operation the patient gradually obtained relief; and after the canula was withdrawn, the bed-rest was removed, and he was able to lie down."[1] This patient completely recovered, and was in perfect health the other day when Mr. Wheelhouse, in reply to my inquiries, kindly informed me as to the state of the patient. In the second of Dr. Clifford Allbutt's patients Mr. Teale drew off, as Mr. Wheelhouse had done, through a fine canula five ounces of fluid which gave the patient great relief. The reaccumulation of the fluid called for a second operation, which was performed with considerable relief. Finally, however, this patient, a girl, died of bronchitis.[2]

The operation has been performed within the last three years on three occasions, and I owe the references to these cases to the kindness of Mr. Holmes. M. Villeneuve, in 1873, operated by means of the aspirator, on a child with arching and fluctuation over the præcordial region. He punctured the tumor at its most prominent part, and removed two syringefuls of serum. On withdrawing the canula a jet of liquid spirted out of the wound, which remained open owing to the internal wall of the cavity having been very much thinned by the repeated application of blisters, seven of them having been placed one after another, without any improvement, on the same place. A pericardial fistula, yielding pus, was estab-

[1] Trousseau et Laségue, Arch. G. de Méd. 1854.

[2] Trousseau, Clinical Medicine. New Syd. Soc. iii. 365.

[3] Bulletin de l'Academie Royale de Médecine, xxi. 142.

[1] See British Medical Journal, Oct. 10, 1868, p. 385.

[2] See Dr. Clifford Allbutt's important paper, Lancet, 1869, i. 807.

lished and did not heal up until the sixth month after the operation.[1] In the other case, a man in whom paracentesis of the chest and abdomen had already been performed, Dr. Valtosta, in 1874, opened the pericardium by making an incision over the fifth space, commencing about half an inch from the sternum. The layers of muscles were then carefully divided and an elastic dilatation was felt. A puncture was made in this, the point of a small trocar was introduced, and about ten ounces of fluid was removed with immediate relief. This patient died four weeks after the performance of the operation.[2] M. Chairon contributed a third case in 1875, in which more than 1000 grammes (about 33 ounces) of liquid were removed from the pericardium. The result is not given. With reference to the method of operation, he says the spot to be preferred is the fifth intercostal space, at an intermediate point between the nipple and the sternum, rather nearer to the former, always being guided by the apex of the heart. The aspiratory method should, he considers, be preferred.[3]

[Dr. W. Pepper,[4] of Philadelphia, performed this operation successfully in 1877. The patient was a girl, seventeen years of age, apparently moribund from cardiac embarrassment and dyspnœa. These symptoms were at once relieved, and at the end of a month the patient could walk about. Fifteen months afterwards, she died from a complicated attack of pleurisy with ascites.

Dr. J. B. Roberts[5] states that, to the year 1879, paracentesis pericardii had been performed seven times in America. Altogether, he has found authentic records of forty-nine instances of the operation; of these, twenty-three were followed by recovery, and twenty-six by death.

Dr. Pepper mentions[6] one case in which large pleuritic and pericardial effusions being both present, he removed the fluid from the pleural cavity by aspiration; designing to follow this, if needful, with pericardial paracentesis. The fluid in the pericardial sac, however, was absorbed without farther interference, under medical treatment.—H.]

Proposed Operation for Paracentesis of the Pericardium.—This operation cannot well be called for unless the amount of effusion into the pericardium be so great as to compress the venæ cavæ and the auricles, the œsophagus, trachea, and left bronchus, and the descending aorta, so as to interfere with the action of the heart, swallowing, breathing, and the supply of blood to the abdomen and lower limbs. Under these circumstances the pericardial sac is greatly distended downwards towards the abdomen, and the heart itself is elevated. The result is that the mass of the fluid occupies a large space below the heart, measuring between one and two inches from above downwards, between the lower surface of the ventricles and the floor of the pericardium, where it is formed by the central tendon of the diaphragm; which is depressed downwards almost or quite to the level of the upper border of the sixth space, in the manner represented in the figures at pages 544, 551, 553, and 576, and also, in principle, in Pirogoff's important work.

When it is considered that in these serious cases the lower border of the heart is above, while the mass of the fluid is below the level of the lower edge of the fifth cartilage, I advise that the fine trocar, such as that used by M. Aran, Mr. Wheelhouse, Mr. Teale, and M. Chairon, should be inserted into the distended pericardium at a point just above the upper edge of the sixth cartilage at the lowest part of its curve, more than an inch within the mammary line; and that the instrument should penetrate gently inwards with a direction slightly downwards, so that it may advance into the collection of fluid below the level of the heart; and that the liquid should be slowly and gently extracted by the use of a syringe or the aspirator. By this proceeding the collected fluid will be alone penetrated and the heart will be quite untouched. Extensive incisions, and the injection of irritating fluids should be of course avoided.

In every case in which the heart has been previously healthy, and is of the natural size, its lower border is elevated above the level of the fifth space when the effusion into the pericardium is at its height, so that in such cases the procedure I have advised, which has the sanction of Aran's and Chairon's operations, can be performed with ease and safety.

When, however, the heart is enlarged owing to the existence of valvular disease of some standing, the heart is sometimes, as in the cases spoken of at page 549, to be felt beating in the fifth or even the sixth space at the time of the acme of the effusion, when the urgent distress and danger of the patient may demand paracentesis of the pericardium. Under such circumstances, which can be readily discovered by ascertaining the position of the impulse — which should always be some distance above the point of penetration, for a thin layer of fluid interposes itself between the surface of the heart

[1] London Medical Record, iii. p. 532.
[2] Ibid., iii. p. 275, 532.
[3] Ibid, p. 694.
[4] Am. Journal of Med. Sciences, April, 1879, p. 430.]
[5] Phila. Med. Times, Aug. 16, 1879, p. 546.]
[6] Ibid., p. 560.]

above its lower border, and the front of the chest—another point than that just indicated in the fifth space must be chosen for the operation. This point should then be selected at the space between the left edge of the ensiform cartilage and the right border of the seventh cartilage in the epigastric region; or, if needful, owing to its margin being covered by the seventh costal cartilage, the ensiform cartilage, at its left border, may itself be perforated, first with the point of a bistoury, and then with the fine trocar. Trousseau states that Larrey advised that the puncture of the pericardium should be made through this space; but in the operation which he performed with a view—erroneous in this instance—to enter the pericardial sac, that great surgeon, as we have seen, entered the cavity of the chest between the fifth and sixth ribs. The lower border of the fully-distended pericardium is usually a little above, and sometimes even below, the lower end of the ensiform cartilage, as in Fig. 88, page 558; which is from a case, exactly in point, with mitral regurgitation and enlargement of the heart; and the pericardium may therefore be safely punctured through a point corresponding to the middle or the lower portion of that cartilage. The presence or absence of the impulse of the right ventricle in the epigastric space, and the position of the lower border of the pericardial dulness in that space, must be previously ascertained. Those two important points of diagnosis, which can be readily made, will prove a safe guide to the surgeon as to the place which he should select for the operation, which he will rightly fix sufficiently below the seat of the impulse, so as to avoid the heart; and sufficiently above the lower border of the pericardial dulness, so as to prevent the canula being tilted upwards when the floor of the pericardium elevates itself as the sac is being emptied. When he pushes the trocar onwards he must use all the precautions so clearly described by Mr. Wheelhouse, so that if the point of the instrument comes upon the front of the heart, he may withdraw the trocar at the same time that he gently presses the canula forwards and downwards.

In the great majority of cases the fluid, after it has reached its acme, soon begins to lessen, and continues to do so steadily from day to day. Under these circumstances I do not advise the use either of aperients, which tend to disturb and lower the patient, or of diuretics. If, however, the quantity of the fluid is stationary, or lessens very slowly, then diuretics may sometimes be of use.

ADHERENT PERICARDIUM.

By Francis Sibson, M.D., F.R.S.

The discovery of adherent pericardium during life is in some cases impossible, and in some, doubtful or difficult; but in others, and these are amongst the most important cases, its existence may be ascertained during life on reasonable and well-ascertained grounds.

When the adhesions are partial, or when the heart, though completely adherent, is small, is not bound by external adhesions to the anterior walls of the chest, and is covered to the natural extent by the lungs, their expansion being free and unconstrained, then the varying relation of the heart and lungs to the chest is quite natural, and the diagnosis of the adhesions is impossible. If the adherent heart be enlarged, and is not attached to the lower half of the sternum and the cardiac cartilages by combined pericardial and pleural adhesions, so that the active or automatic and the passive or respiratory movements of the heart are scarcely or but little interfered with, the inspiratory expansion of the lungs is freely permitted, and the diagnosis of the adherent pericardium may be difficult, obscure, or even impossible.

When, however, the heart is, as usual, enlarged, being often affected with valvular disease, the adhesions may be short, fibrous, and binding; and the front of the organ may be fixed to the two lower thirds of the sternum and the adjoining cartilages by pleuro-pericardial adhesions, so that the automatic and respiratory movements of the heart, and the inspiratory expansion of the lungs are restrained: thus the discovery of the adhesions during life may generally in such cases be made by a careful study of the physical signs; its diagnosis being the more cer-

tain and easy in proportion as the heart is more enlarged, and more firmly fixed to the anterior walls of the chest.

Anatomical Description of Adherent Pericardium.

Partial Adhesions.—Pericardial adhesions vary greatly in firmness of tissue and length of fibre, and when they are partial they are usually longer than when they are general.

Four conditions seem to regulate the position, extent, and firmness of partial adhesions of the heart. (1) The amount of movement of the various parts of the heart and arteries; for it is evident that the more limited the movement of any part, the greater must be its tendency to adhesion: the relation of the surrounding sac (2) to the heart; and (3) to the outer borders of the pericardium, which are close to the heart, and are therefore more often adherent; (4) the gravitation of the heart in the fluid, since the posterior or depending parts of the heart, when the patient lies on the back, attach themselves readily to the parts on which they rest.

Partial adhesions take place most frequently near the apex and along the line of the ventricular septum; at the outer border of the left ventricle and the outer side of the right auricle, where the movements of those cavities are most limited, and to which parts the outer borders of the sac cling; the posterior surfaces of the left auricle and of the ventricles which rest upon the sac; and the great arteries at their higher parts, where the extent of their movement is least, and where they are most contiguous to the pericardium. The visible commencement of the ascending aorta is often free from adhesions, owing to the hollow, containing liquid, formed in front of that part of the vessel, between the appendix of the right auricle and the origin of the pulmonary artery. In several instances a patch of the right ventricle, to the right of the septum, and midway between the pulmonary artery and the lower border of the ventricle, was adherent when the rest of the ventricle was free; and it is to be remarked that this patch is the part of least movement, or stable equilibrium, of the walls of the right ventricle (see fig. 62, page 401). A frequent seat of partial adhesions is a point a little above and to the left of the apex of the heart. These adhesions near the apex frequently become stretched and attenuated, and at length give way. Several pendulous, filamentous, fibrous bands often hang from this point, near the apex, on the surface of hearts that are free from internal disease; but which display white fibrous patches on their surface; the filaments and the patches being evidently alike the result of a previous attack of pericarditis.

The parts of the surface of the heart and arteries that are usually not adherent when other parts are so, are the front of the right ventricle, especially in the neighborhood of the right auricle and pulmonary artery, and above its own lower border; the appendix and ventricular border of the right auricle; and the parts of the aorta and pulmonary artery nearest to the heart, those being the parts that have respectively the greatest extent of movement during the action of the heart, as may be seen in the figures at page 401.

General Adhesions.—The adhesions are formed of fibrous threads of variable and often of considerable length, and they usually allow of a fair amount of movement of the heart. Long and loose adhesions interfere but little with the free play of the heart; but short, close, and firm attachments embarrass the action of the organ. The length of the fibres of adhesion varies over the different parts of the heart; their length usually corresponding to the amount of movement, and the power exercised by the respective parts during the action of the organ. The adhesions are generally longer at the apex than elsewhere: those over the left ventricle are longer than those over the right ventricle; those over the auricular portion of the right ventricle are longer than those over its body and near the septum, and I believe that the same applies to the left ventricle also. The adhesions over the right auricle are much shorter than those over the right ventricle; and the auricular appendix is contracted in size by the fibrous covering. The attachments of the left auricle, the aorta, and the pulmonary artery are generally closer than those of the right auricle.

When the adhesions are long and loose, and the heart is free from valvular disease, and from any other influence tending to cause enlargement of the organ, the size of the heart is usually natural. It was thus in two of the cases examined after death at St. Mary's Hospital, in four cases that I observed at Nottingham, in many of those referred to by Dr. Stokes, in ten briefly described by Dr. Gairdner, and in 34 out of 90 cases collected by Dr. Kennedy.

When pericardial adhesions are associated with valvular disease, the heart is always enlarged. It was so in 25 out of 26 cases, and in the remaining instance, a case with mitral contraction, the heart was rather large. I have compared a double series of cases of valvular disease side by side, in one series with, and the other without adherent pericardium, and, not going here into details, I may say that the cases with adhesions were on an

average five and a half ounces heavier than those in which there were no adhesions, an increase that was to a considerable extent accounted for, in many instances, by the augmented thickness and weight of the pericardial sac. The increased size of the heart would seem, therefore, in such cases, judging by this analysis, to be traceable more to the affection of the valves, than to the adherent pericardium. We find, however, that in two-thirds of the cases without valvular disease in which the pericardium was adherent, the heart was enlarged (12 in 19); and in one-fifth of them it was rather large (5 in 19); while in only one-tenth of them the organ was of natural size (2 in 19). These proportions are borne out by Dr. Kennedy's important analysis of collected cases of adherent pericardium, who found that in fifty instances the heart was enlarged, in thirty-four it was of natural size, while in five it was atrophied. We may therefore conclude that in cases with the double affection of valvular disease and adherent pericardium, the valvular disease is the essential cause of the enlargement of the heart; yet that the adhesions, by giving an additional spur to the action of the organ, add to the more important enlarging effect of the valvular disease of the organ.

It is the natural effect of pericarditis for the inflammation to spread from the pericardial to the pleural surface of the fibrous sac. When, therefore, the pericardium becomes adherent to the heart in those cases, it becomes adherent also to the walls of the chest in front of the pericardium. These pleural adhesions often occupy an extensive space in front of the chest, and may extend from the second left cartilage to the sixth; from the manubrium to the upper half of the ensiform cartilage; and from the right border of the sternum to the apex of the heart, to the left of the nipple line, as in the cases referred to in former pages, and there described. Though these are extreme instances, yet they are typical of many cases with pleuro-pericardial adhesions.

When the adhesions are short and powerful, and when, being pleuro-pericardial, they bind the walls of the heart extensively to the walls of the chest in front of them, a great and constant strain is put upon the ventricles; for they cannot contract upon themselves to expel their contents until they have dragged the sternum and cartilages powerfully inwards. The ventricles thus expend their force in two directions, one towards the interior to expel their contents, resisted in doing so by valvular incompetence; the other from the exterior, to compel the front of the chest, which is united to them like a solid buckler, to share in their contraction. Under these influences the ventricles tend to undergo a change in form, and to become flattened out, the one in front of the other. Two cases observed by me in Nottingham were thus influenced. The enlarged and thickened right ventricle, instead of sweeping half round the left ventricle, usually cone-shaped, lay directly in front of it; and the septum between the ventricles, instead of bulging forwards into the right cavity, became flattened.

When the adhesions, being extensive and pleuro-pericardial, are not short and close, but of moderate length, and do not, therefore, bind the sternum and cartilages to the heart like a buckler, they do not seriously embarrass the commencing action of the ventricles; but during their contraction, the ventricles at length begin to draw upon the walls of the chest; and in the course of the systole they drag those walls inwards.

When the adhesions are, as usual, longer and less solid, the ventricles contract more after their wont, and retain more or less perfectly their power. The right ventricle is usually enlarged as well as the left, but not always, for the size of the ventricles is necessarily influenced by the valvular affection. When that affection is mitral or mitral aortic, the right ventricle shares the labor and the enlargement with the left ventricle; when the aortic valve is alone affected, the left ventricle is often alone enlarged; and when there is mitral obstruction, the enlargement may mainly affect the two auricles, that of the ventricles being somewhat moderate.

The ventricles, when the pericardium is adherent, tend to enlarge outwards in every direction, and especially upwards to the manubrium, as well as downwards, into the epigastric space, to the right, and to the left. The great arteries are lifted up on the top of the ventricles into an unusually high position, and are crowded into the narrowed space at the top of the chest, almost as high as the root of the neck.

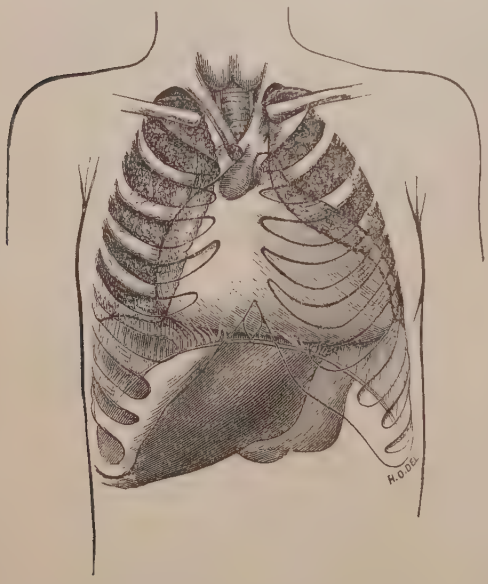
When the adhesions are dense, strong, and contracted, they sheathe the whole heart in a tight, tough envelope, which grasps the auricles and ventricle, prevents their free expansion, and forcibly lessens the organ.

Physical Signs of Adherent Pericardium.

Clinical History. (A) *From a succession of Observers.*—Dr. Burns, in 1809, gave cases to show that when the pericardium is adherent, pulsation is felt in the epigastrium—a sign that had been previously observed by Korner—caused, he says, by the repercussions of the heart affecting the liver, which is the immediate seat of

the pulsation.[1] He gives a case of adherent pericardium in which Dr. Rutherford found a strong pulsation of the heart, accompanied by a jarring motion, most remarkable at the contraction of the ventricles. Heim, according to Kreysig,[2] observed that a hollow appeared under the ribs during each systole when the pericardium was adherent. Sander[3] found, in a case of adherent pericardium with great enlargement of the heart, deepening of the space on the left side of the ensiform cartilage, followed quickly by a shock, perceptible to the hand; fulness over the cardiac cartilages; and extensive impulse over the front of the chest.

Fig. 95.



Corvisart[4] noticed that in these cases respiration is high, and this he connects with the trouble of the whole heart caused by the laborious action of the diaphragm, to which it is attached by the adhesions.

Dr. Hope,[5] in 1839, observed that pericardial adhesions sometimes caused a prominence of the cardiac cartilages, sometimes an abrupt jogging motion of the heart, corresponding with the systole and the diastole, that with the diastole having the character of a receding motion suddenly arrested. In the recital of four of his cases, to which his general account does little justice, he states that they presented a second or diastolic shock or backstroke.

Dr. Williams,[1] in 1840, remarked that when the pericardium adheres both to the heart when enlarged, and to the walls of the chest, the heart pulsates in close contact with those walls; so that the pulsations are felt very widely, extending upwards as well as downwards, drawing in the intercostal spaces at each systole; and that respiration does not lessen the region of cardiac dulness on percussion, and of impulse. Dr. Law, in a communication that I have not been able to find, states that change of posture does not alter the position of the impulse.

In my paper on the situation of the internal organs, I, in 1844,[2] described four cases of adherent pericardium, and gave figures showing the position of the internal organs after death, two of which figures I reproduce here (see Figs. 95, 96). In one of these cases, a young woman, the heart was small in size, and presented during life no physical sign of disease of the heart, but the pulse was very feeble; she had palpitation, dyspnœa, and anasarca; and her lips were blue.

The heart was very large in the three remaining cases, two of which had mitral regurgitation, and the third had narrowing of the mitral, aortic, and tricuspid orifices. One of the two cases with mitral disease has been already described, and is figured at page 559. In the other case of the same class, the impulse was very strong and jogging; shaking and heaving the whole chest. The apex protruded strongly; the lower half of the sternum advanced firmly at the beginning of the systole, and fell back gradually and firmly during its continuance. The lower end of the ensiform cartilage receded during the systole; the impulse was irregular, 140 to 160 (*see* figure 95).

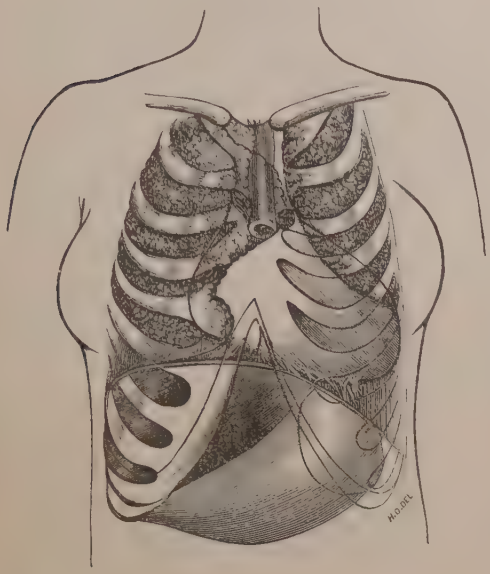
The remaining case with adherent pericardium presented physical signs that differed materially from those observed in the two other cases. The obstructed, mitral, and aortic apertures tested by the cone, each measured half an inch, and the tricuspid orifice three-quarters of an inch. The heart was very large, weighing thirty-two ounces; and all its cavities, and especially the ventricles, shared in the enlargement. The following were the

[1] Burns, on the Diseases of the Heart, p. 62.
[2] Kreysig, Die Krankheiten des Herzens, ii. 625.
[3] Hufelunand Bibliothek d. p. Heilkunde, Bd. 51, 120.
[4] Corvisart, Sur les Maladies du Cœur, p. 35.
[5] Dr. Hope, on the Diseases of the Heart, p. 194.

[1] Dr. Williams, on the Diseases of the Chest, p. 24.
[2] Prov. Med. Trans.

physical signs: "Strong protruding impulse at the apex between the sixth and seventh ribs. During the systole, the sternum and the left and right costal cartilages over the right ventricle became steadily depressed; immediately after the systole they advanced with a shock."[1] (See Fig. 96.)

Fig. 96.



In the general description I thus defined the character of the impulse in the two classes of cases just given: "The sternum, costal cartilages, and xiphoid cartilage are heaved forward firmly and steadily at the beginning of the systole; and during its continuance those parts fall back steadily and quickly, coinciding with the mode of systolic contraction of the right ventricle. In some cases the sternum and costal cartilages spring forward with a jerk during the diastole."

M. Bouillaud,[2] in 1846, described a sign by which he had been able to announce the existence of adherent pericardium in six or seven cases. It consisted in evident retraction of the pericardial region; the movements of the heart not being free, but embarrassed or curbed. He does not state during what period in the revolution of the heart's action the depression of the pericardial region took place.

Skoda,[3] in 1852, published an important paper on the diagnosis of adherent pericardium, in which he gives a critical account of most of the communications just analyzed, and reports of three cases observed by himself. In the first case, a youth, there was dulness on percussion, equal in extent during inspiration and expiration, from the second left space to the ensiform cartilage, and from the middle of the sternum to the left nipple; and fulness over the second space, which advanced during the systole and sank in during the diastole; the third, fourth, and fifth spaces deepened with the systole and filled out with the diastole; the heart's impulse was feeble, and the apex-beat was imperceptible. The heart sounds were natural, but the second sound was split over the pulmonary artery. The pericardium was tied to the walls of the chest by filamentous bands, and was universally adherent to the heart, which was natural in position; the right ventricle was enlarged, the right auricle was changed into a stiff crumbling tuberculous mass, and the conus arteriosus was widened, its walls being only a line in thickness.

The second case, which passed through all its stages under Skoda's eye, a youth, was admitted with pericarditis. The friction sound, then loud and extensive, became feeble and limited to the apex on the 15th, and was lost on the 19th day. On the 37th day there was a systolic deepening of the third, fourth, and fifth spaces, and the apex-beat was imperceptible. A month later, when he left the hospital, during each systole, besides the indrawing of the spaces, there was indrawing of the lower half of the sternum, which sprang forward after the systole with a perceptible shock. He was admitted ten weeks later with pneumonia, when the heart-signs were unchanged, and he died fully six months after his first admission. The right ventricle was enlarged; the valves were healthy; the heart, which lay in the middle of the chest, was firmly adherent to the pericardium, which was, in turn, strongly glued to the walls of the chest by a tuberculous exudation.

Skoda's third case was a man, with narrowing of the mitral orifice, ascites, and œdema. The region of cardiac dulness remained unchanged during inspiration and expiration. There was a considerable deepening of the fifth space during the systole, after which the hollow quickly disappeared, and a shock was perceived there at the beginning of the diastole. After his death, five months later, the pericardium and pleura were found to be universally adherent, and the right side of the heart was considerably enlarged.

These cases, published by Skoda, form a valuable addition to the clinical history of adherent pericardium, for the true

[1] Loc. cit. p. 562.

[2] Traité de Nosographie Médicale, i.

[3] Zeitschrift der Gesellschaft der Aerzte zu Wien, 152, i. 306.

points of diagnosis have here been clearly observed, stated, and confirmed; and are given with force, and as the effects of the central cause, the doubly adherent pericardium. They do not, however, present any new points of diagnosis, for it will have been seen, in the previous narrative, that he has been anticipated by one or more authors in the observation of each diagnostic sign. Thus the systolic deepening of the intercostal spaces had been observed by Heim and Dr. Williams, the return shock over the previously retracted space by Sander, and the great extent of the cardiac space upwards, and the non-diminution of that space, by Dr. Williams and myself; while the retraction during the systole of the lower half of the sternum, and its advance with a shock immediately after the systole, was observed by myself in the case already given.

Great diagnostic value is to be attached to the principal points specially illustrated by Skoda's paper, namely: the systolic indrawing of the lower sternum or intercostal spaces by the contraction of the adherent heart; and the diastolic shock or back-stroke that immediately follows, given by the return elasticity of the chest-walls.

Cejka,[1] in 1855, published four cases of adherent pericardium, three of which confirm, with more or less precision, the points illustrated in Skoda's paper. In one of them, with contraction of the aortic orifice, there was systolic indrawing of the third, fourth, and fifth spaces, and so strong a blow was given by the return elasticity of the chest walls that it was like the impulse of the heart. In another instance, an old man with adherent pericardium, a chronic affection of the lungs, dilatation of the aorta, and thickening of the mitral valve, the fifth and sixth spaces were drawn inwards with each systole, and became quickly even with each diastole. The impulse was not perceptible, and there is no note of diastolic back-stroke. In the third patient, with aortic aneurism, the vaulting of the sixth left space, caused by the systole, gave place towards the end of the case to a slight drawing inwards of the corresponding region. Cejka's fourth case of adherent pericardium, also with aneurism of the aorta, presented no impulse and no apparent drawing inwards during the systole.

Clinical History. (B) *Cases observed in St. Mary's Hospital and at Nottingham.*—1. *Cases examined after Death.*—The pericardium was completely adherent in fifty-one, and partially so in nine of the cases free from Bright's disease, recorded after death in St. Mary's Hospital up to the year 1870. Besides these, seventeen of the cases with Bright's disease had universally, and three of them had partially, adherent pericardium.

Rheumatic pericarditis had evidently been the cause of the adhesions in more than one-half of the cases, since of those with complete adhesions, 29 in 51 that were free from Bright's disease, and 9 in 17 with Bright's disease, had valvular disease of the heart; while the valves were affected in 7 out of 8 of those with partial adhesions that were free from Bright's disease, and the three cases of that class with that affection.

General adhesion of the pericardium was rarely associated with disease of the aortic valve (2 in 32), and with mitral obstruction (1 in 21), in cases free from Bright's disease, while that affection was very frequent in such cases with mitral and mitral-aortic valve disease (13 in 33 of the former and 11 in 31 of the latter affection). Adherent pericardium was present in one case with disease of the tricuspid valve. Partial adhesions of the pericardium were noted in one case with aortic regurgitation, in two with mitral obstruction, in none with mitral, and in two with mitral-aortic regurgitation, without Bright's disease; since the aortic valve was affected in 1 in 4 of the cases, while only two had mitral and two had mitral-aortic disease. Among the cases of complete (17) and partial (3) adhesions with Bright's disease, 4 (in 21) had aortic valve-disease, 5 (in 29) had mitral and 2 (in 20) had mitral-aortic valvular disease, and 1 (in 9) had mitral contraction.

Aneurism of the ascending aorta was the evident cause of adherent pericardium in three instances (3 in 25), and cancer of the heart in one (1 in 10).

There was no other affection of the heart or aorta, excepting enlargement of the organ itself, in more than one-third of the cases with complete adhesions (19 in 52). The adhesions were not accompanied by any other affection in less than one-half of these cases (7 in 19), and they were complicated in more than one-half of them with pyæmia (in 2), apoplexy (in 1), pneumonia (in 3), empyema (in 2), phthisis (in 3), or peritonitis (in 1). All those affections, excepting the last two, were acute; and they could not, therefore, have given rise to the adhesions. Phthisis, and especially empyema, which is so often associated with phthisis, may, owing to the duration of those diseases, have induced first pericarditis and then adhesions. Notwithstanding this, the whole of those cases may be taken into account when considering the effect of pericardial adhesions on the size of the heart, for none of them by themselves cause enlargement of that organ, excepting pneumonia, and, less often, phthisis, both of which

[1] Vierteljahrschrift für die praktische Heilkund, 1855, 128.

affections tend to increase the right ventricle in size.

The heart was enlarged, its valves being thickened but competent in one instance, in fully two-thirds of the cases with adherent pericardium that were free from any other cardiac disease, and in which the size of the heart is mentioned (11 in 16); it was rather large in three of them; and in only two instances was the heart of its natural size. We may however, I think, estimate that in one-third of these cases the adhesions did not cause an increase in the size of the heart. These results do not differ materially from those arrived at by Dr. Kennedy,[1] who found that in 90 cases of adherent pericardium in which valvular disease was not present, the heart was of natural size—"healthy"—in 34, or fully one-third, hypertrophied in 51, or three-fifths—being dilated also in 26—and atrophied in 5.

It is proved that pericardial adhesions do not necessarily cause enlargement of the heart. I saw four cases in Nottingham in which the heart was of natural size and one in which it was lessened; Dr. Gairdner[2] gives brief notes of ten cases in which the heart was not morbid, and by inference was not affected in size; and Dr. Stokes[3] informs us that Professor Smith found that general adhesions of the pericardium correspond with atrophy or with hypertrophy of the heart in nearly equal proportions.

We may, I think, safely conclude from what has gone before that adherent pericardium may, and often does, exist without influencing the size or healthy function of the heart; that in a few rare instances it may induce atrophy of that organ; and that in nearly two-thirds of the cases it tends to cause an increase in the size of the heart, both as regards the thickness of its walls and the capacity of its cavities.

We have just seen that the heart was enlarged in the majority of the cases of adherent pericardium that were free from any other affection of the heart itself. When we take this into account it is natural to expect that the heart should be more enlarged in cases with valvular disease when they are affected with adherent pericardium than when they are not so; and the analysis of the cases of this class that were recorded at St. Mary's Hospital by taking a simple average of the weights of the hearts with valvular disease, with or without pericardial adhesions, gives some support to this anticipation, as will be seen by the examination of the following summary of the average weight of the heart in those cases.

Average weight of the heart in cases of valvular disease with and without adherent pericardium. The cases were not affected with Bright's disease except where specified.

Mitral regurgitation, pericardium adherent (4)	average weight,	21	ounces.
Ditto, pericardium not adherent (14)	"	16·6	"
Ditto, with Bright's disease, pericardium adherent (3)	"	*25*	"
Ditto, pericardium not adherent (19)	"	*19·4*	"
Mitral obstruction, pericardium adherent (1)	"	21	"
Ditto, pericardium not adherent (14)	"	14	"
Aortic regurgitation, pericardium adherent (2)	"	26·7	"
Ditto, pericardium not adherent (23)	"	22	"
Mitral-aortic regurgitation, pericardium adherent (6)	"	26·3	"
Ditto, pericardium not adherent (12)	"	22	"
Total of combined valvular diseases, without Bright's disease, pericardium adherent (13)	"	23·3	"
Total of combined valvular diseases, without Bright's disease, pericardium not adherent (63)	"	19	"

This method is far from doing scientific justice to the question before us; for cases of all ages, both sexes, and various degrees of disease, are brought together under one common heading, although in reality many of these cases differ materially from each other. Notwithstanding this, a rough and ready answer is given to us that is probably not far from the scientific truth. We find, then, that the average weight of the heart in the thirteen cases of valvular disease, with adherent pericardium, was 24⅓ ounces, while its weight in sixty-three cases of a like kind, in which the pericardium was not adherent, was 19 ounces, or 5¼ ounces less than the first series. It is to be kept in view that the pericardium was included with the heart in the first set of cases, and what its average weight may be under the varying circumstances I do not know. It may, however, I think, be concluded that in the cases of valvular disease of the heart the existence of adherent pericardium tended to increase the size and weight of the heart, but not to a great extent.

[1] Edinburgh Medical Journal, iii. 986.
[2] Ibid. Feb. 1851.
[3] Dr. Stokes, Diseases of the Heart.

The size of the heart, as we have seen, has been usually described; its weight being often given, in the cases with adherent pericardium observed in St. Mary's Hospital. The relative size of the different cavities of the heart has, however, only been described in 11 of these cases. I have, therefore, with a view to discover the influence that the presence of adherent pericardium may have on the size of the various cavities of the heart and the thickness of their walls, brought together 18 additional cases from various sources—or 29 in the whole—in which the general condition of the various cavities of the heart was described, and which are given in the following summary:—

Cases with adherent pericardium in which the size of the different cavities of the heart was described:—

1.—Cases in which both ventricles were enlarged (hypertrophy and dilatation) 16

Of these, 6 were free from valvular or other heart disease (1 had Bright's disease); 10 had valvular disease (3 aortic, 2 mitral, 3 mitral-aortic, regurgitation, 2 mitral contraction).

2.—Cases in which the right ventricle was enlarged, the left being not so (in 1), or small (in 1), or not described (in 3) 5

Of these, 3 were from valvular disease, 1 had mitral regurgitation, and 1 aneurism of the aortic sinuses.

3.—Cases in which the left ventricle was enlarged, the right being small (in 1), or not described (in 7) 8

Of these, 3 had no valvular disease, 1 had aortic, and 3 mitral regurgitation, and 1 had aneurism of the apex of the left ventricle.

Total 29

There was valvular disease of the heart (15), or aneurism of the heart (1) or aorta (1) in 17 of these cases, and as those affections exercise a definite influence of their own on the size of the cavities of the heart, they must be left out of view in considering the direct effect of adherent pericardium on those cavities. The same must be said of one instance with Bright's disease among the remaining 12 cases in which there was no valvular or other affection of the heart or aorta. Hypertrophy and dilatation of both ventricles existed in 5; of the right ventricle in 3; and of the left ventricle in the remaining 3, of these 11 cases. From this it would appear that adherent pericardium, when it produces enlargement of the heart, tends to affect both ventricles to an equal but varying degree.

2. *Physical signs observed during life in cases with adherent pericardium admitted into St. Mary's and the Nottingham Hospitals.*—I have observed nine cases with adherent pericardium in St. Mary's Hospital, and have added one recorded there by Dr. Markham; and have examined seven such cases at Nottingham, four of which I published in 1844, and have given briefly above. There was no valvular disease of the heart in three of these seventeen cases, while in the remaining fourteen, one or more of the valves was affected, mitral regurgitation being present in nine of them, mitral-aortic regurgitation in three, and mitral obstruction in two, of those cases.

In one of the three cases in which the valves were healthy, in which case Bright's disease was present, the sounds of the heart were natural but weak, and the presence of impulse was not noted. In another of them, a man, with empyema and lardaceous disease of the kidney, the heart being only slightly enlarged, the impulse was at one time imperceptible, but afterwards, when it could scarcely be felt over the ribs, it was perceived over the ensiform cartilage. In these two cases, and in that of the same class already alluded to at page 608, in which the heart was small, the presence of adherent pericardium could not, I think, have been discovered during life.

The signs of the heart were not noticed in one of the cases in which adherent pericardium was associated with mitral regurgitation, an old man who presented various sonorous noises over the lungs. In one of two cases, both men, with mitral disease, observed at Nottingham, in which the heart was very greatly enlarged, the left ventricle was greatly hypertrophied and dilated, the right being so to a minor degree; and the impulse was feeble, the second sound, distinct over the sternum, was scarcely audible at the apex, and the lungs were œdematous. In the other case, with hypertrophy of both ventricles, the impulse was inconsiderable, but was diffused over the whole left mammary region.

The next case is an important one, reported by that careful and accurate observer, Dr. Markham, for it shows that the apex-beat may be strong, and far to the left, in some unusual cases of adherent pericardium. In this patient, a girl, the impulse was heaving and extensive, and was violent far to the left of the nip-

ple line, and beneath the sixth rib. The second sound was very loud over the pulmonary artery, but was absent at the apex. M. Aran likewise describes a case of adherent pericardium, in which the apex-beat was present in the sixth space, three and a half inches from the sternum, and the systolic impulse was strong and progressive, and was not followed by a diastole impulse. Skoda takes exception to my observation that the apex protruded extensively to the left in two of my cases published in 1844, given briefly above at pp. 608, 609. We shall see that the apex-beat is usually feeble, and does not often extend far to the left in cases of adherent pericardium; but it was certainly otherwise in this case of Dr. Markham, in that of M. Aran, and, I would say, also in my two published cases. It appears to me that in this patient, and in the other cases just given, there was no sign characteristic of adherent pericardium.

The next instance was too ill for careful physical examination, and presented a feature unusual in cases with pericardial adhesions. The healthy impulse was much more diffused than natural, being present in the epigastric space and four or five intercostal spaces, and the lower ribs retracted during the diastole, which is a rare occurrence. The apex-beat, which was felt in the fifth and sixth spaces, did not extend outwards so far as the nipple line. The two following instances present features that were sufficient to characterize them during life as being affected with adherent pericardium. In the first of these cases, the left ventricle was hypertrophied, the right ventricle was small, and both the auricles were very large. The apex-beat was seated in the sixth space, an inch to the left of the nipple line, and 5½ inches from the sternum, and in spite of the great and extensive hypertrophy of the left ventricle, was feeble. The second sound, which was heard over the right ventricle, was faint at the apex. There was, on the 54th day after admission, a diffused impulse chiefly over the cardiac cartilages, extending down to the seventh costal cartilage, and to the ensiform cartilage. The impulse advanced quickly and fell back suddenly during the systole, and was followed with a sharp sudden shock or jerk over the whole region of the impulse. There was slight pulsation of the liver below the ensiform cartilage. Breathing was rather high, the movement being chiefly at the upper part of the chest, with retraction at its lower part. The other case, equally remarkable, and the last of the series with mitral incompetence, had points of close resemblance to the last, with points of marked difference. In this case the front of the heart adhered strongly to the inner surface of the sternum through the medium of the pericardium. The walls of the right ventricle and auricle were much hypertrophied, while the left ventricle was only somewhat thickened; thus reversing the conditions that were present in the former case. There was some fulness over the region of the heart. The impulse over the heart, and especially over the right ventricle, was very extensive, spreading from the third to the seventh cartilage; and from the right cartilages, across the sternum and ensiform cartilage, to the sixth left space, an inch and a half beyond the nipple line. The impulse was peculiar, and told remarkably on the sternum, first heaving that bone forwards with sudden force, and then drawing it backwards with great strength. "The heart" (or rather the front of the chest) "seemed to be dragged backwards during each systole. The apex-beat was feeble, low down, and far to the left, in the sixth space, an inch and a half beyond the nipple line. There was some pulsation of the liver in the epigastric region. The second sound was loud and plunging over the right ventricle, and feeble at the apex, where a mitral murmur was loud and extensive. Afterwards the fulness over the heart, and the extent and force of the impulse lessened, but the beat of the heart retained its remarkable character, first advancing, and then forcibly retracting, during the systole. Later still the apex-beat, which was very weak, extended only a very little beyond the nipple line. Notwithstanding this contraction of the region of the impulse, it extended from right to left over a width of six inches. A deep inspiration caused a marked lowering of the upper and lower borders of the region of the impulse, in spite of its great extent. After a few days he became drowsy, felt tight in the chest, and died three weeks after his admission." It is to be remarked that while in the previous case a diastolic shock or back-stroke followed the systolic retraction, which was preceded by a systolic advance; in this case there is no note of back-stroke, though I cannot vouch for its absence; but the sudden systolic heave followed by a forcible systolic retraction of the sternum and cartilages, as if those parts were dragged backwards by the heart clinging, as it were, to its buckler, pointed definitely to adherent pericardium as the cause of the chain of signs.

The two cases of adherent pericadium with mitral-aortic incompetence present, like the last two cases, physical features that denote the presence of the adhesions, though not perhaps with the same emphasis as the two first related. In the first case, a youth, the heart was of very great size, so as completely to cover the left lung. On his admission, three months

before his death, the impulse was gradual, but ended abruptly with a shock; and extended from the third cartilage to the sixth, but scarcely beyond the nipple line; there was also a marked general pulsation over the whole liver, both in front and at the right side. A month later the impulse had extended itself to the left, being diffused, and shaking the whole of that side of the chest, the apex-beat being an inch and a half to the left of the nipple line. Afterwards the impulse extended more to the right and was felt in the epigastrium, but its characteristic features are not again described. The other instance was a boy, and in him the heart, which was considerably enlarged, clung so close to the sternum and cartilages that it was found best to remove the viscera *en masse* from behind. There was fulness over the cardiac region, and the beat of the heart, which was extensive, reaching down to an inch and a half below the sternum, and extending thence to the seventh cartilage, was of a peculiar character, beginning with a diffused heaving impulse, which gave way to a sudden and sharp retraction. He always said, after this examination, that he felt better, though he really was not so, and eight days later he died.

The two remaining cases with adherent pericardium had mitral contraction. In one of them, a young woman, the heart was very large; the impulse extended from the second space to the seventh costal cartilage and the ensiform cartilage, and, even when she lay on the left side, the apex beat was feeble. As in the last case, there was strong pulsation over the whole liver, extending from the front to the back. The remaining case with adherent pericardium and mitral contraction was observed by me in Nottingham in 1835, and although it presents no signs characteristic of the adhesions, is perhaps of interest, as being, so far as I know, the earliest case in which the so-called presystolic murmur was described. The size of the heart is not given, but there was no hypertrophy of either ventricle. The mitral opening was half an inch in diameter. A thrill, extending over a large space, was communicated to the hand when applied over the apex, which was terminated by a jerk. A peculiar purring sound was heard at the apex, the vibrations being longer and louder as the time progressed, the sound ending in a strong, loud, clear jerk, synchronous with the pulsation. The sound occupied two-fourths of the time, no other being audible at the apex.

Résumé of the Physical Signs observed in Cases of Adherent Pericardium.—The steady retraction of the lower half of the sternum during the whole of the systole of the ventricles, and the sudden starting forwards of the lower half of the sternum at the beginning of the diastole with a return shock or blow, was observed in my own case, published in 1844, and in one of Skoda's given in 1852.

The drawing inwards of the cardiac intercostal spaces during the systole was first observed by Heim, and afterwards by Dr. Williams, by Skoda in three cases, and by Cejka in three more.

This sign, which is sometimes present in other cases renders the existence of adherent pericardium probable, and especially if this sign is still present when the patient draws a deep breath; but if it is followed by a diastolic shock the diagnosis of that affection is certain. The existence indeed of a diastolic back-stroke taken by itself pronounces that the heart is adherent. This sign, which generally gives the impression of a double impulse, was first noticed by Sander; afterwards by Dr. Hope in four cases of adherent pericardium; in the two typical instances just given and described respectively by myself and by Skoda, who observed it in another instance; by Cejka in one, and by myself in two others given above.

A double movement of the systolic impulse, first forwards with a heaving motion, then backwards with a forcible retraction, was observed by myself in a case in the Nottingham Hospital, to the description of which Skoda takes exception, and afterwards in three other cases in St. Mary's Hospital. The outward pressure, equal in every direction, of the blood contained in the ventricle during its contraction naturally forces forwards the walls of the chest in front of it at the beginning of the systole. During the continuance of the systole, the adherent sternum resists the contraction of the heart, but in the struggle the bone yields, and is drawn forcibly inwards by the active ventricle.

The non-diminution of the region of pericardial dulness and of the impulse was observed by Dr. Williams; and the absence of change in the position of these signs when the patient lay on the left side was noticed by Dr. Law.

The non-diminution of the area of pericardial dulness and impulse is undoubtedly a valuable sign of adherent pericardium; in one of my cases, however, the impulse below was unusually strong at the end of expiration, and in another of them the upper and lower borders of the impulse palpably descended during a deep inspiration. This is indeed different from the diminution of the extent of dulness and impulse, and, what is still more important, from the bodily transfer during a deep breath of the seat of the dulness and impulse from the cardiac cartilages and the first space near the nipple, to the epi-

gastric region, including the ensiform cartilage and the adjoining seventh costal cartilage. One of my cases illustrates in its own manner the other point just referred to—the non-shifting of the seat of the impulse when the patient turns on the left side. In that case, when the patient lay on the left side, the apex-beat, which was an inch and a half to the left of the nipple line, and in the sixth space, was very feeble. This is very different from the great transfer of the position of the apex-beat from the fifth space, a little lower than the nipple, and within the mammary line, to the sixth or seventh space, two inches to the left of that line, which was observed to be the case in several patients, in whom the chest was healthy, by Dr. Humphreys, Dr. Coupland, and myself, in the Middlesex Hospital.

These, so far as I know, are the only signs that are characteristic of adherent pericardium; but there are certain other signs that, without ranking in precision with those just named, have their significance.

The drawing inwards during the systole of the space between the ensiform cartilage and the seventh costal cartilage, was noticed by Sander in a case of adherent pericardium; and in another case, I observed that the tip of the ensiform cartilage was retracted during the contraction of the ventricle.

There was pulsation of the liver in four of my cases, which was limited to the epigastric space in two of them, but in the two others extended over the whole organ, in front, at the side, and in one even behind. Burns considered that the impulse so often present in the epigastric space in cases of adherent pericardium is due not immediately to the heart itself, but to the pulsation of the liver.

It is evident, from the brief recital of the cases that has just been given, that a great variation in the extent, force, character, and position of the impulse exists in cases of adherent pericardium.

The impulse was imperceptible in one of Cejka's, and at an early period in one of my own cases of adherent pericardium; and it was feeble in one of Skoda's and two of my own cases; it was heaving during the systole and very extensive in one of Dr. Markham's cases, and in one of my own; it was tumultuous and very irregular in one of my cases; it was strong and very greatly extended, both upwards to the second space, and downwards to the epigastric space and the seventh cartilage, and to the right and left, across the chest, from a full inch to the right of the lower half of the sternum, to a full inch to the left of the nipple line in the sixth space, in cases observed by Dr. Hope, Dr. Markham, and myself; and in two of Dr. Hope's cases the violent action of the heart was observed over the whole front of the chest.

The apex-beat is, as a rule, feeble, even when it extends from an inch to an inch and a half to the left of the nipple line, being felt in the sixth space. Sometimes indeed, as in one of Skoda's cases, it is imperceptible; and at others it is situated, even when there is general enlargement with hypertrophy of the ventricles, to the right of the nipple line, as occurred in one of M. Aran's cases in which the apex-beat was in the fifth space, two and a half inches from the sternum; and in two of the cases given by Dr. Gairdner, who points to this restraint of the apex as a probable element in the diagnosis of adherent pericardium.

There are, however, important exceptions to the rule that the apex-beat is usually restrained in its action and sometimes in its position by adherent pericardium, for in two cases published by me in 1844, the apex-beat was far to the left and low down, strong, gradual, and protruding; and as we have seen, the apex-beat presented the same condition in Dr. Markham's, and to a less degree in M. Aran's important cases.

The impulse was found in the epigastrium in Mr. Burn's cases, in two of Dr. Hope's, and in four of my own.

M. Aran, in 1844, gave the extinction of the second sound as the unique sign of adherent pericardium, on the strength of the absence or great feebleness of that sound in those cases reported by him. He does not distinguish between the second sound over the pulmonary artery and right ventricle, and that over the left ventricle. Dr. Markham describes the second sound as being, in his case with mitral incompetence, very loud, heard like a beat, over the pulmonary artery, while there was no second sound over the apex. In one at least of my cases observed at Nottingham the second sound was loud or natural over the right ventricle, while it was indistinct and dull at the apex, and in two of the cases given above the second sound, loud over the pulmonary artery and right ventricle, was feeble at the apex of the heart.

The last physical sign that I shall consider is the movement of respiration in relation to adherent pericardium. In two cases of adherent pericardium observed by myself in Nottingham, the inspiratory movement of the abdomen at its centre was equal to that at its sides: although in health, the central movements are from two to three times as great as the lateral movements of the abdomen. At the same time in both

those cases the lower half of the sternum fell inwards, or was drawn backwards, and the left ribs, from the fourth to the sixth, either retracted or were stationary, or had much less movement during inspiration, than the corresponding right ribs. The retraction of the sternum was caused by the forcible displacement downwards of the central tendon of the diaphragm, where it forms the floor of the pericardium; and as under these circumstances the lungs could not interpose themselves between the heart and the sternum, that bone was partly forced backwards by atmospheric pressure and partly dragged backwards by the adherent heart, when drawn somewhat downwards by the diaphragm.

ENDOCARDITIS.

By Francis Sibson, M.D., F.R.S.

Endocarditis, to a greater extent even than pericarditis, is chiefly associated with acute rheumatism. The extent to which this is the case will be seen by the study of the accompanying table at page 621, from which it may be seen that endocarditis without pericarditis was established in one-third of the cases, or in 107 out of a total number of 325. If to these we add those cases with pericarditis that were also affected with endocarditis, amounting to 54, we find that endocarditis attacked one-half of the cases of acute rheumatism, or 161 in 325. In addition to these cases, in which the presence of endocarditis was rendered certain by the character of the signs and symptoms observed during the attack, there was a considerable proportion of the cases, amounting to one-fourth of the whole (76 in 325), in which endocarditis was either threatened (in 63) or very probable (in 13). Endocarditis is not, however, limited to acute rheumatism, being also present in a considerable proportion of cases affected with chorea, and in a small but uncertain number of those with pyæmia and Bright's disease. Cases, also, of established valvular disease of the heart are subject to intermitting attacks of endocarditis affecting the diseased valves.

I shall, in this article, (1) first give a brief account of the anatomical appearances that present themselves after death in endocarditis, and then (2) a clinical history of rheumatic endocarditis, as it presented itself in the cases with acute rheumatism under my care in St. Mary's Hospital, during the years 1851 to 1869–70; those cases being divided into two series, an earlier series from 1851 to 1866, and a later series, treated by means of rest, from 1867 to 1869–70.

I.—The Anatomical Appearances Observed in Cases of Endocarditis.

The anatomical appearances found after death in cases of endocarditis have been well described from actual observation in the excellent and readily available works of Rokitansky,[1] Hasse,[2] and Rindfleisch,[3] which have been well translated; and in the original and interesting lectures of Dr. Moxon[4] and manual of Dr. Payne.[5]

The inflammation of the interior of the heart is as a rule limited to the left ventricle, this being evidently due to the great labor to which that ventricle is subjected when it drives the blood into the arteries of the system, and to the comparatively slight effort with which the right ventricle sends its blood through the vessels of the lungs. In the fœtal state, the right side of the heart, which is then the most powerful side, and has the greatest amount of work to do, is subject to endocarditis, judging by the frequency with which the pulmonary valves are adherent, so as to contract the orifice of the pulmonary artery. Dr. Norman Cheevers finds that sixty such cases have been observed by various authors. The mitral

[1] Rokitansky, Pathological Anatomy, Syd. Soc. iv. 175.

[2] Hasse, Pathological Anatomy, Syd. Soc. 124.

[3] Rindfleisch, Pathological Histology, New Syd. Soc. i. 279.

[4] Dr. Wilks and Dr. Moxon, Pathological Anatomy, 125.

[5] Dr. Jones, Dr. Sieveking, and Dr. Payne, Pathological Anatomy, 384.

and aortic valves are the chosen seat of endocarditis, and especially the mitral valve. It is not, however, the whole of either valve that is the immediate seat of the inflammation; which, as a rule, is limited to the lines and surfaces of contact of the valves, close to the edges of their flaps where they come together and press against each other so as to close their respective apertures. The aortic valve is shut by the blood quietly filling the sinuses towards and at the end of the systole and during the diastole. The blood, when the sinuses are filled, presses the sides of the flaps against each other with a diffused and equal but firm pressure. This pressure is made on the first closure of the valve at the end of the systole, by the blood filling the sinuses; but this pressure is suddenly reinforced by the back-stroke or return wave of blood, caused by the recoil of the distended aorta and arteries, which propels the blood equally in every direction, forwards and sideways, as well as backwards with a return stroke, which beats on the aortic valve sinuses, and the ascending aorta, and which causes the second sound, which follows the closure of the valve by the tenth of a revolution of the heart's action. Afterwards the pressure of the aortic flaps upon each other is kept up during the diastole by the pressure of the blood, due to the steady contraction of the coats of the aorta and its branches. The pressure upon the aortic flaps bears, not upon their exact margins, but upon their surfaces of contact, a little within those margins, and upon the sesamoid bodies; and the endocarditis affects, not the exact margins of the flaps, but their surfaces of contact.

The mitral valve is shut on exactly the same principle as the aortic valve, by the pressure of the blood driven during the systole into the small open cells on the under or ventricular surface of the valve, in the manner described and figured at page 392. The force with which the blood presses upon the closed mitral valve, owing to the contraction of the ventricle, is much greater than the force with which the blood presses upon the aortic valve, owing to the recoil of the previously distended walls of the aorta. The flaps of that valve are pressed together by the backward portion only of the effect of the recoil of the aorta walls, which expands itself in every direction; and that force of recoil is itself but a portion of the original propulsive force of the left ventricle, which presses with its full power upon the closed mitral valve. The surfaces or lines of contact and closure of the mitral valve extend along and just within the borders of its two flaps. This border of contact is not a mere edge, but a surface or line of adaptation, made up of the small bead-shaped cells, that dove-tail into each other along the margins of the flaps; those flaps being held in their place by the simultaneous contraction of the papillary muscles, acting on their tendinous cords; the result is that the margins of contact of the mitral flaps press against each other when the valve is shut with much greater tension, force, and concentration, than the margins of contact of the aortic valve; under the triple agency of a finer margin of contact, greater pressure of blood, and the muscular force and tendinous traction proper to the valve. The mitral valve, which is situated in the muscular centre of the ventricle and in the focus of its internal inflammation, is more immediately and frequently subjected to endocarditis than the aortic valve, which has broader surfaces of contact, less pressure of blood, and no muscular and tendinous traction.

Endocarditis, as I have said, does not therefore attack the very rim of the flaps of the mitral valve at the attachment of their outspreading tendinous cords, but the line or margin of contact just within the edges of the valves. When the mitral valve is inflamed, a frill of small bead-like granulations lines the whole proper border of contact and closure of the valve; and tends to prevent their perfect adaptation, and to cause regurgitation through the valvular aperture when the ventricle contracts. These prominences consist of a swelling and granular disintegration of the connective tissue, with softening of the intercellular structure. Each of these prominences is covered by a cap of fibrin deposited from the blood in the manner well represented by Rindfleisch.[1] Endocarditis affects the surfaces of contact of the aortic valve in the same way that it affects those of the mitral valve.

This is the usual manner in which endocarditis affects the mitral and aortic valves, whether the parent affection, rendering those parts prone to inflammation, be acute rheumatism, chorea, or pyæmia. Sometimes, however, the inflammation deepens at its original seat on the surfaces of contact of the mitral valve, and extends beyond those surfaces, so as to affect a large portion of the flaps of the valve on their ventricular surface. Under these circumstances, the inflamed, softened, and thickened structures may undergo granular degeneration, and its ventricular layer may become broken or ulcerated. The auricular layer of the valve thus tends to yield before the pressure of the blood, which forces its way through the breach in the ventricular layer, and to form pouches or aneurisms protruding into the left auricle. The auricular layer may then be involved in the inflammation, and become in turn subjected to

[1] Loc. cit. p. 281, fig. 87.

granular disintegration and breaking up of tissue, so that the flap of the valve may become perforated. The fibrin of the blood deposits itself everywhere on the inflamed surfaces, often in the form of vegetations, which may become extensive; and thus the fibrin often lines, closes, and conceals the perforation.

We have already seen how many points in its favor, as regards its tendency to endocarditis, the aortic valve presents over the mitral; and it presents another in this respect—that while the pressure of the blood bears directly upon the inflamed surface of contact of the mitral valve during its closure at the time of the systole, the pressure of the blood does not bear upon the inflamed ventricular surface of contact of the aortic valve when it is closed at the time of the ventricular diastole, but upon the uninflamed upper or aortic surface of the valve. Although this condition, favorable to the aortic valve, exists, I have seen preparations in which a small aneurism, or aneurisms, of one or more of the flaps of the aortic valve protruded downwards into the ventricle.

The advantages are not, however, entirely on the side of the aortic valve when it is affected with endocarditis; for a serious counterbalancing disadvantage exists under such circumstances, as I shall now mention. The sesamoid body, and the margin or surface of contact of the valve on each side of the sesamoid body, which are the seat of endocarditis when it affects the aortic valve, receive the direct pressure of the column of blood in the aorta; and those parts, which are softened by the inflammation, tend therefore to be pushed downwards towards the ventricle during the ventricular diastole; with the effect of sometimes producing retroversion of the sesamoid body, and to a greater or less extent of the softened flap, of which it is the centre. We here see the great disadvantage in which the inflamed aortic valve is placed from the want of tendinous cords and papillary muscles to support its flaps when rendered soft and yielding by endocarditis.

Another special evil accruing to the aortic valve from a similar class of cause, is the tendency of the sesamoid body, and the adjoining portion of the flap affected with endocarditis, to lay hold of deposits of fibrin from the regurgitating stream of blood, with the effect of establishing a chain of fibrinous vegetations, which form one upon another, and which hang pendant into the left ventricle, being forced in that direction by the return current of blood. When this chain of fibrinous concretions forms upon either the right or the left posterior flap of the valve, it is driven downwards and backwards by the stream of regurgitation, so as to beat against and rest upon the anterior flap of the mitral valve, with the effect of causing ulcerative endocarditis of that flap. As the blood regurgitating from the aorta into the ventricle beats upon that flap, it parts with its fibrin which clings to the inflamed surfaces of the mitral valve, and forms on these a second chain of fibrinous concretions.

The flaps of the mitral valve are, as we have seen, the principal seat of endocarditis, but inflammation may also attack the papillary muscles, and especially where they are brought into contact with each other towards the end of the systole, and cause fibroid degeneration of those muscles. The tendinous cords may also sometimes become inflamed, softened, and disintegrated, when the grave result of rupture of the cord may ensue.

I have just given a series of notable instances of the occurrence of endocarditis, locally excited by the contact with each other of the two opposing surfaces of the valve; of two adjoining papillary muscles; and of a pendant chain of fibrinous concretion beating against the anterior flap of the mitral valve. These are not the only parts of the interior of the heart that may be inflamed from this cause, for wherever two surfaces of the endocardium come into contact with and rub against each other, endocarditis may be excited in both of those surfaces. The influence of the labor of the left ventricle and the mutual contact of its internal surface in tending to produce endocarditis is illustrated in an original and able manner by Dr. Moxon. I would refer to his work and to the others already named for the study of the various effects of endocarditis.

Among the effects of endocarditis, I would here simply name the formation of vegetations on the inflamed valves, already in part illustrated; the production of embolism by the washing away from the vegetations of fibrin into the current of the blood; the ulceration of the surface of the endocardium; the establishment of valvular disease from the thickening and enlargement of the valves; the contraction, adhesion, or retroversion, and perforation of their flaps; the rupture of the tendinous cords; the formation of aneurisms of the valves; the fibroid and atheromatous degeneration of the fibrous and muscular structures of the ventricle; the production of aneurisms of the heart; and other effects that will be found described in the works to which I have referred.

II.—CLINICAL HISTORY OF RHEUMATIC ENDOCARDITIS.

The accompanying analytical tables of 325 cases of acute rheumatism under my

care in St. Mary's Hospital during the years 1851–66, show the proportion in which those cases were free from endocarditis, and were threatened with or attacked by that affection; and the number that were attacked by pericarditis, distinguishing those with established endocarditis; also those in which endocarditis was doubtful, and those in which it was absent.

The analyses contained in the tables sufficiently indicate the reasons for arranging the cases in the manner adopted.

TABLE SHOWING THE CONDITION OF THE CASES OF ACUTE RHEUMATISM, WITH ESPECIAL RELATION TO THE ABSENCE OR PRESENCE OF ENDOCARDITIS.

I.—Cases of Acute Rheumatism in which there was no Endocarditis.

Affection of joints somewhat severe or moderate, no general illness, no palpitation, signs over heart not named	2
Joint affection slight, some general illness, heart not named	13
Joint affection not, or scarcely severe, some or little general illness, heart sounds healthy	10
Joint affection not, or somewhat severe, some or considerable general illness, heart not named	5
Joint affection not severe, some or considerable general illness, heart sounds healthy	10
Joint affection severe, some general illness, heart not named	6
Joint affection somewhat severe, considerable general illness, heart sounds healthy, or loud and ringing	7
Joint affection severe, some general illness, heart sounds healthy	11
No description of state of joints, or general illness, heart sounds feeble	1
Joint affection not, or rather severe, slight or no general illness, slight prolongation of first sound	7
Joint affection rather severe, slight or no general illness, doubtful occasional obscure murmur	1
Previous valve-disease, mitral regurgitation	2
Death, delirium	4
I.—Total	79

II.—Cases of Acute Rheumatism in which Endocarditis was threatened.

Some general illness, pain over the cardiac region, heart not named	1
Great general illness, pain left side, or region of heart, signs of heart not named	2
Great general illness, pain left side, heart sounds healthy	3
Great general illness, pleurisy, heart sounds healthy	1
Great or considerable general illness, pain left side, or region of heart, heart sounds healthy	8
Great general illness, delirium, pain left side	1
Considerable general illness, first sound very loud	3
Considerable general illness, doubling of first sound	1
Considerable general illness, first sound or heart sounds feeble or indistinct	3
General illness, pain over region of heart or left side, first sound indistinct or muffled	2
Slight general illness, prolonged first sound	13
Great general illness, prolonged first sound	3
Great general illness, lung affection, prolonged first sound	4
General illness, pain in region of heart or chest, prolonged first sound	10
Little general illness, faint or obscure murmur early or late in the attack	5
Considerable general illness, obscure murmur after cessation of attack (endocarditis probable)	1
Previous valve disease, considerable general illness	2
II.—Total	63

III.—Cases of Acute Rheumatism in which Endocarditis was probable.

Great general illness, pulmonary apoplexy in 1, prolonged first sound (situation unknown), almost a murmur in 1, a pulmonic murmur in 1	2
Great general illness, severe cough in 2, prolonged first sound at apex, almost a mitral murmur in 2, almost a tricuspid murmur in 1, a pulmonic murmur in 3	4
Great general illness, prolonged first sound at right ventricle, almost a tricuspid murmur, and a pulmonic murmur	1
Slight general illness, tricuspid murmur, ending in prolonged first sound in 1	2
Slight general illness, previous or established mitral regurgitation murmur did not vary materially in 1, murmur became louder in 1	2
Considerable general illness, previous or established mitral-aortic regurgitation, aortic murmur absent at first in 1, mitral murmur became musical in 1	2
III.—Total	13

IV.—Cases of Acute Rheumatism in which Endocarditis *was present without Pericarditis.*

Prolongation of first sound, almost a murmur, pain in heart 1, in chest 1, extreme general illness		2
Tricuspid murmur, murmur absent on recovery	7	
Tricuspid murmur, murmur lessening on recovery	6	
Tricuspid murmur—Total	—	13
Mitral murmur, murmur disappearing on recovery	25	
Mitral murmur, murmur lessening on recovery	10	
Mitral murmur, murmur established on recovery	14	
Inflammation of mitral valve, died, murmur in 1, no note of murmur in 1	2	
Mitral endocarditis total, mitral murmur in 50, no note of murmur in 1	—	51
Aortic murmur, murmur disappearing on recovery	5	
Aortic murmur, aortic regurgitation established on recovery	5	
Aortic murmur—Total	—	10
Mitral-aortic murmur, murmur disappearing on recovery	3	
Mitral-aortic murmur, mitral murmur established, aortic murmur disappearing	2	
Mitral-aortic murmur, mitral-aortic regurgitation established	4	
Mitral-aortic murmur—Total	—	9
Previous valvular disease, mitral regurgitation	6	
Previous valvular disease, mitral and tricuspid regurgitation	3	
Previous valvular disease, mitral regurgitation, adherent pericardium aortic regurgitation	1	
Previous valvular disease, aortic regurgitation	3	
Previous valvular disease, mitral-aortic regurgitation (tricuspid murmur 2)	9	
Previous valvular disease—Total	—	22
IV.—Total cases of Endocarditis		*107

V.—Cases of Acute Rheumatism with Endopericarditis.

Heart Previously healthy, 46.	Tricuspid murmur,	3	Murmur disappearing on recovery,	1	3
			Murmur established on recovery,	2	
	Mitral murmur,	36	Murmur disappearing on recovery, mitral 17, aortic 1, mitral aortic 1	19	
			Murmur lessening on recovery, mitral,	8	43
	Aortic murmur,	1			
	Mitral-aortic murmur,	6	Murmur established on recovery, mitral 11, mitral-aortic, 5	16	
Total cases of endocarditis in which the heart was previously healthy					46
Cases of endocarditis with previous valvular disease, mitral 5, mitral-aortic 3					8
Total cases with endopericarditis					54
IV., V.—Total with endocarditis					161
VI.—Cases of Acute Rheumatism with Pericarditis; Enodcarditis being doubtful					3
VII.—Cases of Acute Rheumatism with Pericarditis in which there was no Endocarditis					6
V., VI., VII. Cases of acute rheumatism with pericarditis.—Total					63
Grand total of cases of acute rheumatism					325

* 108 cases of endocarditis appear in the tables at pages 475–476. I find that one of those cases has been accidentally enumerated twice over, a woman, aged 23.

I have considered the cases of endocarditis according to the character of the valvular affection of the heart due to the inflammation of the interior of the ventricle, and have arranged these cases into those (I.) with an uncomplicated tricuspid murmur; (II.) with mitral regurgitation; (III.) with aortic regurgitation, (1) not accompanied by a mitral murmur, and (2) accompanied by a mitral murmur; (IV.) with prolongation of the first sound without a murmur; (V.) with endocarditis supervening upon previous valvular disease.

I.—Cases of Rheumatic Endocarditis with an Uncomplicated Tricuspid Murmur.

In a moderate proportion of the cases of rheumatic endocarditis under my care in St. Mary's Hospital during the fifteen years ending 1866—amounting to 13 out of a total number of 107, or one in eight—there was a murmur over the right ventricle from regurgitation through the tricuspid valve, without a mitral murmur. In nearly all of these cases there was a greater or less amount of general illness, and in one-third of them (4) there was pain in the region of the heart. A tricuspid murmur was present also in 2 cases, in which endocarditis was probable, and in 2 that have been included, with a little doubt, among the cases of pericarditis.

In the majority of these cases the murmur had disappeared when recovery was established; and in the remainder the murmur was then diminishing in loudness, extent, and clearness.

This tricuspid murmur is usually pres-

ent over the body of the heart, or, in other terms, over the right ventricle; and extends from the lower half of the sternum to a line a little within the left nipple, which line corresponds with the ventricular septum, and from the third to the sixth cardiac cartilage. The presence of this murmur in these cases over the right ventricle in the early stage of endocarditis, and that, too, when no other murmur prevails, naturally suggests to the mind at first sight that it is due to endocarditis affecting the right ventricle and the tricuspid valve.

This inference is, however, forbidden by the following considerations:—

(1) Endocarditis and disease the result of endocarditis of the tricuspid valve, are very rarely discovered on dissection in those who have died from rheumatic inflammation of the interior of the heart, or from valvular disease, the effect of such inflammation.

(2) The tricuspid murmur, when uncomplicated with disease of the mitral valve, was not established in any of my cases, but had either ceased altogether, or was steadily declining on the recovery of the patient.

(3) The tricuspid murmur was frequently associated with a mitral murmur, and less often with a mitral-aortic or an aortic murmur of recent origin.

A tricuspid murmur was present over the right ventricle in one-half, or 27 in 50, of the cases with recent mitral murmur. In 7 of those 27 cases the presence of a tricuspid murmur was somewhat doubtful. In eight of those cases the mitral was preceded by the tricuspid murmur, and in six of these the tricuspid murmur had ceased to be audible when the mitral came into play. In thirteen other cases both murmurs were present when they were first noticed, which was at the time of admission, in fully one-half of those patients. The mitral murmur appeared before the tricuspid in five cases. The tricuspid murmur disappeared when the mitral murmur was still audible in two-thirds of the cases (16 in 27); both murmurs ceased at the same time in seven instances; and in four the tricuspid murmur outlived the mitral. A tricuspid murmur was also present in one-third (3 in 10), of the cases of endocarditis with mitral disease of old standing.

A tricuspid murmur was present in two or three of the eight cases of mitral-aortic, and in about four of the ten cases of aortic, regurgitation of recent origin; and in two of the five cases with aortic, and none of the seven instances with mitral aortic valvular disease of old standing affected with endocarditis.

(4) I have observed tricuspid regurgitation as a marked and lasting feature in a case of button-hole contraction of the mitral valve; in several instances in which the tissue of the lung was permanently condensed, owing to repeated attacks of bronchitis; in patients affected with contracted granular kidney, in whom obstruction of the pulmonary circulation, with enlargement of the right ventricle, had followed upon obstruction of the systemic circulation, with its attendant tension, dilatation, and thickening of the systemic arteries, and hypertrophy of the left ventricle.

These circumstances point irresistibly to the conclusion that the tricuspid regurgitation is usually due to the so-called "safety-valve" function of that valve, and not to endocarditis of the right side of the heart. In all these cases resistance to the flow of blood through the lungs has induced tension of the pulmonary artery, and distension of the right ventricle and auricle, with, as a result, incomplete closure of the tricuspid valve. The pent-up blood flows back through that aperture, and upon the veins of the system; with the effect of distending those veins, and of giving proportionate relief to the blood gathered up in excess in the pulmonary vessels. At each contraction of the right ventricle, indeed, instead of the whole of the blood flowing forwards into the over-charged pulmonary artery, a portion of it flows backwards into the right auricle, and venæ cavæ.

Inflammation of the left side of the heart, even when there is no regurgitation through the mitral orifice, impedes the flow of blood from the lungs into that side of the heart; and the accumulation of the blood in the pulmonary vessels, thus caused, induces and is relieved by the tricuspid regurgitation.

The tricuspid murmur was present on admission in two of the thirteen cases of endocarditis in which that murmur existed without mitral regurgitation. In nine of the remaining cases, the tricuspid murmur was not observed until from two to seven days after admission, and generally on the fourth or fifth day. In one case the murmur did not appear until the 26th day after admission.

In nine of these instances the duration of the illness before their admission is stated. In one of them the murmur appeared on the 7th day; in five, from the 10th to the 12th; and in two, from the 14th to the 16th day after the beginning of the attack of acute rheumatism; and we may therefore infer that the tricuspid murmur generally comes into play about the 10th or 12th day of the primary attack.

In four instances the murmur was preceded by a prolonged first sound over the right ventricle, and in one by a very loud, and in another by a peculiar booming first sound.

In five of the cases there was direct evidence of endocarditis at the time of admission, in the shape of pain in the heart, and a prolonged first sound; although the murmur did not pronounce itself fully until several days had elapsed. In two of them, indeed, the murmur did not appear until there was a marked improvement in the general symptoms.

The duration of the tricuspid murmur in these cases was very variable. In two instances it was only observed once, and in eleven it disappeared in from two to nineteen days; in eight the murmur when last noticed had become much more feeble, and in three of these the first sound became prolonged at the apex, at the time that the tricuspid murmur was diminishing. In three cases a pulmonic murmur, which indicates lessened tension of the pulmonary artery, appeared when the tricuspid murmur was lessening.

From these observations we are entitled, I consider, to infer: 1. That the appearance of a tricuspid murmur over the body of the heart, extending from the sternum to the nipple, and limited to that region, which corresponds to the right ventricle, is usually the effect and the evidence of endocarditis affecting the left side of the heart. 2. That when this murmur is neither coupled with nor replaced by a mitral murmur, we may safely foretell that when the inflammation leaves the heart, the valves will be perfect and the organ free from disease.

A tricuspid murmur, as I have already remarked, is often the prelude, and for a time the accompaniment, of mitral murmur in cases of rheumatic endocarditis. The latter murmur, however, in two-thirds of the cases (16 in 27) outlives the former, which is essentially a transient murmur. I have already given the proportion in which mitral regurgitation is accompanied, preceded, or followed by a tricuspid murmur (see p. 622).

The duration of the tricuspid murmur in these cases, in which it was associated with a mitral murmur, though variable, was usually short. In ten instances it was only heard once, and that generally on the day of admission, but in one-half of these the existence of the murmur was doubtful; in six cases it was audible for from two to seven days, and in seven from nine to sixteen days; while in three, after a short duration, it vanished and reappeared after about twenty days, and in another case after a much longer period.

The tricuspid murmur appeared much earlier in a large proportion of those cases in which it was associated with mitral regurgitation than in those in which it was the only murmur audible. The murmur was present at the time of admission, or on the second day—in two-thirds of the cases (19 in 27), in which there was both a tricuspid and a mitral murmur, and in only one-sixth (2 in 13) of those in which the tricuspid murmur was alone audible. This contrast between the two sets of cases is more striking if we date the murmur from the beginning of the attack. The tricuspid murmur appeared on or before the eighth day in at least nine cases in which there was both tricuspid and mitral regurgitation; and in one only in nine of the cases in which the tricuspid murmur was alone audible. In one of the cases in which both murmurs were in full play on the day of admission, the patient had been ill only two days, in two others four days, and in three others a week. These cases of combined mitral and tricuspid regurgitation, in respect to the more rapid development of the murmur, and not in that respect only, present greater intensity, energy, and rapidity of inflammation in the left cavities of the heart, than in the cases in which the tricuspid murmur was alone audible. In almost all the cases of tricuspid incompetence there was at the time of admission great general illness; but this and other points of clinical interest must be reserved until mitral regurgitation is specially considered. In four of these cases the tricuspid murmur was replaced on its disappearance by a transient prolonged first sound over the right ventricle. The tricuspid regurgitation reappeared after being absent for a longer or shorter period in five of the patients. In four of these the renewed tricuspid murmur was conjoined with mitral murmur, but in the remaining one it cropped up alone 47 days after it had disappeared, and 34 days after the cessation of the mitral murmur.

The tricuspid murmur is easily recognized by its position and character. It is distributed over the right ventricle from the sternum to the nipple and from the 3d cartilage to the 6th, it usually stops at the septum, occasionally extends over the right auricle, to the right of the lower sternum, and is sometimes audible over the epigastrium below the lower boundary of the heart. The tricuspid murmur is usually grave or even vibrating in tone, and superficial, and it begins with an accent or shock, and ends with the second sound.

In cases of extensive mitral regurgitation without tricuspid murmur, the first sound is feeble while the second is intensified over the pulmonary artery, owing to the tension of that artery, the second sound being usually loud over the right ventricle, and sometimes even at the apex.

When, however, mitral is coupled with tricuspid regurgitation, the blood is thrown back upon the right auricle and the venæ cavæ, the tension of the pulmonary artery is relieved, and the first sound

over that artery is moderately loud, or prolonged, or even murmuring; and the second sound, though perhaps rather loud, ceases to be intensified.

The mitral murmur is usually softer and less grave in tone than the tricuspid, being more like a bellows-sound; it appears also to be deeper; and its point of greatest intensity is situated to the left of the nipple, and, in endocarditis, towards the axilla. When the mitral murmur is loud and vibrating, and especially if accompanied by a thrill over the apex, perceptible to the finger, it is heard very extensively, radiating in every direction. It then becomes audible over the right ventricle. This transmitted mitral murmur over that ventricle is readily distinguished from the tricuspid murmur originating within the right ventricle itself; for the transmitted or mitral murmur is accompanied and more or less masked by the normal first sound of the right ventricle; while the immediate or tricuspid murmur, besides being grave and shallow, begins with an accent, and is inseparably incorporated with the first sound of the right ventricle.

When the mitral and tricuspid murmurs coexist, it is usually easy to distinguish them from each other upon the principles just stated; for the tricuspid murmur over the right ventricle is then palpably more superficial than the apex murmur, instead of being less so, as it is when the mitral is alone audible; the first sound of the right ventricle does not mask or muffle the murmur; and the difference in tone of the two murmurs is perceptible, the mitral being soft and smooth, the tricuspid grave or vibrating. Two cases were typical instances of this difference in tone of the two murmurs when thus coexisting; in one of them the mitral murmur was a soft bellows-sound, while the tricuspid murmur was grave; and in the other the tricuspid murmur was harsh and grating, while the mitral was soft. When the mitral murmur is rasping and vibrating in character, the difficulty of distinguishing the presence of a conjoint tricuspid murmur is increased. An instance of this was presented by a patient in whom the apex murmur was short and rasping, while there was a bellows sound over the right ventricle. Here the rasping mitral murmur might have become softened by its transmission through the ventricle.

It is sometimes difficult to distinguish between a tricuspid murmur and a friction sound, especially when the latter is murmur-like in character, as it frequently is at the beginning and towards the end of an attack of pericarditis. The chief points of distinction are—that the friction sound is usually double or to-and-fro in character; the tricuspid murmur being single: the friction sound is not exactly rhythmical with the heart sounds, those sounds being readily heard distinct from the friction sound when that sound is not loud and grating, so as to extinguish every other noise; the tricuspid murmur is incorporated with the heart sounds; the friction sounds starts off without a shock, and retains the same tone throughout; the tricuspid murmur begins with an accent or shock. The pressure test usually clears up every doubt. When the stethoscope is applied over the right ventricle with increased force, the tricuspid murmur may be intensified, but is not materially changed in character; while the friction sound is usually both intensified and changed in tone, it ceases to be murmuring, and becomes grazing, rubbing, grating, or creaking in character.

When pericarditis supervenes upon a tricuspid murmur, the pressure test is sometimes in the early stage almost essential to the discovery of the friction sound; sometimes, however, the patient under these circumstances is so ill that you cannot make pressure. Local pain will then usually guide the treatment, and time will clear up the obscurity.

In five of my cases, aortic regurgitation was accompanied by a tricuspid murmur; and in two of these by a mitral murmur also.

Cases of endocarditis with aortic regurgitation present obstruction to the flow of blood through the lungs, and so may cause tension of the pulmonary artery and tricuspid regurgitation; more, however, owing to the inflammation of the interior of the left cavities and the mitral valve itself, than to the aortic regurgitation, which is rarely sufficient in volume to induce congestion in the lungs. This is shown by the clinical fact that there were four instances with tricuspid murmur in the sixteen cases of endocarditis in which there was recent aortic regurgitation, in seven of which there was mitral regurgitation also; while there was no instance of tricuspid murmur in the fourteen cases of endocarditis in which there was aortic regurgitation owing to the previous disease of the valve, in one-half of which cases there was mitral regurgitation also.

A tricuspid murmur was present in three cases of endo-pericarditis; and in two of those cases the murmur was persistent; while in one of them it disappeared, after the recovery from acute rheumatism.

I will give here the proportion in which a tricuspid murmur was present in cases of acute rheumatism with endocarditis under my care from October, 1866, to 1869, treated by means of rest.

There were altogether 31 cases of endocarditis in a total of 74 of acute rheumatism, and in none of those thirty-one cases

was one tricuspid murmur present without a mitral or other murmur.

While the tricuspid murmur unaccompanied by another murmur was absent in those cases; although it was present in the proportion of one in eight of such patients treated during the previous fifteen years; the proportion in which the conjoint tricuspid and mitral murmurs were present was fully maintained in the cases treated by rest. Mitral regurgitation was present without aortic regurgitation in twenty of those cases, and of these, tricuspid murmur was present in nine, or if we add two doubtful cases, in eleven instances.

In none of these instances did the tricuspid murmur precede the mitral; in four the two murmurs appeared at the same time; in four the mitral preceded the tricuspid murmur by from one to three days, and in one (45) by nine days.

In three of these cases the mitral murmur outlived the tricuspid; in two it was the reverse; in three they were combined to the last, and in the remaining case the mitral murmur probably lasted beyond the tricuspid.

The relation of prolongation of the first sound over the right ventricle to tricuspid murmur will be considered at pages 628, 639.

II.—Cases of Rheumatic Endocarditis with a Mitral Murmur.

The mitral and the tricuspid valves, while they correspond in general structure and function, differ essentially in the construction and arrangement of their flaps and in the whole setting of the valve.

The tricuspid valve, as I have already stated, is composed of three great flaps and several intervening small ones, which meet somewhere about the centre of the valve; and the aponeurotic ring which forms the base of those flaps is surrounded on all sides by muscular walls. (See figs. 47, 48, p. 385; and figs. 59, 60, 61, pp. 395, 396.)

In health, when the ventricle is not over-distended, the flaps of the valves adapt themselves to each other perfectly, and close the tricuspid aperture completely during the contraction of the ventricle.

When, however, the cavity is over-distended, as it is under the various circumstances which I have already described, the flaps of the valve adapt themselves only partially to each other, especially, so far as I have observed, at the meeting-point of the three great flaps, and regurgitation ensues. The so-called "safety-valve" function of the valve is thus brought into play, with the effect of relieving the tension of the vessels of the lungs, and throwing the blood backwards upon the veins of the system.

The result is that the tricuspid murmur is, with rare exceptions, not a sign of inflammation of that valve, but of the over-distension of the right ventricle, caused by obstruction to the flow of blood through the lungs.

The mitral valve is formed of one great semilunar or convex flap, the base of which is incorporated with the powerful aponeurotic structure that is continuous with the two posterior sinuses of the aorta; and of a crescentic or horse-shoe flap, complex in structure, being formed of three segments, set in the muscular walls at the base of the left ventricle. The setting of the base of the valve is therefore two-thirds muscular and one-third aponeurotic. There is no tendency in the aperture to widen outwards at the base of the valve equally in all directions, for the aponeurotic structure, when healthy, though elastic, is practically unyielding. The single anterior semilunar flap, held in check by its proper cords and fleshy columns, fills up the posterior crescentic flap with perfect adaptation. The edges of the opposed flaps press against each other with increasing force in proportion to the increasing pressure of the blood on their under surfaces; and the over-distension of the left cavity does not, owing to the structure to which I have alluded, readily tend to widen the orifice and open up the valve. The healthy mitral valve, therefore, when the left ventricle is not greatly enlarged, possesses only under circumstances of extreme backward pressure or forward resistance a function like the "safety-valve" function of regurgitation with which the tricuspid valve is endowed. Such a function of the mitral valve would indeed be the opposite of a "safety" valve function, for it would immediately endanger the lungs by throwing the blood backwards upon their vessels. (See figs. 47, 48, page 385; and figs. 52–58, pp. 391–393.)

The result is that when the right ventricle is over-distended, it relieves itself backwards through the tricuspid aperture upon the veins of the system; and that when the left ventricle is over-distended, it, with rare exceptions, relieves itself directly forwards upon the arteries of the system, and so the lungs are spared in both instances.

I derive the more important evidence of the correctness of this view from the well understood pathological history of aortic regurgitation from widening of the orifice of the aorta, owing to atheroma of its walls. In those cases the cavity of the left ventricle becomes greatly, sometimes enormously, enlarged, and yet I know of

comparatively few instances of this kind in which the mitral valve was therefore incompetent.

Mitral regurgitation, without disease of the structure of the valves, occurs most frequently among cases in which there is great arterial tension owing to Bright's disease, and great consequent distension of the left ventricle; in which cases there is often also an atheromatous, or thickened state of the mitral valve, with, as an effect, widening of the fibrous portion of that aperture, and possible regurgitation.

Mitral murmur is, as a rule, neither a sign of over-distension of the left ventricle, nor of a supply of blood to that cavity too small in amount, or too thin in quality.

The existence then of a mitral murmur in a first attack of acute rheumatism is a direct sign of inflammation affecting the left side of the heart.

Mitral regurgitation, not connected with previous disease of the valve, and without aortic regurgitation, was present in 50 out of 107 cases of rheumatic endocarditis under my care in St. Mary's Hospital, from 1851 to 1866, and in 20 of 31 such cases treated by rest from 1866 to 1869. In twenty-five of the earlier series of cases the murmur had disappeared, and in ten others it was lessening at the time of the patient's recovery, while in fourteen of them the murmur seemed to be established; and it was absent in one and present in the other of two fatal cases of mitral endocarditis at the time of death. In the cases of the later series the corresponding numbers were thirteen, four, and three, the latter being the only cases in which the murmur was established at the time of the patient's recovery.

In one-half of the cases of both sets the mitral murmur was heard on the day of admission or the next day; the numbers being 28 in 50 of the first set, and 9 in 20 of the second set. The murmur presented itself within six days of admission in three-fourths of the remainder, or seventeen of the earlier and nine of the later series, and from 8 to 17 days after admission in the remaining cases, amounting to one-seventh of the whole.

Among the thirty-seven cases of endocarditis, combining the two series, admitted with mitral murmur, one-third, or eleven, had been ill from 2 to 7 days, nearly one-half, or fifteen, from 8 to 14 days, six from 2 to 4 weeks, two for a longer time, and three for an unknown period.

The mitral murmur became audible after admission in thirty-six cases, and of these the murmur appeared in six during the first 7 days, in eleven from 8 to 14 days, and in eight from 15 to 28 days after the beginning of the attack of acute rheumatism; in six at a later period; and in three at a time unknown.

The mitral murmur may be present in full force on the third day of the attack, or its appearance may be delayed until the fortieth day. In a fair proportion of the cases, amounting to one-fourth, it is developed during the first week, and in the larger number, or two-thirds, before the end of the second week.

General illness. — In nearly every case of endocarditis the patient presents great or considerable general illness. Thus in sixty-two of the seventy-one cases of mitral endocarditis the illness was great or considerable, in two it was definite, and in five it was slight; while in two there is no description of the general state of the patient.

In most of the few exceptions to this rule of the presence of great general illness in these cases, the murmur was established at the time of their admission, and the severity of the attack was already mitigated or passing away.

Those cases in which there was no endocarditis, present a very different aspect, since in scarcely one-third of them was there considerable general illness.

As might be expected, constitutional illness was more severe and frequent in those instances in which there was a threat of endocarditis, though its existence was not actually demonstrated by valvular incompetence, since in nearly two-thirds of them the general illness was either great or considerable.

The illness in cases of endocarditis is peculiar. It differs from and is superadded to that due to simple rheumatic inflammation of the joints, and is such as to call the attention of the physician to the state of the heart.

The face may be flushed all over, the forehead, nose, lips and chin being of as high a color as the cheeks, a state that is usually associated with profuse perspiration, drops of sweat standing in beads on the surface—a condition, however, that may be present in cases with severe affection of the joints without endocarditis. Thus when endocarditis exists the face loses the brightness, glow, and smoothness, and the variety of hue and tone of health, and becomes clouded, being dusky, dull, or ashy in hue, or glazed, or unduly white, or even of a bluish tint. The countenance, no longer expressive of interest in things and persons around, or even of pain in the limbs, is marked by internal trouble. The aspect of the patient is altered, often profoundly so, being anxious, depressed, or indifferent. The eye loses its lustre and expression, and becomes heavy and dull.

Sleep is often absent, the nights being restless; but this is perhaps more often due to the inflammation of the joints than to that of the interior of the heart.

The nervous system is often gravely

affected. Delirium at night, the patient wandering, muttering, and complaining, is occasional, but rare; it occurred in two instances, in which the affection of the heart was evidently the primary exciting cause of the mental trouble. In another patient the head was confused on the third day.

Choreal movements, as we have seen, are in some instances a definite effect of endocarditis, especially of the non-rheumatic kind, traceable frequently to cerebral embolism; but choreal movements, and indeed embolism, were of very rare occurrence in my cases of rheumatic endocarditis uncomplicated with pericarditis. In one instance the patient, previously anxious, and with sordes on his teeth, was nervous and fidgetty; and in another, starting appeared on the 6th day, having been preceded on the 4th day by pain in the heart.

Sickness is occasionally present. It was so in four of my cases. These cases, however, point not to the stomach as the cause of sickness, but rather and usually to the state of the nervous system, and more immediately to that of the brain itself; as in a case in which giddiness and sickness appeared together, and in another in which sickness was preceded by restlessness.

Failure in the power of the heart is an occasional occurrence in cases of endocarditis. Thus, two of my patients were attacked with fainting. One of these fainted on the day of admission, and again on the thirteenth, and on the following day was sick, so that failure of the heart may be a cause of sickness. In the other case pain in the heart and fainting appeared on the seventeenth day after admission. We may fairly attribute the fainting in these cases to the actual failure of the heart itself, caused by the internal inflammation of that organ.

The pulse is often quick, feeble, and fluctuating. I believe that it is dichrotous, but I have not employed the sphygmograph in any case of endocarditis, being perhaps deterred by the state of the wrist.

Perspiration is often especially profuse and of long continuance; sudamina being also present in some of the more severe cases.

The breathing is usually affected, being more or less quickened. In rare instances pulmonary apoplexy or extravasation is the result of the difficulty to the flow of blood through the lungs, which is the general effect, varying in degree, of endocarditis.

The chain of symptoms here described points mainly to the affection of two great functions. The nervous power is lowered; and the circulation of the blood through the fine vessels of the lungs and the body is enfeebled.

Pain in the Region of the Heart.—Pain in the region of the heart, sometimes severe and lasting, sometimes slight or transient, amounting perhaps only to uneasiness, was present in about one-fourth of the cases of tricuspid and of mitral murmur belonging to the earlier series, and in one-half of the later series, treated by rest. If to these we add other cases having mitral or tricuspid murmur in which there was pain in the left side, or in the chest; the proportion thus affected reaches to nearly one-half in the first series, and to fully one-half in the second.

The pain in the heart was sometimes, but not generally, severe. In a few instances the pain was increased or excited by pressure. We may fairly infer that in those cases pericarditis was imminent or was actually present, though not, except in rare instances, with such intensity as to cause even a transient friction sound.

Palpitation was very rarely complained of, but fainting, as I have already stated, occurred in two instances.

Prolongation of the First Sound occurring during the Early Period of Mitral Endocarditis.—In one-half of my patients affected with mitral regurgitation, as we have just seen, a murmur was established at the time of admission. In one-half of the cases in which the murmur was not thus established, prolongation of the first sound preceded, and was merged into, the murmur. In all but one of those cases the first sound was prolonged at the time of admission, and in that case and two others a tricuspid murmur was then in full play.

The tricuspid murmur was likewise heralded by prolongation of the first sound in one-half of the cases in which that murmur was not already present at the time of admission.

In a number of the cases, the exact position of the prolongation of the first sound was not defined; but wherever it was so, the mitral murmur was preceded by prolongation of the first sound at the apex; and the tricuspid murmur by prolongation of the first sound over the front of the heart, or the right ventricle.

I think that no cardiac sign is more readily recognized than prolongation of the first sound, and yet there is none so difficult to define. That this is so, however, is natural, for it is a transition sound. It forms, as we have just seen, the transition from a clear healthy first sound to a murmur; and as we shall see, at a later period, in a large proportion of the cases, it forms a transition between a mitral or tricuspid murmur when dying out, and the restoration of the healthy first sound. In one-half of the cases in which the prolongation preceded the murmur, there was a double transition, the murmur being both preceded and followed by prolongation of the first sound. This

prolongation is sometimes so like a murmur that it is difficult to make the distinction, and this is especially the case just before the time of transition, when the prolongation precedes the murmur; and just after that time, when it follows the murmur.

Prolongation of the first sound is the absence of silence and the presence of a wavering, grave, feeble sound during the interval between the first and second sounds. It is not the prolongation of the shock of the first sound which is itself significant, being sometimes a precursor of the more telling signs of endocarditis. The prolongation of the first sound is not the same as the natural loud vibrating character of that sound over the superficial cardiac region which is almost always present in cases of anæmia, when the muscular force of the ventricles is maintained, and even in excess, but when the blood is scanty and thin, being deficient in red corpuscles.

Prolongation of the first sound is, I repeat, a feeble, indeterminate, wavering sound, that fills up the space between the first and second sounds, which space is silent in health. It presents every gradation, from a sound so feeble that it is with difficulty discovered, to a sound so murmurlike that it can scarcely be distinguished from the murmur into which it so often ripens. Prolongation of the first sound was noticed on the first day of observation in fourteen cases; the prolongation developed into a murmur in two-thirds or nine of those cases before the seventh day after admission; and in the remaining third, or five, between the seventh and fourteenth days. In two other instances the prolongation, absent on the day of admission, appeared on the following day, and in the other after a lapse of four days.

It is evident that in all these cases the endocarditis was present before the appearance of the murmur for a period of time at least as long as the previous period of duration of the prolongation of the first sound.

There are other modifications of the first sound, besides its prolongation, that point to endocarditis, if they do not indicate it, which have been, in a few instances, the precursors of murmur. It will be sufficient if I simply name them. They are—1. Loud heart sounds, the first being sharp, the second ringing; or both sounds may be ringing. 2. Healthy sounds with powerful action of the heart. 3. Roughness of the first sound. 4. A humming noise over the right ventricle, and in one case at the apex, where it was associated with murmur. 5. Doubling of the first sound (over the ventricle), which occurred in two cases associated with a prolonged first sound, which was not followed by a murmur in one of those cases. 6. Feeble first, loud second sound, followed by tumultuous action of the heart and mitral and aortic murmurs. 7. Extensive presystolic murmur (*rrrp*) present in one case for five days, followed in succession by loud heart sounds (6th day), doubling of the second sound (15th day), and a faint mitral murmur, not limited to the apex. 8. Loud "plunging" first sound over both ventricles, present on the 4th day, followed by prolongation of the first sound on the 6th, and mitral murmur on the 8th; and 9, muffling of the first sound, which in one case succeeded the murmur, which was extinguished by an attack of pain in the heart, followed by fainting.

All the above varieties in character of the first sound were, in the instances referred to, followed within a very few days by a mitral murmur.

The only one of these varieties of the first sound that I would speak of is the last: the peculiar "plunging" sound. I call it so for want of a better name. The sound is something like what I have heard in the working of a steam-engine. It was as if the piston made a peculiar plunging sound when it dipped down and reached the bottom of its play. I have heard this sound in at least three cases. One of them was attacked afterwards with delirium, long torpor, almost coma, extreme depression, and pericarditis, but no murmur. In all the cases, the constitutional symptoms more or less threatened endocarditis.

Besides these peculiarities of the first sound preceding mitral murmur, there is one other affection of the sounds of the heart that I would name; and that is a complete silence of both sounds; which occurred in one case threatened with endocarditis, in which a mitral murmur did not appear. In that case there was tenderness over the heart, fighting for breath, a piercing pain between the chest and back, and great depression, lasting for some days. On the 8th day she looked more bright, on the 9th the sounds of the heart were audible, on the 14th its impulse had returned and was gaining power, and on the 26th day the sounds were of natural loudness, and there was no murmur.

In most of the cases of endocarditis with mitral murmur there is undue, but not great, strength of the impulse of the right ventricle, which may be seen and felt between the cardiac cartilages to the left of the lower sternum. This is found even in the earlier stages, and before the appearance of the mitral murmur.

It is evident from what has just been stated, that while in some cases that murmur bursts into full play at the commencement of the attack, being audible on admission, and on the 3d, 4th, 5th, 6th, or

7th days after the seizure; in others it is not audible until a period varying from the 8th to the 30th day, although there is unequivocal evidence that the inflammation in the left side of the heart was present before and at the time of admission. This evidence consists in the existence of a tricuspid murmur, or a prolonged first sound, or pain in the region of the heart or in the chest, with great or considerable general illness.

The inflammation of the valve cannot cause regurgitation until perfect adaptation is prevented by the formation of small prominences, covered with a deposit of fibrin upon the surfaces or lines of contact of the margins of the valve, or by the softening and yielding of its flaps.

In three of the cases tricuspid or mitral murmur became audible after admission, when the patient's illness increased. In ten other cases, however, it was the reverse, for in all of them the murmur came into play when the patient's health began to improve.

We are therefore, I conceive, warranted in assuming that in a considerable number of the cases, the active stage of the endocarditis is passing away at the time of the appearance of the murmur.

Progress of Cases of Endocarditis with a Mitral Murmur.—Cases with a mitral murmur from endocarditis affecting a valve previously healthy, may usually be readily distinguished from those in which the murmur is due to established disease of the mitral valve by the character, seat, and area of the murmur, its changes, duration, and transition, its cessation or establishment; by the size of the heart and the force, extent, and position of its impulse; and by the nature of the first and second sounds over the right ventricle, the pulmonary artery, the aorta and great arteries in the neck. The mitral murmur is always situated over the apex and body of the left ventricle, and the ventricular septum. The centre of the murmur and its point of greatest intensity and purity is usually just below the left nipple. Sometimes it is limited to this point, but in general it covers a larger area, spreading inwards towards the right ventricle, outwards and upwards towards the axilla and over the lung, and downwards over the stomach. This area is rarely extensive, being usually limited by a diameter of from two to three inches.

When the heart is high, owing to the elevation of the diaphragm, and when the left ventricle is exposed in consequence of the shrinking of the overlapping portion of the left lung, the murmur extends upwards towards the axilla, and even above the mamma, and a little outwards rather than downwards. The direction of the murmur upwards towards the axilla is peculiar to the mitral murmur of endocarditis, for when disease of the valve is established, the lungs expand downwards to an unusual extent, and so muffle or arrest the murmur in its course towards the axilla.

The extent of the area of the murmur depends much upon its character. A smooth, soft, bellows murmur, especially if it is rather feeble, is in general limited to the apex and left ventricle; so also is a weak, grave murmur. But when it is vibrating, loud and almost musical, and especially if a thrill is felt by the finger over the apex—then the area of the murmur is extensive. Sometimes, indeed, it is so all-pervading that it may be heard over the whole cage of the chest, front and back, and even upwards into the neck and downwards over the abdomen.

It is only in established mitral disease, or in very rare cases of endocarditis with extensive mischief to the valve, that we find this pervading vibrating murmur with perceptible thrill.

In cases of established mitral disease the murmur is usually audible to a greater or less extent over the region of the stomach, often coming quite down to its lower boundary. The vibration in the left ventricle, which rests immediately upon the stomach, the diaphragm alone interposing, awakens a corresponding vibration in the stomach, and as this takes place in a hollow sac, its tone is often metallic, and it thus sometimes imparts a musical character to the murmur at the apex.

In cases of endocarditis with mitral regurgitation, the murmur is often so feeble that it is limited to its birthplace, and is unable to generate corresponding vibrations in the adjoining organs. In these patients the murmur is inaudible over the stomach; but in other cases of endocarditis, according to the loudness and penetrating quality of the tone, the murmur makes itself heard over a greater or less portion of the stomach, at that part of it nearest to the apex of the heart.

The murmur was heard over the lower part of the back of the chest in only two of the fifty cases of endocarditis with mitral murmur of the first series, and in one of the twenty cases of the second series. In one of these cases the murmur was audible over the lower part of the back, the lungs being condensed, on the 4th day, but it was not again heard in that position. In another such case the murmur was heard over the back of the chest from the 27th to the 34th days after admission, but ceased to be so on the 36th; and in the third case the murmur was heard below the shoulder blades for the first time on the 18th, and for the last time on the 42d day. After that date the murmur was less loud, and its area was correspondingly lessened.

I have to add to these, one case of death with inflammation of the mitral valve; the anterior flap was softened and enlarged, its edge and that of the posterior flap were covered with lymph or fibrine, and the valve permitted extensive regurgitation through the mitral aperture. The patient, a young man previously in good health, had been ill a fortnight with acute rheumatism; when admitted, he had an anxious expression, hurried and difficult breathing, and sickness. A loud mitral murmur, beginning with a sharp shock and followed by the second sound, extended forwards almost to the sternum, where the heart sounds were healthy, and backwards to below both shoulder blades. From the 9th day to the 11th he raised phlegm tinted with blood, he was propped up in bed, and there was dulness and fine crepitation over the left lower lobe. On the 14th he sat forward in bed in great distress, breathing with difficulty. In the course of that day he died, and on dissection he presented the inflammation of the mitral valve and the extensive pulmonary apoplexy that were evidenced during life.

The patients usually lay flat in bed, their pain being increased by movement, and as the back was not examined, some of these might have presented a murmur over the lower lobes of the lungs behind; but when we regard the limited area over which the murmur was usually heard in front and at the side, it is evident that it could scarcely have been audible behind. I think it probable that three cases, in addition to those just named, may have been exceptions to this rule, and perhaps two others, for in them the murmur was loud, while in the first three it was vibrating in tone.

The mitral murmur at the time of its first appearance, or of its transition from prolongation of the first sound, is as a rule either weak and grave; or it is a soft, feeble, bellows murmur, and is therefore limited in area.

The mitral murmur invariably begins with an accent or shock, which corresponds with the shock of the impulse, and it generally ends with the second sound. It fills up, in fact, the space between the first and second sounds, that space being often lengthened, so as to admit of greater prolongation of the murmur, with the effect of altering the rhythm of the heart. Sometimes the murmur does not quite fill up this space, so that there is a distinct silent pause between the end of the murmur and the second sound. The presence of the accent or shock at the beginning of the first sound distinguishes an endocardial murmur from an exocardial or friction murmur.

The pressure test comes in to settle the difficulty of distinguishing one condition from the other. If the noise be endocardial, the sound may become louder from the closer application of the stethoscope, when pressed upon the walls of the chest; but the quality of the noise is unaltered, it is rhythmical with the heart sounds, it retains its accent or shock, it fills up the space between the first and second sounds, and it ends exactly with the second sound.

But if the noise be frictional, it usually loses its murmur-like tone when the pressure is made—and becomes rustling or grazing, grating or creaking in character; it extinguishes the first and second sounds of the heart, which were previously heard side by side, but not incorporated with the murmur; it brings out a double sound where there was but a single one before, a sound to-and-fro in character, or a noise not unlike that made by the sharpening of a scythe, with a single down-stroke during the beat of the heart, and a double up-stroke during its pause. Sometimes the mitral murmur is masked or confused at the apex by the coexistence of a vibrating systolic noise. The interposition of a piece of paper or cloth between the stethoscope and the surface of the chest annihilates this vibrating noise, and the mitral murmur is then heard with perfect purity and clearness. The interposition of the lung effects the same end—for this vibratory noise is heard only where the heart is in direct contact with the walls of the chest; and hence, when using the naked stethoscope, we meet with cases in which the murmur is more smooth and bellows-like just to the left of the apex or towards the axilla, than it is over the apex itself. For this effect, however, the layer of lung must be thin and the tone of the murmur must be penetrating. In cases of endocarditis, with mitral regurgitation, the murmur is often muffled by a rumble, or a comparatively feeble vibration. The interposed paper or the intervening lung extinguishes this vibrating noise, and brings a pure, soft, bellows murmur into play.

The changes that the mitral murmur of endocarditis undergoes during the progress of the case are remarkable, and they vary in almost every instance. These changes consist in alterations of its tone, loudness, and area; in its transition from a true murmur to prolongation of the first sound; in the substitution of a tricuspid for a mitral murmur, or the reverse, or the companionship of the two murmurs; in the suppression and reawakening of the murmur; and frequently in its final extinction, either directly or by passing again into prolongation of the first sound, which precedes the restoration of the healthy sounds of the heart.

In one-fourth of the cases (18 in 70) the mitral murmur was only heard on one occasion.

Of 50 cases, in all of which the mitral murmur was heard more than once, that murmur was of equal loudness during the successive observations in one-fifth (11); became gradually weaker in one-third (17), but in six of these it passed through a double oscillation and increased and lessened a second time; became gradually stronger in one-fifth of the cases (11), in one-half of which it again gradually declined; was suspended and then renewed for a time in one-fourth of the cases (12), when the murmur again faded away; and it sometimes yielded to the healthy sounds of the heart, and sometimes to prolongation of the first sound. In two instances, already included in the abstract just given, there was a double disappearance and reawakening of the mitral murmur, which in one of them met with final extinction, while in the other it became established.

The changes in the area of the murmur corresponded in a considerable degree to the changes in its loudness, the former widening as the latter increased, and narrowing as it diminished.

In the great majority of the cases, and especially in those in which the murmur disappeared, the tone of the murmur underwent but little change. It became progressively louder and feebler, more clear and more obscure in almost every instance, but it usually retained its original character.

The murmur was observed to be soft and smooth, approaching to the character of a bellows sound, in less than one-half of the first series of the cases of endocarditis with mitral regurgitation, and in less than one-third of the second series; the cases in each series in which the murmur was not characterized amounting to fully one-third of the whole.

In a small proportion of the first series and a large proportion of the second series of cases, the murmur was grave in character, being in some of them feeble, and in a few loud and almost vibrating.

Musical, sawing, and rasping murmurs formed but a small proportion of the total number of cases, and these were they that passed through a series of changes in tone and character.

One case, a youth, was a notable and rare instance of the variety of changes in tone through which the mitral murmur may pass. He had been ill a fortnight, and had suffered from pain in the heart. On admission he presented a tricuspid murmur. To this a loud mitral murmur was added on the 3d day, when he was very ill. On the 8th he was better, and from that day to the 15th the murmur was weak, soft, and smooth. On the 21st it was louder, and on the 29th it altogether changed its tone and became musical. After this, without apparent cause, it underwent two variations, having first the character of a sawing and then of a bellows sound. The tone of the murmur then again altered, and it became grave, and finally on the 52d day it had regained its lost musical character.

We must now answer the important practical questions suggested by these observations, what are the character and progress of the murmur when the attack tends to end in perfect restoration of the efficiency of the valve? and what, when it tends to become permanently incompetent, owing to the establishment of mitral disease?

The answer may be already almost gathered from what has gone before. When the murmur is permanently feeble, soft, and smooth, with an approach to, or even the formation of, a gentle bellows sound, or when it is feeble and grave, the complete restoration of the efficiency of the valve may be anticipated. In illustration of this statement we find that the murmur was feeble, soft, and approaching to a bellows sound in 14 of the 25 cases of the first series that ended in recovery of the valve; and in 4 of the 10 that left with a lessening murmur, the corresponding number in the two like classes of cases of the second series being 5 in 17, while of the 17 cases that ended in established valve disease out of a total of 71, the murmur was feeble in none, and was smooth or soft in 6, most of which presented a definite bellows murmur.

The feeble grave murmur was more frequently developed in the later than in the earlier series of cases, but in both its presence was almost always followed by the restoration of the function of the valve.

When the loudness of the murmur steadily diminished, or when it first rose and then fell, or when after disappearing it reappeared and again faded away, the integrity of the valve was generally regained.

When the murmur was loud, its area being extensive; when it presented a sharply-defined loud, bellows, musical, sawing, or rasping sound; when it was vibrating in tone; when it steadily increased in loudness, or only slightly rose and fell to rise and fall again, without a temporary disappearance; then valvular disease was, as a rule, though not invariably, permanently established. One patient, a nurse in the hospital, left with a loud mitral murmur, but after a time, when she resumed her work, the murmur had given place to healthy heart sounds.

Condition of the Heart and the Great Vessels in Cases of Endocarditis affecting the Mitral Valve.—In these cases there are, as I have already illustrated, many affections of the heart besides imperfection of the mitral valve with its attendant

murmur. When inflammation affects the great central cavity of the heart, the pivot of its action, the whole organ is involved, and every part of it becomes, in succession, modified in its action; and in the force, movement, and sounds by which it makes that action known.

Inflammation of the fibrous structure of the left side of the heart is as essentially a part of acute rheumatism, as is inflammation of the fibrous structure of the joints. The inflammation may commence in the heart at the same time that it commences in the limbs. It attacks that part of the heart that is working with the greatest force, just as it attacks those parts of the limbs that are subjected to the greatest labor. The increasing inflammation of the joints calls forth increasing force in the action of the left ventricle, and so stirs up and adds to the inflammation that may have already existed in that cavity from the commencement of the attack.

This inflammation of the ventricle, like the inflammation of every other organ, lessens the power of the muscular cavity to expel its contents, and to propel the blood round the vessels of the system. This imperfect transmission of blood to the system, the demand for which is increased by the inflammation in the limbs, causes distension of the left auricle, and impedes the transmission of the blood through the lungs. This induces distension of the pulmonary artery and its branches, with, as its effects, accentuation—or loudness and sharpness, or shock—of the second sound, with relative feebleness, or even absence, of the first sound over that artery; and distension of the right ventricle, with increase in the action of its walls and in the force and extent of its impulse.

We have, thus, two ventricles beating side by side, the left one, the seat of the inflammation, beating with lessened power; the right one, with increased force.

The increased fulness and force of the right ventricle tend, when they pass certain limits, to reverse the flow of a portion of the blood, and to send it from the right ventricle back into the right auricle; with the effect of relieving the distension of the arteries of the lungs, increasing the fulness of the veins of the system, and producing a tricuspid murmur.

After a time, the whole volume of the blood is diminished, and the proportion of its red corpuscles is lessened; and then appear as later and secondary effects, a murmur over the pulmonary artery, and sometimes a murmur over the aorta and its great branches—murmurs that are due to the lessening of the contents, and relaxation of the walls of those vessels.

Such murmurs in the great arteries appear, however, also in the early stages of the affection, in the aorta more frequently, owing evidently to the lessened power of the inflamed left ventricle, and the diminished supply of blood that is therefore sent into the aorta, the walls of which are thus relaxed; and in the pulmonary artery occasionally, for reasons that have yet to be ascertained.

The close study of the condition of the heart and great vessels generally throws more light upon the degree of the inflammation of the heart, and its effect on the vital powers of the organ, than does the simple observation of the mitral murmur.

I shall now rapidly review the conditions of the heart and great vessels as they presented themselves in the cases of endocarditis with incompetence of the mitral valve—that valve being previously in the virgin state and uninjured.

The Impulse of the Heart.—I find that I have taken notes of the state of the impulse in one half of the cases with mitral incompetence, or in 25 out of 50 of the first series, and 9 out of 20 of the second.

The beat of the heart was, as a rule, not extensive or strong. It showed itself rather in the higher than the lower cardiac intercostal spaces, being present in only one instance below the fifth space, less frequently in that space than in the fourth, and sometimes even in the third space. While the impulse at the apex was in general feeble or absent; that of the right ventricle, though rarely powerful, was usually somewhat increased in strength, being present in the third and fourth, and even the fifth spaces between the cartilages. This impulse of the right ventricle was not as a rule marked or strong, but it could be felt diffused over those spaces when the ball of the palm of the hand was applied over them, or when the fingers were pressed gently into the spaces.

In a few instances the action of the heart, and especially the impulse of the right ventricle, was strong and diffused or powerful, or even tumultuous and violent, soon after admission; and then the size of the heart, which was not in general notably affected, became enlarged, the chest over the cardiac region being more prominent than over the corresponding space on the right side.

In one or two patients the impulse presented a peculiar shock.

But the distinctive feature with regard to the impulse in a fair proportion of the cases was its variation during the successive periods of the disease. Thus, in one instance, it was feeble on the first day in the fourth space, very strong on the 3d day, moderate in strength in the fifth space on the 8th day, and in the third and fourth spaces on the 12th day. In another patient the impulse was felt in the second and third spaces on the 2d day, when

there was pain in the heart; on the 5th, the pain still continuing, the heart beat violently; from the 6th to the 18th the pulsation was strong in the second space, and from the 28th to the 34th it was diffused from the third to the fifth spaces. In this case mitral disease was established, and the gradual extension of the impulse of the right ventricle told with precision the story of the increasing valvular disease in the left ventricle.

The study of the impulse conveys the most important lesson in all cases of endocarditis. Its absence may tell of the want of vital power; and its excess in the right ventricle, while it is wanting in the left, shows lessened power from inflammation in the latter cavity, and consequent increased labor in the former. Its gradual increase in force, and enlargement in area, with persistence of mitral murmur towards the period of the termination of the attack of endocarditis, and after its cessation, mark advancing and established valvular disease; and its extent and force point out the amount of the back-flow of blood from the left ventricle into its auricle, and the obstacle to the onflow of blood through the lungs induced thereby. The impulse of the right ventricle is, in short, a measure of the extent of the injury to the mitral valve, and of the consequent resistance to the circulation through the lungs.

The impulse of the right ventricle was diffused and strong, extending out to the nipple, in a considerable proportion of the cases in which there was a tricuspid murmur.

In a few instances the impulse of the right ventricle was so high as to be present in the second space; but generally the pulsation felt in that space was due to the presence there of the distended pulmonary artery, when that pulsation was double, the second impulse being more smart and shock-like than the first. In these cases the pulmonary artery was distended, the first sound was feeble or absent, while the second was unusually loud and strong, penetrating the ear with a shock.

The apex beat is, in cases of endocarditis with mitral regurgitation, usually slight, sometimes absent—during the early period, before the mitral murmur is developed, owing to the weakened muscular power of the inflamed left ventricle; and —after the appearance of the murmur, owing to the relief afforded to the organ by the greater ease with which its surcharge of blood is sent backwards into the auricle than forwards into the aorta.

There are, however, certain exceptional cases of great interest, several of which have come under my observation, in which the left ventricle beats with great force, and unduly to the left.

In three of these cases there was extensive pulmonary apoplexy, or pneumonia of that type, in the lower portion of the left lung.

One was a youth, with hurried and difficult breathing, tinted phlegm, and dulness over the lower portion of the left lung, which was solid and lessened in size, owing to pulmonary apoplexy. The condensed and solidified lung shrank away from its natural position between the walls of the chest and the apex of the heart; and the apex was therefore completely exposed, beating with all its force upon the fifth space more than an inch beyond the left nipple. At that time there was no mitral murmur, but as soon as the lung began to recover itself, the murmur came into full play. When the lung again expanded, it covered the apex of the heart, and its beat was no longer perceptible. The whole heart in this case was displaced to the left; and its displacement was still greater in the sister case, in which the apex beat was situated three inches beyond the nipple line; the impulse of the right ventricle was placed to the left of the costal cartilages; and the double pulsation of the pulmonary artery, with a strong second shock, was present in the second space above the mamma.

A fourth case, when admitted, had pain in the region of the heart, and the apex beat was situated an inch and a half to the left of the nipple. Five days later the extreme limit of the impulse had shrunk one inch, being seated half an inch to the left of the nipple.

Accentuation of the Second Sound, with Silence, Feebleness, or Prolongation of the First Sound over the Pulmonary Artery.—Accentuation of the second sound over the pulmonary artery, in the left second space, is a well-established sign attendant upon mitral regurgitation, and it may be present in every degree.

The second sound may be more or less loud and sharp or ringing—or it may penetrate and strike the ear with a loud shock; when a double impulse is to be felt over the pulmonary artery, the first being gentle and gradual, while the second gives to the hand a smart shock.

This increase in loudness and sharpness of the second sound is due to distension of the pulmonary artery, owing to the difficulty with which the blood travels through the vessels of the lungs.

Whenever the blood thus accumulates in the lungs, whatever be the cause, the same effect is induced. In cases of phthisis, and notably when there is hemorrhage from the lung and shrinking of its tissue, the pulmonary artery, enlarged and tense, displaces the lung superficial to it, and presses against the second space; where there is a double impulse

the first gentle, the second felt and heard as a shock. In bronchitis, emphysema and pneumonia, there is the same distension of the pulmonary artery, but greater in degree. The interposition of the lung, enlarged owing to the disease, screens the pulmonary artery from the hand and the ear, so that over it the second sound is often not unduly loud; but it is so in some instances over the right ventricle.

Whenever the tension of the pulmonary artery is thus so great as to cause a strong and loud shock with the second sound, the first sound is either almost silent, or feeble, or faintly prolonged.

When the blood is sent into a tight and full artery, it makes but little, often no sound, either in the shape of shock or murmur; but the second sound caused by the smart and strong reflux of the blood upon the walls and closed valves of the artery, makes a loud, sometimes a ringing or metallic sound. The same occurs in the aorta when it is enlarged and rendered tense, owing to the difficulty with which the blood leaves the arterial system in advanced cases of contracted granular kidney. When you listen over the aorta a single sound is often heard, a loud ringing metallic second sound, the first being almost or absolutely silent. Sometimes in these cases the artery is so large and tense that it presses against the second right intercostal space, producing there a double pulsation, the first gentle and gradual, the second smart and with a shock.

I find that I have described the second sound as being loud or sharp or ringing in about one-half of the 50 cases of the first series and 9 of the 20 of the second series of cases of endocarditis with mitral murmur, and in 5 of 13 of those of the first series with an uncomplicated tricuspid murmur. This does not of course include all of this class.

It was noticed that the second sound was sharp or loud in the early period in a large proportion of the cases in which that sign was observed, or in 13 out of 25 of the first series, and 7 out of 9 of the second series.

In all but six of the patients in whom it was noticed that the second sound was intensified, it continued to be loud down to a late period, to the time in fact of approaching recovery.

Loudness of the second sound may be associated with each of the signs, singly or in combination, that are habitually found in cases of endocarditis with inflammation of the mitral valve. It accompanied a mitral murmur, either alone or in combination with a tricuspid murmur in 22 of the cases; in about 15 cases it was allied with prolongation of the first sound over the left and sometimes the right ventricle; and in 8 cases it was joined to tricuspid regurgitation, which was, however, combined with other important signs in every instance but one. The first sound of the pulmonary artery was affected, when the second sound over that artery was loud or sharp, on ten occasions, in different patients: in 4 of these there was a pulmonic murmur, in 4 the first sound was prolonged, being generally free from shock, and in 2 it was silent or scarcely audible.

These numbers, however, taken by themselves give a very inadequate idea of the relation of the first to the second sound of the pulmonary artery in cases when that second sound is intensified. Thus, as we have just seen, pulmonic murmur was followed by a sharp second sound in four instances, but there were altogether 32 cases in which a pulmonic murmur was heard, and in only four of them was it stated that the second sound was thus affected at the time when the pulmonic murmur was audible. In one of the cases in which there was a pulmonic murmur on admission, the second sound was free from accent; while on the 3d when the pulmonic murmur had disappeared, that sound was slightly accentuated over the pulmonary artery. Again, in only two of the cases is it noted that the first sound of the pulmonary artery was silent or scarcely audible when the second sound was loud. Since, however, my attention has been drawn to the relation of the first to the second sound of the pulmonary artery, in every instance that I have observed accentuation of the second sound, especially with, but even without shock, the first sound has been either very feeble, being occasionally prolonged, or almost or even quite silent. This condition was signally marked in a case of chorea under my care in the hospital, a boy, who on admission, presented no mitral or other murmur over the heart. After gaining ground steadily he became rather worse, his temperature rose, he had pain in his chest, and the second sound was loud, the first feeble over the pulmonary artery; and six days later a mitral murmur came into play. At the same time the right ventricle, previously quiet, beat with great force, and a strong shock was felt over the pulmonary artery with the second sound. On listening over that vessel, a loud second sound penetrated the ear and struck it as it were with a shock, and the first sound was silent, the second sound being alone audible to all who listened. After a short time he became very ill, and for two days he passed his evacuations involuntarily in bed. He kept both hands flexed on his wrists, and his fingers on his hands. He soon began to improve, and gradually as this boy gained strength, speech, power to move, and freedom from irregular

movements; and as his lungs enlarged, the mitral murmur being still audible, the second sound though still loud lost its shock, the second impulse ceased to be felt over the pulmonary artery, and the first sound, though feeble, became more and more audible.

In a fair proportion of the cases in which the second sound was sharp and loud at the early period of the disease, that sound retained its character unaltered through all the surrounding changes in the sounds of the heart. Let us take one case. At first there was a tricuspid murmur, the second sound being sharp; on the 6th day there was a mitral murmur, and the second sound was loud; next day the murmur was less marked, but the second sound was still loud; and on the 11th the murmur had given place to prolongation of the first sound over the right ventricle, and yet the second sound still remained loud. In another instance on the 9th day there was an obscure mitral murmur, on the 16th there was mitral, tricuspid and direct aortic murmurs, on the 19th these had all vanished, and on the 23d the tricuspid and direct aortic murmur had returned; and yet on each occasion there was the same sharp second sound over the pulmonary artery. I could give several instances of this kind and would refer to four cases. In these instances the sharp second sound went on drumming, like the tomtom in the streets, whatever was the variety of the surrounding noise, or even when there was freedom from murmur or prolongation of the first sound.

The intensified second sound is, however, by no means always so unvarying in its note. Thus, in one very interesting case on the 11th day the second sound was very loud over the pulmonary artery, the first being scarcely audible; on the 34th both sounds were loud over the ventricles, the second being very loud; and next day all the sounds were natural.

I must refer to one other case, in which on admission the first sound was faint, the second loud over the pulmonary artery, the first sound being prolonged over the ventricles; on the 13th day the two sounds were equal over the artery and there was a feeble murmur at the apex; on the 27th the second sound was again louder than the first; and on the 40th a singular change took place, the first sound being louder than the second over the pulmonary artery—while over the aorta it was the reverse, and on the 50th day the natural standard was regained, the second sound being louder than the first.

The close study of the second sound and of its relation to the first over the pulmonary artery, is of practical importance in cases of endocarditis affecting the mitral valve. It may foretell the coming murmur in the early, and betray the recently extinct murmur in the later, period of the disease; and during its progress, it points by the degree and force of its accent to the amount of the resistance to the pulmonary circulation, the intensity of the internal inflammation of the ventricle, and the extent to which the function of the ventricle is impaired. It is, in short, a tell-tale sound pointing to the agency in the central cavity of the heart which gives it birth. The intensified second sound of the pulmonary artery, or that of the aorta, is associated as we have seen with a corresponding feebleness, or even silence, of the first sound of each of the vessels respectively. The observation of the one sound demands a corresponding observation of the other sound. When the artery is distended, it enlarges, lengthens, and advances, and comes gradually into contact with the second intercostal space, displacing the intervening lung from before it. You can then feel the double pulsation of the great artery; the first movement is gentle, gradual, barely perceptible to the touch; the second strikes the walls of the chest and the applied hand with a sudden smart shock or tap. When you listen to it the ear takes in the same effect through another sense; the first sound is in extreme cases silent, or is soft and gentle, feeble and perhaps somewhat prolonged; while the second penetrates and strikes the ear with a loud shock, often ringing and metallic. Over the pulmonary artery, as I have just said, that subdued sound or even silence, and this shock, betoken tension of the artery, and obstacle to the flow of blood through the vessels of the lungs; whether that obstacle be caused by a back flow, due to inflammation or disease, with incompetence, of the mitral valve; or directly to disease of the lung itself, whether from phthisis, contracted lung, pneumonic bronchitis, or emphysema; the shock being in these last cases shielded from the hand and muffled to the ear by the interposition of a couch of lung, thickened by the undue expansion of the air cells induced by the disease.

When the aorta is thus distended, pushing aside the lungs, beating with a double pulsation upon the second right intercostal space, over the ascending aorta, the first gentle and gradual, the second, a smart shock, the first feeble or even silent, the second, a loud ringing, metallic shock, you know that the blood forces its way with difficulty through the fine vessels of the system, and that the cause of this is the contamination of the blood, induced by advanced granular contraction of the kidney.

Two conditions are needed for the production of this double effect, one, that just spoken of, the obstacle to the onflow

of the blood; the other, the force with which the pulsating ventricle sends its blood into the artery. Lessen that force, and the supply of blood is lessened, the proportion of blood in the vessels and the power to pass it on is brought more into equipoise; the tension of the blood being relieved, the first sound becomes again audible, and the shock of the second sound is subdued, so that it becomes merely unduly sharp or loud.

Additional observations are wanted on this important practical point of the relative loudness of the first and second sounds over the pulmonary artery and aorta; combined with information as to the poisoning and accumulation of the blood, structural change in the walls of the arteries, and vital power. The two sounds must be listened to, and their relative intensity noted, which I do by the ready method of figures of varying size written on a diagram of the body on which the outline of the ribs is traced. The size of each figure denotes the relative intensity and actual loudness, judged of by the ear, of the two sounds. When the first sound is silent, and the second is loud and with a shock, I mark it thus, $^{0}/_{2}$; when two sounds are equal, thus, $1/2$; when the first is louder than the second, thus, $1/_{2}$; and when the second is louder than the first, thus, $^{1}/2$. Every shade can be thus rendered. Combined sphygmographic and cardiographic tracings, some of which I have made, in these cases, will give positive and scientific accuracy to our knowledge.

Doubling of the Second Sound.—In two of the cases of endocarditis with mitral murmur, there was doubling of the second sound. One of these came in with doubling of the first sound, or almost a murmur at the apex, on the 4th day a peculiar plunging first sound, with scarcely any second sound, appeared over the ventricles. On the 6th day there was doubling of the second sound. On the 8th day mitral and pulmonic murmur appeared, followed by a tricuspid murmur, and on the 10th these murmurs had all vanished. In the other case the doubling of the second sound appeared late and was very tenacious. There was a mitral murmur up to and on the 23d day, when the second sound was prolonged over the pulmonary artery. On the next day there was doubling of the second sound over that artery. The second second sound was louder than the first—and this proved that the later sound was the pulmonic, the earlier the aortic sound. In this instance the doubling of the second sound, which lasted to the 60th day, disappearing on the 69th, was due, I consider, to the longer time occupied by the right ventricle than the left in emptying itself, owing to the resistance to the flow of blood through the lungs.

Pulmonic Murmur.—A systolic murmur over the pulmonary artery, at the second left space, was heard in a considerable number of the cases of endocarditis with mitral murmur. This number amounted to one-third of the first series, or seventeen in fifty-two, and to one-half of the second series, or ten in twenty. This murmur was present also in one-third of those cases of endocarditis affecting the left side of the heart, in which there was tricuspid, but not mitral, murmur. In more than one-half of those cases the pulmonic murmur appeared towards the close of the attack, when all the acute symptoms had gone by, when the period of convalescence was approaching or established, when the patient was pale and thin, having lost a large proportion of the red corpuscles from the blood, and was weak from the exhausting nature of the disease. In nearly one-half of the remaining cases this murmur appeared at the middle period, and in one in four of the whole number it was audible soon after the admission of the patient.

The murmur almost always occupied a well-defined limited area at the edge of the sternum in the second space, just over the pulmonary artery. It never extended as far as the right edge of the sternum, but it could be heard very feebly in the first space, and occasionally in the third. When the position of the pulmonary artery was unusually low, the murmur moved downwards, being then heard strongly over the third space, and feebly over the second and fourth spaces.

The pulmonic murmur rarely presents a smooth soft bellows sound, but is usually grave and superficial, without however being large in character or very loud. The murmur appeared as a peculiar scratching noise in one-half of the cases, or 4 out of 8, in which the sign appeared soon after admission, and besides these in one on the 8th and in another on the 21st day. The scratching nature of the sound when I first observed it (I found it noticed in one case as early as the year 1852) was very puzzling. It strongly suggested friction sound. But it differed in these respects: it was always systolic, being never to-and-fro; pressure sometimes highly intensified, but never altered it in tone; it clung to one spot; and it gradually disappeared without passing into a wide-spread double friction sound. Its noise was exactly like that made by scratching slowly and gently with a pin on a deal table.

The cause of the pulmonic murmur is exactly the same as that of the aortic "anæmic" murmur, which is audible only during the systole. It is due to the

blood being very thin and lessened in quantity, and propelled into the vessel when its walls are relaxed, with undue force, by the ventricle.

When the pulmonary artery is flaccid, its contents have free room to vibrate as they move onwards in the current of the circulation, and therefore pulmonic murmur is engendered. The pulmonic murmur thus indicates that there is relaxation of the pulmonary artery, or a condition the opposite to that of tension of the artery. The second sound following the murmur may be loud, but it is usually feeble. It is loud if, during and towards the end of the contraction of the right ventricle, the pulmonary artery becomes tense; its walls then recoil with force upon their contents and propel them with equal pressure in two directions, forwards into the vessels, and backwards upon the ascending pulmonary artery, its sinuses, and valve, where the back-stream strikes with a sudden shock, the shock of the loud second sound. The second sound is, on the other hand, feeble if the flaccid artery does not become distended during the systole; when the recoil of the walls is therefore weak, and when the back-wave breaks with only moderate force upon the roots of the artery.

Silence or feebleness of the first sound is the opposite in character and cause to pulmonic murmur. If the artery is distended when the ventricle begins to contract, the column of blood moves steadily forwards, the walls of the vessel and its contents are not thrown into vibration, and the first sound is either absent or feeble. The extreme tension of the pulmonary artery thus induced, leads, when the blood has ceased to enter it, to the recoil of its walls with excessive force upon their contents, which are driven with a strong back-stroke or shock upon the walls, sinuses, and valve of the artery. When the lung is displaced from before the pulmonary artery, thus distended, this shock is felt by the hand and heard striking against the ear with a loud metallic sound.

Pulmonic murmur, as we have just seen, came into play most frequently when the disease was passing away. It was therefore rarely, or only once or twice, associated with a mitral murmur when at its zenith, and uncomplicated with other murmurs. In fully one-half of the cases (13 in 24) it accompanied prolongation of the first sound, or a feeble mitral murmur; in nearly one-half of them (9) it appeared with a conjoint mitral and tricuspid murmur; and in one-fourth with a simple tricuspid murmur, a companion sign that was therefore present in three-fourths of the cases. In one-fourth of the cases (6) it was traced side by side with an anæmic murmur over the aorta or carotid artery; and thrice it was unaccompanied by any murmur. Nearly all these instances point to a state in which the tension of the pulmonary vessels was either not yet established or was passing away.

A pulmonic murmur was audible in a large proportion (or 5 in 13) of those cases that I have classed as being probably affected with endocarditis. In all of these cases there was prolongation of the first sound. In three of them it was noticed soon after admission, and in the two others at a late period of the illness.

A pulmonic murmur was heard in a small proportion of the cases in which endocarditis was either threatened or only transient, amounting to 7 in 63 of the first series, and 2 in 22 of the second, or one-tenth of the cases. In all of these but one it appeared at a late period, when the intensity of the disease was passing away.

Pulmonic murmur is not then a direct sign of endocarditis. Its presence, however, in the early period of acute rheumatism usually points to endocarditis, and to the actual or approaching presence of a mitral or tricuspid murmur.

Its existence at a late period in a case of endocarditis generally points to relief in the severity of the disease, to the cessation of the inflammation of the heart, to a definite removal of the tension of the pulmonary artery, due to congestion in the lungs, and to the establishment, for a time, of the opposite state of that vessel, its walls being relaxed and the quantity of its blood diminished.

The pulmonic murmur, then, while it is a sign threatening inflammation of the interior of the left side of the heart in the early stage of acute rheumatism, is a sign of the passing away of endocarditis when it appears at a time when that affection has been established. Pulmonic murmur never becomes permanent. It generally diminishes rapidly when the patient leaves the bed, and gains color and strength, and in the convalescent patient it is often inaudible when standing or after walking, when it may be still heard if the patient is lying down.

I have heard the pulmonic murmur in several cases of enteric fever, when it indicates the condition of which I have just spoken, or relaxation of the pulmonary artery.

The pulmonic murmur usually, I believe, tends to become less vibrating and more feeble during the progress of the systole, when the artery is becoming less relaxed, and to die out at the end of the systole when the vessel is becoming tense, and the stream of blood is being gradually brought to a stand-still.

Tricuspid Murmur in Cases of Endocarditis with a Mitral Murmur.—I have already illustrated this sign when I described tricuspid murmur in cases of endocarditis of the left side of the heart without mitral murmur. I refer to that part at pages 622–625, and shall here therefore only state generally the conditions under which this murmur is found.

A tricuspid murmur is not, as we have already seen, a sign of inflammation of the right side of the heart, and of the tricuspid valve; but of inflammation of the left side of the organ and of the mitral valve. When the left ventricle is weakened by that inflammation, it sends less blood into the vessels of the system, and an undue amount of blood therefore accumulates in the vessels of the lungs. The pulmonary artery is over-filled, and the left ventricle is distended. The "safety-valve" function of the tricuspid valve is then brought into play, regurgitation takes place, and by throwing a portion of the blood backwards upon the veins of the system, it lessens the pressure of the blood forwards upon the arteries of the lungs.

Tricuspid regurgitation, then, while it declares the presence of inflammation of the left ventricle and the mitral valve, relieves the congestion in the lungs, which is one of the worst effects of that inflammation.

A tricuspid murmur is present in nearly one-half of the cases of endocarditis with mitral murmur. A tricuspid murmur may precede a mitral murmur, accompany it, alternate with it, or waken up after it has disappeared. A tricuspid murmur, then, is a friendly sign—it warns you of inflammation elsewhere, and relieves the ill effects of that inflammation. It is a danger signal, and a break, lessening the mischief.

Aortic Systolic Murmur (Anæmic).—A direct aortic murmur was noticed in twelve of the seventy cases of endocarditis with mitral murmur, and there were others in which its presence was doubtful. This murmur appeared in the early period of the disease in eight of the cases, and in the later period in four. In three of the patients in whom the murmur appeared early, it lived through the whole of the attack; and in one other of them, after vanishing for a time, it again appeared when the patient was recovering.

In all the cases but one, the aortic murmur was associated with conjoint mitral and tricuspid murmurs, and in fully one-half of them, seven, the aortic was coupled with a pulmonic murmur, usually at a late period of the disease. These twin murmurs, the aortic and pulmonic, are due to the same cause, a deficient supply of blood in the great arteries, which are therefore imperfectly filled. Their walls are consequently flaccid, and their contents have free room to vibrate as they move onwards in the current of the circulation.

The direct aortic murmur is much less frequent than the pulmonic murmur in cases of mitral endocarditis. But the aortic murmur appears early in the attack much more frequently in proportion than the pulmonic murmur. The reason of this would appear to be that in the early stage the inflamed left ventricle sends its blood with insufficient force and volume into the aorta, and vibrations with their consequent murmur therefore ensue. At a later period, the lessened volume of the blood circulating through the body, and the diminution of its red particles, lead to the formation of the murmur.

The question is an interesting one, and is not easy to answer, why the pulmonic murmur is so much more frequent than the aortic at the later period of the affection? May it not arise from two influences? (1) The increased size to which the pulmonary artery has attained during the period of its tension, when the disease was approaching to and at its acme; and (2) the greater relative influence that the diminished supply of blood has upon the comparatively restricted area of the arteries of the lungs, when compared with the much larger area of those of the body?

Prolongation of the First Sound occurring at a late Period in Cases of Endocarditis with Mitral Regurgitation.—We have already seen that in a considerable proportion of those cases of endocarditis that are admitted before the appearance of a mitral murmur, that murmur is preceded by prolongation of the first sound.

Prolongation of the first sound (as we have seen at page 628) may develop into a tricuspid or mitral murmur, and when the murmur fades away, it may give place to a renewal of the prolongation of the first sound. This was precisely what occurred in one case, a patient in whom, when admitted, the first sound was prolonged; on the 10th day a tricuspid murmur was audible, which was replaced on the 19th by prolongation of the first sound. In another case the sounds were at first healthy, but the first sound was prolonged at the apex on the 4th day, a tricuspid murmur appeared on the 6th, which yielded on the 9th to prolongation of the first sound over the right ventricle, and on the 48th day the sounds were again healthy. In five cases with mitral murmur a similar chain of transformations took place. In one of these a mitral murmur, which appeared on the 5th day, superseded prolongation of the first sound at the apex; that murmur became weaker on the 10th, and was joined on the 12th by three other grave feeble murmurs, a

tricuspid, a pulmonic and an aortic. On that day the murmurs were audible when the patient lay down—but they passed into prolongation of the first sound when he stood up—and on the 20th day that prolongation was only audible when he lay down, the sounds being healthy when he stood up; owing evidently to the greater amount of blood that was then demanded by the body and the lungs, and was consequently supplied to the aorta and pulmonary artery.

In a few of the patients the murmur during the illness yielded for a time to prolongation of the first sound, and then reappeared. One case, a female patient, was a notable instance of the variety of transformation sounds that may occur in this disease. When admitted, she presented a mitral or tricuspid murmur; on the 3d day the first sound was prolonged, and on the 6th the sounds were natural. But on the evening of that day a mitral murmur set in which remained for several days, being joined by other murmurs. On the 14th those had vanished, the first sound being prolonged. On the 16th a tricuspid murmur appeared, which was exchanged for a mitral murmur on the 27th, which from that date became permanently established.

In many instances the position of the prolongation of the first sound is not specified, but when it is, the situation of the murmur as a rule corresponded with that of the prolongation of the first sound out of which it grew and into which it faded —both being present at the apex when the murmur was mitral, and over the right ventricle when it was tricuspid.

The passage from murmur to prolongation and the reverse was often very gradual; they often each glided insensibly into the other. The prolongation was often murmur-like in character, and the murmur was often so obscure as to be quite as fitly ranked with prolongation.

In several of the patients, prolongation of the first sound over one ventricle was accompanied by a murmur over the other. Thus in three cases a tricuspid murmur was associated with prolongation of the first sound at the apex; and in another instance a mitral murmur was coupled with prolongation over the right ventricle. One case is an example of both kinds in succession. At the time of admission, when the patient was very ill, the sounds were loud, the first being sharp. From the 2d day to the 7th there was a tricuspid murmur with prolongation of the first sound at the apex; and on the 21st there was a double exchange, a mitral murmur being joined by prolongation of the first sound over the right ventricle. Sometimes there was a double prolongation of the first sound, at the apex, and over the right ventricle, as occurred in four cases. I have noticed this coupling of the sign only in cases observed at a later period, and I am certain that it occurs much more frequently than my earlier notes would indicate. In a large proportion of the cases the murmur passed into prolongation of the first sound towards the period of convalescence. This was noticed in six of the thirteen cases of endocarditis with tricuspid murmur; in sixteen of the forty-one cases of endocarditis with mitral murmur of the first series, in one of which that sign gave place finally to a permanent mitral murmur; and in twelve of the twenty of the same class of the second series.

Prolongation of the first sound is the first whisper of an approaching murmur, the last of a departing one. It is a sign of coming danger, and it usually betokens, towards the conclusion, a favorable issue.

Prolongation of the first sound, or an obscure murmur, was heard in seven of the seventy-nine cases of the first series, and in none of the fourteen cases of the second series that were classed as having had no endocarditis.

Of those patients in whom endocarditis was threatened or probable, the first sound was prolonged, or there was a doubtful murmur, in forty-three of the seventy-six cases of the first series, and eighteen of the twenty-six of the second series. In more than one-half of the cases thus affected there was great general illness (35 in 61), and of these in fifteen there was pain in the region of the heart.

We must look then upon prolongation of the first sound as a sign of actual, or probable, or threatened, inflammation of the heart; whether we regard its presence in those cases of pronounced endocarditis with a mitral or a tricuspid murmur, or in those of probable or threatened endocarditis, in which the murmur was not declared.

The duration, the degree, and the progress of endocarditis is not to be estimated by the presence of a mitral murmur alone, but by the effects also of the inflammation upon the body, the lungs, and the heart. The face is anxious and dusky; there is sometimes pain in the heart; the breathing is quickened and oppressed; the impulse of the left ventricle is weak, while that of the right is unduly strong; the circulation through the lungs is impeded; the pulmonary artery is distended, its first sound is silent or feeble, and its second is accentuated; a tricuspid murmur is often present, sometimes alone, sometimes conjointly with a mitral murmur; prolongation of the first sound precedes and follows the mitral and tricuspid murmurs; and anæmic murmurs are often heard both over the pulmonary artery and the aorta, during the early and also

the late period of the disease, but rarely during its acme; the pulmonic murmur being more frequent at the period of convalescence, the aortic murmur during the early stage of the disease.

III.—Cases of Rheumatic Endocarditis with Aortic Regurgitation. (1) Not Accompanied by Mitral Murmur. (2) Accompanied by Mitral Regurgitation.

(1) *Aortic Regurgitation, not accompanied by Mitral Regurgitation.*—Incompetence of the aortic valve is much less frequent in rheumatic endocarditis than incompetence of the mitral valve. There was a diastolic aortic murmur not accompanied by a mitral murmur in ten; and there was a mitral murmur without a diastolic murmur in fifty of the first series of cases—while there was mitral regurgitation in twenty, and aortic regurgitation in none of the later series of cases. This brings up the cases of mitral in relation to aortic regurgitation to the proportion of seventy of the former to ten of the latter. Besides these, eight of the first series and one of the second presented both mitral and aortic incompetence. This makes the total number of cases in which there was aortic regurgitation eighteen, and the total number in which there was mitral regurgitation seventy-nine.

In more than one-half of the cases of endocarditis with aortic regurgitation, there was no mitral murmur (10 in 18). The mind naturally infers that in these patients the inflammation was limited to the aortic valve, and did not extend to the mitral. The close examination of the cases, however, leads, I consider, to the conclusion that in all of them there was inflammation of both the mitral and the aortic valves.

A mitral murmur appeared in one of the ten cases for a single day, and was not again heard. That was the only case in which this, the central and immediate sign of mitral endocarditis, was noticed. In the others, however, the more important secondary signs of inflammation of the interior of the left ventricle were present.

In five of the cases a tricuspid murmur was audible over the right ventricle. In three of these that murmur was heard before the murmur of aortic regurgitation came into play; in one, the two murmurs were present on the day of admission; and in the fifth case, the tricuspid murmur appeared a week later than the aortic, but the aortic murmur was preceded by prolongation of the first sound, which was present on the day of admission.

The first sound was prolonged over one or both of the ventricles in six of the cases; in three of which there was, and in three there was not a tricuspid murmur. In two of the three in which there was no tricuspid murmur, prolongation of the first sound preceded the aortic murmur.

Thus eight of the ten cases of endocarditis with aortic incompetence, without mitral murmur, presented either a tricuspid murmur, or prolongation of the first sound over the ventricles, or both signs. In six of them, one or other of those signs preceded the appearance of the aortic incompetence; in one other the patient came in with both aortic and tricuspid regurgitation murmurs; and in the remaining one only did the aortic murmur precede by three days the prolongation of the first sound. The ninth case was admitted with aortic regurgitation, and he suffered from pain in the region of the heart.

The tenth case, a female patient, was an anomalous and doubtful one. She was very ill when admitted, when she had pain in the left side, and the sounds of her heart were rough. On the twelfth day a soft double murmur was audible in the second left space which was probably due to aortic incompetence.

(2) *Cases of Rheumatic Endocarditis with Aortic Regurgitation, accompanied by Mitral Regurgitation.*—In eight cases mitral and aortic incompetence were combined, and in six of these the mitral murmur preceded the aortic. Both murmurs were present on admission in one of the two remaining cases, and they appeared together in the other one on the seventh day after admission.

These illustrations, and the considerations that I have just advanced, appear to me to render it conclusive, that the inflammation always commences in the interior of the left cavities, affecting primarily the mitral valve; and that it extends at a later period, and in a limited number of cases to the aortic valve.

These facts lead us to expect that in cases of endocarditis the aortic diastolic murmur appears at a later period than the mitral murmur. In two only of the cases was the aortic murmur heard on the day of admission. One of these had been ill a week, and that was the earliest date of the appearance of the murmur. In three of the patients the aortic murmur appeared from the 7th to the 10th days, in one-fourth of them (5) from the 10th to the 15th days, and in more than one-half (10) from the 22d to the 88th days, after the beginning of the attack of acute rheumatism.

We have seen that aortic regurgitation is preceded with rare exceptions by a mitral or tricuspid murmur, or a prolonged first sound over the ventricle, or in other words by evidence, immediate or

secondary, of inflammation of the left cavities of the heart and the mitral valve.

In a small proportion of the cases, amounting to three in eighteen, the murmur of aortic regurgitation was preceded by prolongation of the second sound over the aorta or the carotid artery. This prolongation of the second sound over the aorta before the appearance of the aortic diastolic murmur, has evidently the same relation to that murmur that prolongation of the first sound has to a mitral or tricuspid murmur. It is a transition sound, and is the immediate herald of the coming complete murmur of regurgitation.

An anæmic systolic aortic murmur sometimes precedes the appearance of the diastolic murmur made by aortic regurgitation; but it more often comes at the same time or later, when the two sounds combine to form a true double murmur. This double murmur was present in eleven of the eighteen cases of endocarditis with aortic regurgitation, in four of which the systolic murmur was audible before the diastolic murmur, in five they appeared together, and in two the latter murmur came first into play.

The situation of the aortic diastolic murmur of endocarditis is ruled by the position of the aperture of the aorta, and the direction of the back current flowing through it into the left ventricle.

The murmur is more loud and intense to the left of the middle of the sternum, just over the root of the aorta, than elsewhere. It takes there a direction downwards and to the left, and is audible to the left of the lower three-fifths of the sternum, becoming feebler as it descends, and is lost usually before it reaches the limit of the lower end of the sternum. The murmur was heard also in five cases as high as the lower end of the manubrium, and indeed over that portion of the sternum. In rare cases it is audible at the apex.

In my cases of endocarditis with aortic regurgitation, the most frequent position of the murmur was to the left of the lower portion of the sternum, a space that extended from the middle of the sternum to its lower end, and from the third left costal cartilage to the sixth; a space that is immediately in front of the right ventricle, where it is denuded of lung. The murmur was audible over this space in thirteen of the eighteen cases. In four others it was heard at or to the left of the mid-sternum, a position that is included in the space noted as being to the left of the lower sternum, and which is, therefore, the position at which the aortic diastolic murmur of endocarditis is heard most frequently and with the greatest intensity.

In two of the cases the murmur was audible just below, and in one of these over the manubrium. In none of them is it stated that the murmur was heard to the right of the upper portion of the sternum, a position in which the direct aortic murmur was audible in five of the cases. In the exceptional and doubtful case, the double murmur was restricted to the left second space. There was certainly no regurgitation in that case from the pulmonary artery into the right ventricle, and we are therefore, I think, entitled to consider that it was, like the others, a case of aortic endocarditis, with regurgitation. In a patient under my care in St. Mary's Hospital an exquisite musical plaintive diastolic murmur sprang up at a late period just over and below the lower portion of the manubrium, and over the pulmonary artery in the second space, and was limited to that region. In this case the position of the heart was high and the murmur was heard over a correspondingly high and limited area.

In four, and in four only, of the cases the diastolic murmur was heard at the apex.

When we consider that the current of blood flows from the aorta back into the left ventricle, it seems natural to expect that the murmur of aortic regurgitation should be heard over the left ventricle, into which the stream of blood falls; and not over the right ventricle, which, with its double wall and its full contents, is interposed between the stream of return-blood and the ear. But the fact is the reverse of this. The murmur is always heard in front of the heart, over the right ventricle, and rarely over the left ventricle, to the left of the septum.

After a little reflection, the reason of this curious deviation of the direction of the sound becomes apparent.

When the aortic valve is incompetent, two streams pour side by side into the left ventricle. One of these comes down, in a large volume of blood, from the left auricle, through the mitral orifice, into the left ventricle; and this large living stream of blood occupies and completely fills the whole of the outer portion of that cavity, which is the part that is in contact with the walls of the chest at and beyond the septum, and at the apex. The other stream is that of regurgitation from the aorta. It is a small and an active stream which plays downwards into the innermost portion of the cavity, or that portion of it which lies immediately behind the right ventricle. The large living stream of blood that pours down from the left auricle into the outer part of the left ventricle, through the mitral orifice, cuts off the inner, deeper, and finer current flowing back from the aorta into the left cavity, and so silences it. This answers the question, why do you not hear the murmur of aortic regurgitation at the

apex and over the left ventricle? The answer, however, to the second question is still to seek, why do we hear that murmur through the right ventricle, with its double walls and its large volume of blood entering freely through the tricuspid orifice? When thinking out the answer to this question, we must steadily come back upon the facts as to the position of the aortic orifice, the nature of that part of the ventricle immediately in front of the aortic aperture, the direction of the return-current of blood into the right ventricle, the point of the greatest intensity of the murmur, and the bearing of the fading away of the murmur.

The aortic valve lies behind the middle of the sternum, at its left edge; in front of it is the conus arteriosus, which is the shallowest part of the right ventricle, its cavity being there wider than it is deep, and its posterior wall being there pushed forwards by the left ventricle and the root of the aorta and the aortic orifice through which this back-current flows; the walls of the conus arteriosus are here thin, especially the front wall; the blood contained in this part of the right ventricle is not in lively motion during the diastole, for it is above the current of blood from the right auricle into the right ventricle; and that current pours across from right to left, low down into the larger, deeper, and lower portion of the ventricle behind the lower part of the sternum and upper part of the ensiform cartilage. The murmur rapidly loses loudness and intensity as it approaches the lower part of the sternum in front of the tricuspid current, and it is lost before we reach the top of the ensiform cartilage.

We now see that the murmur of aortic regurgitation has a shorter way to travel, and passes through a less troubled blood, by passing straight through the arterial cone of the right ventricle, immediately in front of the aortic aperture, than it would if it were to force its way through the large and deep living current of blood that flows from the left auricle, through the mitral orifice, into the left ventricle, and that completely occupies the body and outer or left side of that cavity, where it presents itself at and beyond the septum and at the apex.

When active endocarditis passes away and leaves the aortic valve permanently incompetent, the murmur becomes more intense, and its area more extensive. The diastolic murmur may then be present over the whole length of the sternum, extending to the right of that bone at its upper portion; and slightly to the right, and to a great extent to the left of that bone at its lower portion; the area of the murmur sometimes extending as far outwards as the region of the apex of the heart.

The murmur of aortic regurgitation in cases of endocarditis is usually soft, smooth, and like a bellows sound. Sometimes it is musical, the note being fine and plaintive, limited in area to the middle of the sternum, or a little above that point, not penetrating, and easily obscured by the other sounds of the heart, and by respiration. It was thus in one case—a very pale woman aged 49. On her admission she presented tricuspid, carotid, and loud mitral systolic murmurs, and a musical diastolic murmur over the middle of the sternum. On the fourth day she was better, and all the murmurs were less marked; and on the sixth they were gone. Next day there was an obscure musical diastolic murmur, which also disappeared in a few days. In one case, on the 101st day after admission there was a double musical murmur to the left of the lower sternum. In another case, already alluded to, an exquisite musical murmur appeared just below the manubrium, extended to the left during the time of convalescence, was limited in area, and disappeared in about a week.

In another patient, a man, who came in with a mitral murmur, which established itself, a distinct double murmur appeared for the first time on the 69th day. Six days later the diastolic murmur appeared as a long whistle, but it resumed its usual character on the following day. One other patient that presented a peculiar musical diastolic murmur was a woman, aged 40, ill with acute rheumatism for four days, who came in with a faint blowing tricuspid murmur, which went on the third day, when she had pain in the heart. On the tenth she was better in every respect, but a peculiar diastolic murmur appeared to the left of the lower sternum, like the twang of a harp-string, which was still audible next day; but this was soon replaced by an ordinary short diastolic murmur to the left of the mid-sternum, which ceased after a few days, when both sounds were a little prolonged. Dr. Broadbent observed this case with me.

In another patient, a man affected with acute rheumatism and endo-pericarditis, a loud, grave musical murmur sprang up in the course of the illness, a vibrating murmur, with a perceptible thrill over the aorta, in the second right space, where the murmur was most intense; but the sound was heard to a great extent over and even below the chest. This murmur became established.

Of the remaining cases (14), in nearly one-half (6) the murmur was soft, or like a bellows sound, and this was undoubtedly its predominant character in the rest, although in them the precise nature of the murmur is not stated.

In about one-half of the cases of rheu-

matic endocarditis with aortic regurgitation, the murmur disappeared when the patients were under observation; while in the greater proportion of the remaining half, the murmur became fixed, being associated with established mitral regurgitation in two-thirds of those cases.

It was interesting and a source of anxiety to watch the progress of the murmur, dwindling and disappearing in the former set of cases, and ripening into permanent valvular disease in the latter set.

We have already seen that the fine musical diastolic murmurs with a limited area disappeared, while the louder ones of that class became established.

The character of the early murmur of aortic regurgitation gave little ground for foreseeing whether the incompetence would be permanent or transient. Thus in three, if not four, instances, a diastolic murmur, obscure, faint, feeble or confused at first, ripened later into an established aortic valve disease.

The history of the murmur, its development or decay, the widening out or contraction of its area, and the presence or absence, the increase or diminution of the characteristic signs of aortic regurgitation attendant upon the murmur; give more information as to the actual state, progress, and probable future of the patient than the exact character of the murmur on any particular day.

A statement of the duration of the murmur, and of the attendant secondary signs in the cases in which the valve completely regained its function; and a brief recital of the leading points in one or two of the cases that ended by producing aortic valve disease, will illustrate practically the probable future prospect of the affection in these important cases.

The diastolic murmur was short-lived in all but three of those cases that ended in restoration of the function of the valve, its duration being from one to eight days. In the three others the murmur, which diminished steadily in loudness, or sometimes remitted, lasted from fifteen to fifty days.

We shall be the better able to understand the extent to which these cases depart from health, and approach to disease, of the aortic valve with regurgitation, by rapidly reviewing the characteristic signs of the established disease, so as to obtain a standard of comparison.

The characteristic signs of permanent aortic regurgitation are—enlargement of the left ventricle, fulness over that ventricle, and undue force of the apex-beat, which extends beyond and below the left nipple; strong visible pulsation of the carotid arteries; sudden hammering stroke and collapse of the pulse, especially when the arm is raised, when the pulse is visible, and is audible with a loud shock that gradually lessens and disappears when the arm is lowered beneath the level of the heart; diastolic bellows murmur over the whole sternum, its maximum intensity being to the left of the middle of the bone; the murmur extending to its left at the lower portion of the sternum, becoming more feeble downwards, and to its right at the upper portion becoming more feeble upwards; a direct aortic murmur, generally audible over the manubrium, and to its right, where there is a true double aortic murmur; and a grave vibrating systolic murmur in the neck, over the visibly pulsating carotid artery, which is not followed either by a second sound or a diastolic murmur.

When the patient sits up, the extent of regurgitation and the collapse of the artery increases; and as a consequence, the diastolic murmur often becomes louder and more intense and extensive over its proper region; and the systolic murmur becomes more grave over the aorta and carotid artery, or is replaced there by a local and sudden shock when the regurgitation is very great so as to empty the ascending aorta during the diastole, the shock being occasioned by the blow with which the advancing column of blood is impelled by the stroke of the left ventricle upon the walls of the empty aorta and carotid artery.

If the incompetence of the aortic valve is caused by great enlargement of the aperture of the aorta, owing to dilatation of the vessel from atheroma, the artery extends to the right of the upper sternum, displacing the lung, and may present there a thrill and a loud vibrating musical murmur, heard, perhaps, at some distance from the surface, and extending over the whole chest, front and back, the neck, and even the abdomen.

My cases of endocarditis with aortic regurgitation ending in complete restoration of the valve, presented, with the exception of the double murmur, to a very slight degree the characteristic signs of disease with incompetence of the aortic valves. The diastolic murmur was present at the mid-sternum, and a little higher, and extended downwards, and to the left, becoming gradually feeble; but it was never heard upwards, over and to the right of the upper sternum, unless it was joined to a mitral murmur. The area of the diastolic murmur was thus limited and it was feeble, very soft, and like a bellows sound, or plaintively musical.

A systolic murmur was present over the aorta, or the carotid artery, or both, in two-thirds of the cases, this being an anæmic murmur, and not one caused by obstruction. It was due, in fact, to the flaccid state of the aorta, caused primarily

by the comparatively small amount of blood sent into it by the inflamed and weakened left ventricle, and increased by the reflux of a portion of that blood sent back again into the left ventricle through the inflamed and insufficient aortic valve. This flaccid state of the aorta allowed the blood contained in it to play freely to and fro in a series of noisy vibrations, with the effect of inducing a grave systolic aortic murmur.

The impulse was rarely notably strong. It was observed in four of the nine cases of this class. The apex-beat was felt close to the nipple in one of these patients; and in another, in whom the murmur lasted long, it was present on admission in the fifth space, outside the mammary line, and was stronger than usual on the 7th day; but it retreated within the nipple line from the 12th day, varying in position from the fourth to the fifth space.

The second sound, which is usually lost over the carotid artery in disease of the aortic valve, was audible in the neck in seven out of the nine cases of endocarditis in which the incompetence of the aortic valve was only temporary. In several of these cases the second sound was at one time or other less clear than natural over the neck, being feeble in two, grave in a third, and in a fourth, first prolonged, then silent, and afterwards natural, but feeble.

Although, then, in these cases, the second sound is still audible, perhaps, over the aorta, and certainly over its branches, the innominate and carotid arteries, it is often palpably modified in character. The presence of a second sound over the great arteries at the root of the neck, and over the ascending aorta, where it is, however, rendered doubtful by being blended with the transmitted presence of the pulmonic second sound, is due to the slight degree of the imperfection of the aortic valve. The shock of the second sound is therefore caused over those parts by the recoil of the walls of the distended arteries after the end of the systole, which sends the blood not only forwards into the arteries, but with a pressure equal in every direction, also backwards with a return-stroke upon the inner walls of the ascending aorta, including its sinuses, and slightly imperfect valve. The aortic second sound, although present, is often modified in tone and blunted, owing to the force of the back-stroke of the blood being impaired; (1) by the reflux of a small portion of the blood into the left ventricle through the inflamed and slightly insufficient valve; and (2) by the lessened supply of blood to the aorta and arteries from the left ventricle, the action of which is weakened by the inflammation of its inner surface. The degree to which the second sound over the neck is rendered feeble, blunted, prolonged, or almost or quite silenced, is a key to the knowledge of the amount of regurgitation, and of the defective supply of blood from the left ventricle. This important element of diagnosis is farther illustrated by what is found in cases of Bright's disease with contracted granular kidney, when the aortic valve is rendered slightly insufficient by the great distension and enlargement of the aorta. Here the blood is sent by the powerful left ventricle into the aorta and the arteries, already rendered tense by the difficult outflow of the poisoned blood through the small vessels; and the relief afforded to the tension by the reflux through the insufficient valves is so slight, that the back-stroke of the blood-caused by the recoil of the arterial valves is still made with so much force that the second sound usually retains the metallic ring, and the first sound the feeble note, so characteristic of aortic tension from Bright's disease.

Some of the cases of endocarditis with aortic regurgitation, ending in disease of the aortic valve, acquired step by step the characteristic signs of the permanent affection.

One case of this class, a man, ill a week, came in with quick breathing, a slightly prolonged second sound, and a rather extensive impulse. On the 5th day a soft mitral murmur appeared, which was loud on the 7th, when a diastolic murmur was also audible over the sternum, which extended next day slightly both to the apex and the neck. A week later there was a combination of mitral, tricuspid, and double aortic murmurs, and an obscure second sound was heard in the neck. At the end of the third week the disease was setting into its permanent form, the impulse being extensive, the carotid pulsation visible, and the second sound absent from the neck. The diastolic murmur, feeble on the 24th day, was loud on the 34th, when it was combined with a mitral murmur, and the apex-beat was strong.

Another patient, a laborer, ill eight weeks, was admitted with profuse perspiration, tremulous hands, rather quick breathing, and a double murmur to the right of the upper half of the sternum. On the 4th day the murmur was louder, and was audible over the right ventricle; but on the 6th he was faint, and the murmur was again limited to the aorta. On the 8th day he felt better, and the aortic murmur was again audible to the left of the lower portion of the sternum, as well as to the right of its upper portion. Variations followed, renewed diminution of the aortic murmur over the right ventricle being joined to renewed illness; but after this the systolic murmur became rasping, especially over the third right cartilage,

and the diastolic bellows sound became again widened in area.

The third case of this class, a woman, ill a week, came in with prolongation of the first sound, but no murmur. On the 3d day an obscure diastolic murmur was audible at the left nipple, and on the 7th this murmur was present along the whole sternum, especially from below the manubrium, and to the right of its upper portion. The second sound was heard in the neck, and the pulse was not distinctly audible at the wrist. On the 15th the diastolic murmur, smooth and prolonged, was more extensive downwards; the second sound, feeble at the apex, was audible in the neck; and a mitral murmur was present for the only time. On the 29th day the pulse was visible at the wrist, and on the 52d, when she was almost well, there was some fulness over the region of the heart, its impulse being stronger over both ventricles, and especially at the apex. The diastolic murmur was most intense at the fourth cartilage, but was audible along the whole sternum, except its summit. The second sound was still present in the neck, and the pulse was not audible.

In these three cases of endocarditis, the affection of the aortic valve advanced steadily, but with variations, under my notice, and during the evolution of the disease its characteristic signs came into play one by one.

The next case, a man, stands alone; the aortic regurgitation, after being suspended for a time, returned, and again lessened, without disappearing.

In the last group of four cases of endocarditis with aortic regurgitation, ending in disease of the aortic valve, the murmur appeared at a late period of the disease.

In one of these patients, a man, the murmur appeared suddenly without warning and in full force on the 88th day, being heard loud along the lower sternum. He had previously presented a variable mitral and an occasional tricuspid murmur. This mitral murmur was suspended during a period when the patient was ill with enteric fever, and when prolongation of the first sound was its temporary substitute.

A second case of this class, a boy, ill a week, came in with pain in the heart, a friction sound, and a mitral murmur, which was still present on the 5th day. After this there is a gap in the narrative until the 49th day, when there was still a mitral murmur. On the 69th day a double aortic murmur suddenly appeared for the first time, and already the pulse at the wrist was audible when the arm was raised. This diastolic murmur varied, increased, and extended to below the ensiform cartilage, but not to the top of the sternum, was once a long whistle, but generally a bellows sound; was accompanied by a mitral murmur at the apex, probably by a tricuspid, and certainly by a direct aortic murmur, there being no aortic second sound. The impulse of both ventricles became extensive, strong, and peculiar, pointing to adherent pericardium; it presented a double shock, one during the systole, and the other at the commencement of the diastole.

In the third case, a woman, one of remarkable interest, a faint diastolic murmur appeared to the left of the lower sternum on the 47th day, having been preceded and accompanied by varying mitral and tricuspid murmurs. In this case the thyroid gland became very large on the 64th day; was a good deal smaller on the 74th, and finally resumed its natural size. There was a distinct double murmur on the 101st day.

The last case presented healthy heart-sounds on the 17th day after admission, and on the 22d a soft diastolic murmur came into play to the left of the lower sternum, and a double aortic murmur just below the manubrium. The pulse was audible when the arm was raised, and the impulse was normal in extent.

These interesting cases of aortic regurgitation, coming on by surprise at a late period in cases of endocarditis, usually with a persistent mitral murmur and extensive and deep-seated inflammation of the interior of the left cavities, show that the aortic valve, though it suffers rarely and slightly when compared with the mitral valve, may silently and without warning, and when the patient appears to be well, break down in its functions by the steady and long advance of a latent inflammation.

When we consider how remote the aortic valve is from the focus of the inflammation, how passive and rigid the structures at the outlet of the left ventricle are in which that valve is imbedded, how gently the flaps of the valve come together, how comparatively slight is the force exerted upon the valve by the back-flow of the blood in the artery, due to the recoil of the walls of the aorta—that vessel being imperfectly supplied with blood by the inflamed and weakened left ventricle—a force that spends itself mainly in driving the blood forwards, and secondarily in impelling it backwards on the valve, it is only natural that the aortic valve should be rarely incompetent during the attack of endocarditis, and more rarely permanently crippled. These cases perhaps point to a gradual extension of the inflammation on the ventricular surface of the valve, and to the gradual yielding of the inflamed and softened valve; which at length gives way suddenly at its margin, and so admits of regurgitation from the aorta into the left ventricle.

IV.—Cases of Rheumatic Endocarditis with Prolongation of the First Sound.

The examination of the cases of endocarditis in which there was tricuspid, mitral, or aortic murmur, alone or in combination, show, I think conclusively, that prolongation of the first sound at the apex or over the right ventricle points to actual or imminent endocarditis.

Thus prolongation of the first sound both preceded and followed a temporary tricuspid murmur in three cases, preceded the appearance of that murmur in two other cases, and followed its disappearance in two additional ones. The first sound was therefore prolonged in one-half of the cases (7 in 13) in which a tricuspid murmur was present without a mitral murmur.

Again, a mitral murmur when present without aortic regurgitation was preceded and followed by prolongation of the first sound in seven cases; and was preceded by it in nine, and was followed by it in twenty other instances. The first sound therefore was prolonged in fully two-thirds (36 in 50) of the cases of endocarditis with mitral murmur in which there was no aortic regurgitation.

Finally, the first sound was prolonged in six of the ten cases of endocarditis with aortic regurgitation in which there was no mitral murmur; and in four of the nine in which there was both aortic and mitral regurgitation or in more than one-half (10 in 19) of the cases of endocarditis with aortic diastolic murmur.

If we combine the three series of cases with tricuspid, mitral, and aortic regurgitation, we find that in a little more than three-fifths of the whole number (53 in 82) the first sound was prolonged over one or other or both of the ventricles, and that this proportion held its ground in each of the three classes of valvular murmur from endocarditis. If we deduct from the 29 patients in whom there was no prolongation of the first sound, those who both came in and went out with tricuspid or mitral murmur, amounting to fully twelve cases, and who could not therefore present prolongation of the first sound preceding or following a murmur, we naturally increase the proportion in which the first sound was prolonged; and this proportion would necessarily be still further increased if we could deduct the unknown quantity of cases in which the prolongation of the first sound escaped observation.

It is evident then that prolongation of the first sound is a sign of transition; that it tends to expand into a mitral murmur when situated over the apex, into a tricuspid murmur when over the right ventricle, and occasionally into a systolic aortic murmur when situated over the aorta; and that when either of these murmurs passes away, it naturally glides into prolongation of the first sound over the region of the lost murmur.

Prolongation of the first sound over one or both of the ventricles in a case of acute rheumatism is in itself then a sign, actual, probable, or threatening, of endocarditis affecting the left cavities of the heart. If it is present when the face is covered with a diffused flush, or is dusky and anxious, when the breathing is quickened or oppressed, or when pain is seated in the region of the heart, and the second sound is intensified over the pulmonary artery, we may at once conclude that the patient is affected with endocarditis.

I have included among the cases of endocarditis two of the patients affected with acute rheumatism, who had prolongation of the first sound without murmur, but in both of whom that sound was murmur-like; and who had also several important symptoms pointing to internal inflammation of the heart, including pain over the heart in one, pain in the chest in the other, and very great general illness. I have ranked seven of these cases with prolongation of the first sound apart, among a class in which endocarditis was probable and I may say almost certain.

In more than one-half, or five, of these nine cases, including both those in which endocarditis was present, and those in which it was probable, the prolongation of the first sound was murmur-like in character. In six of these cases there was a pulmonic murmur; in four the face was dusky; in three there was restlessness or delirium; in two others the sleep was bad; in three there was pulmonary apoplexy, or cough, with phlegm; in one there was pain in the heart; and in two there was pain in the chest.

It is more difficult to settle the exact position of those cases with prolongation of the first sound that I have ranked among those threatened with endocarditis. Among the cases of this class belonging to the first series, amounting in the whole to 63, almost one-half (30) presented prolongation of the first sound: and in five more there was a double murmur; while in nine others the sounds of the heart were affected, the first sound being very loud in three, and doubled in one; while both sounds were feeble or indistinct in five.

Of the 30 patients in whom there was prolongation of the first sound, in one-half (14) there was great or considerable, and in 16 there was slight general illness. I think that we may consider that the fourteen patients with great or considerable general illness, nine of whom had pain in the region of the heart, were probably, or almost certainly, affected

with endocarditis. To these perhaps may be added the four patients who presented an obscure murmur. Three of these, however, had but slight general illness. If we add to the fourteen with great general illness and prolongation of the first sound, the case with an obscure murmur and also with great general illness, we may conclude that fifteen of those who were threatened with endocarditis were almost certainly attacked with that affection.

Among the 79 cases that are ranked among those who had no endocarditis, seven had prolongation of the first sound, and one had an obscure murmur. All of these had but slight general illness, and I think that they have been properly assigned to their present place.

If we examine the cases of the second series, or those treated by means of rest, we find that out of twenty-two cases threatened with endocarditis fourteen presented prolongation of the first sound. Of these nine had pain in the region of the heart, or great general illness, or both, while in one of them the general illness was slight. Eight of these cases may therefore, I think, be almost ranked with the cases of endocarditis.

In two of the remaining cases threatened with endocarditis there was a transient murmur.

V.—Cases of Rheumatic Endocarditis with previous Valvular Disease of the Heart.

Previous valvular disease of the heart was present in 22 of the 107 cases of endocarditis of the first series, and in 7 of the 28 of the second series of cases admitted into St Mary's Hospital under my care during the years 1851—1869–70. Among the cases of the first series, ten had mitral, five had aortic, and seven had mitral-aortic regurgitation, and the seven of the second series had mitral incompetence. Sixteen additional cases with previous valvular disease appear among my 325 cases with acute rheumatism of the first series; and of these eight had endocarditis combined with pericarditis, four had "probable" endocarditis, two were "threatened" with that affection, and only two presented no sign or symptom of endocarditis. We thus see that of the total number of cases of acute rheumatism with established valvular disease (amounting to 38), 30 (or 79 per cent.) had endocarditis; in 6 (or 16 per cent.) endocarditis was probable or threatened; and 2 (or 5 per cent.) had no endocarditis. Compare these cases broadly with the rest of the cases of acute rheumatism in which there was no previous valvular disease. Of the total number amounting to 287, 161 (or 56 per cent.), had endocarditis, of which 54 had pericarditis also; in 73 (or 25 per cent.) endocarditis was probable or threatened, including 3 with pericarditis; and in 83 (or 29 per cent.) there was no endocarditis, including 6 with pericarditis. We thus see that previous valvular disease of the heart, in cases of acute rheumatism, exercised an all-powerful influence in exciting endocarditis. Nor can we wonder at this important result. It has been the key-note, underlying the whole of this long clinical history of pericarditis and endocarditis, that whatever part liable to be affected by the disease, was exposed to the burden of labor, was exposed, in exact proportion to that labor, to the attack of inflammation, the severity and extent of the inflammation being proportioned to the amount of labor.

The presence, then, of established valvular disease, which adds very seriously to the labor of the heart in cases of acute rheumatism, adds very seriously to the probability, the almost certainty, of endocarditis in such cases. We have just seen that the influence of valvular disease, which tells with such force in the production of endocarditis, has but little effect in exciting pericarditis. The reason is, I think, obvious. The great extra work is thrown upon the interior, and not upon the exterior, of the left ventricle, and especially upon its mitral valve. A second local influence, in the altered apertures and roughened surfaces of the mitral and aortic valves, and especially at their margins, comes in to heighten the effect of the local labor in the production of endocarditis.

The two conditions that prevailed through the whole series of cases of established valvular disease with endocarditis are—the variability of the murmur from day to day; and great general illness. That chain of signs distinguished every case, and that chain of symptoms affected all but two of the whole series of instances of endocarditis with disease of one or more of the valves of the heart.

The variability of the murmurs showed itself not only in their greater or less loudness during the successive phases of the disease, but also in their transformation from one tone to another quite different; their extinction, suspension, and reappearance; and their extended, contracted, and shifted areas. This variation in the nature, character, and field of the murmur, is governed mainly by three leading influences:—(1) the changes to which the valves themselves and the interior of the heart are subjected by the inflammation; (2) the varying power of the heart under the influence of increasing general weakness, and returning strength; and (3) the tumultuous action of the heart owing to local pain, or the struggle to pass the blood onwards through

the obstructed orifices; or its intermission and failure from the exhaustion of previous overwork.

I shall illustrate the variable character of the murmur in these cases of endocarditis with previous valvular disease by the brief notes of a few cases, first selecting from among those with mitrai regurgitation, then those with aortic, and finally those with mitral-aortic valvular disease.

The first instance with *mitral* disease that I shall quote was a young woman who had left the hospital four days previously with a mitral murmur, due to a primary attack of acute rheumatism. She came in suffering from a fresh attack, with a distressed, anxious look, a dusky face, rather livid lips, and accelerated breathing. She had pain over the heart, its action being rapid and tumultuous, and an indistinct murmur. On the 3d day there was a loud systolic murmur at the apex, and the second sound was sharp over the pulmonary artery: and on the 4th she had agonizing pain in the heart, its action was tumultuous, and its sound could not be defined; she struggled violently and perspired profusely. Next day a loud systolic murmur, tricuspid as well as mitral, was audible over the whole region of the heart. On the 10th day the tricuspid murmur was audible along the sternum, and a second impulse, with a loud second sound, were present over the pulmonary artery in the second left space. On the 18th she was bright and cheerful, but a cough was still present, and the murmur was softer. On the 23d day she walked about the ward, but on the 29th there was a return of pain on movement, and the murmur was louder. After this she did well, there was a thrill over the heart, the murmur was loud over the apex, and was heard over the left scapula. Here the mitral murmur was obscured when the heart was tumultuous; and was loud and smooth, and joined by a tricuspid murmur, when the health improved and the heart was steady in its action. Another case, with previous mitral regurgitation, had, when admitted, tightness of the chest, pain over the heart, and a loud systolic murmur. Three days later, with less pain, the murmur was almost musical at the apex, and quite so below it over the stomach; two days later she looked better, and the murmur presented a third change, being not nearly so loud; but next day, with returning tightness of the chest, there was a fourth transformation of the murmur, which was rasping or almost musical over the heart; the 10th day, however, with renewed improvement, showed a fifth variation in the murmur, which was no longer rasping; but on the following day there was a sixth change, and the murmur was musical around the apex; after this, on the 13th day, the murmur was grave, this being its seventh variation; its eighth occurring on the 18th day, when it was again musical over the stomach, and when it was joined by a systolic murmur over the aorta. After this, with steady improvement, the murmur was no longer variable. A third case illustrates the variations of the murmur during the convalescent period.

These two cases are typical, but their successive snatches of ever-varying murmur, contrast with the murmur, now swelling, now dwindling, that is found in other and more simple cases. I will just quote one of these. A youth, a carpenter, came in with pain in the chest and a prolonged musical systolic murmur at the apex. This murmur was persistent, but it varied in tone, being grave on the 8th day, when pain was present. The heart's beat was strong.

Each of the remaining seven cases presented features of its own; the variations of the murmur being great and complicated in four of them, and in three of them comparatively simple. In four cases, if not five, the mitral murmur was associated with a tricuspid murmur, in one with a pulmonic, and in one with a direct aortic murmur; while in one the first sound was prolonged over the right ventricle. In one of the cases just enumerated, a diastolic aortic murmur appeared and disappeared, reappeared, and was finally extinguished, the mitral murmur being permanent throughout.

The *aortic* murmurs of established valvular disease scarcely vie with the mitral murmurs in variety of tone, loudness and area, and alternate extinction and return, in cases of rheumatic endocarditis; but I may state that the study of the five cases that I can cite shows that in all these points the diastolic-aortic murmur presents frequent variation; though the systolic murmur of aortic contraction is much less subject to change.

In one case with aortic regurgitation, probably of some standing, tricuspid and mitral murmurs were added temporarily to the diastolic murmur, which varied much and was not always audible during the attack of endocarditis. At the cessation of the illness a double aortic murmur was alone audible. In the other case a double aortic murmur, which went and came again during the illness, was apparently joined on the 28th day by a tricuspid murmur, which had departed on the 34th, leaving a double aortic murmur.

The remaining seven instances had previous *mitral-aortic* valvular disease. Two of the cases belonging to this last group were admitted twice with mitral aortic endocarditis, so that the actual number of patients belonging to it is reduced to five. One of those two patients that

were thus admitted twice with endocarditis, had left the hospital six months previously, after an attack of rheumatic endocarditis, and came in with double aortic, and mitral murmurs; which varied somewhat in loudness and extent, but were substantially unchanged during this illness. Four years later she returned with severe acute rheumatism and endocarditis, and died after a very long illness, albuminuria having been finally added to her ailments. The murmurs underwent several oscillations, sometimes the mitral, sometimes the aortic diastolic murmur, being very loud, while at other times one or other of those murmurs was almost or quite extinguished at the heart; the mitral murmur being however generally distinctly though feebly audible over the back of the chest.

In the three remaining cases the variations in the murmurs were rather in loudness and extent of area, than in the tone and character of the sounds.

The extent and strength of the impulse, and their variation during the attack, are among the most decisive tests of the previous presence of valvular disease in cases of rheumatic endocarditis. As a rule, the impulse in such cases is unduly diffused, strong, and propulsive; and this applies more in degree to cases with mitral-aortic, than to those with simple mitral regurgitation. The extent of the impulse in a case of valvular disease without endocarditis, is a test of the undue amount of labor to which the heart has been put to overcome the obstacle to the circulation of the blood caused by the affection of the valves. The supervention of endocarditis sometimes, by rendering the heart's action tumultuous, increases the impulse; but sometimes its effect is the reverse, and by lowering the power of the heart, it lessens the impulse.

Among the ten cases of endocarditis with previous mitral incompetence, including one in which aortic incompetence sprung up temporarily during the attack, in five the impulse was strong, in one it was diffused, in two it was moderate, in one it was feeble, and in one it was not described. In three of those cases the impulse was stronger during the attack of endocarditis than after it, and in two it was the reverse. The impulse of the left ventricle was usually increased in the cases of established mitral incompetence, but that of the right ventricle was, in proportion, more affected in those cases.

Among the five cases of previous aortic incompetence with endocarditis, including the two that were joined during the attack, one by mitral, the other by tricuspid incompetence, in three the impulse was strong and extensive, especially towards the apex; in one it was diffused but rather feeble; and in one it was of moderate force and extent. The impulse was more extensive during the attack of endocarditis than after it in one case. The impulse was strong, extensive, and unduly far to the left, in five of the seven cases of previous mitral-aortic incompetence with endocarditis; it was diffused but rather feeble in one; and in one it was feeble. The impulse appeared to be strengthened during the period of the endocarditis in four instances, while in one case it was the reverse.

Pain was present over the region of the heart in four of the ten cases of endocarditis with previous mitral incompetence, in four of the five with aortic incompetence, and in four of the seven with mitral-aortic incompetence. There was pain in the side or chest, or tightness of the chest, not including those with pain in the heart—in four of the ten with mitral; in one of the four with aortic; and in three of the seven with mitral-aortic valvular disease. There was no pain either in the heart, chest, or side, in two of the ten cases with mitral; in none of the five with aortic; and in one of the seven with mitral-aortic valvular disease, or in only three of the twenty-two cases under consideration. We have seen that pain in the heart, side, or chest, occurs in by far the largest proportion of such cases; and that pain in the parts named is much more frequent in cases of endocarditis in which the heart was previously affected with valvular disease, than in those cases of endocarditis in which the heart was previously healthy.

The respiration was seriously affected in a very large proportion of the cases of valvular disease with endocarditis. This condition in such cases is inevitable, for the effect of all the diseases of the valves is to interfere with the efficient onflow of the blood towards the system, and therefore to throw the blood backwards upon the lungs. This applies of course with primary and immediate force to incompetence of the mitral valve, which throws a portion of the blood just received back again upon the lungs, with the effect of overcharging the pulmonary vessels. The return of the blood back again from the aorta, owing to aortic incompetence, into the left ventricle from which it has just been sent, is, however, only one short stage forward from the seat of mitral incompetence; and the almost immediate effect of the aortic incompetence is to produce a back-flow of blood upon the pulmonary vessels, and to delay the blood in those vessels and congest them. The presence of this surplus amount of blood in the lungs, which upsets the healthy balance of the circulation through the lungs and the body, compels the respiratory organs to exert themselves to the top of their power, so that they may, if possible, expel forwards into the body the weight of blood that oppresses them. Hence result

laborious, difficult, and rapid breathing, pulmonary apoplexy, pleurisy, catarrh, and bronchitis.

The respiration was rapid in four, the chest was painful or tight in two, and cough with pulmonary apoplexy occurred in another of the cases with mitral valve-disease; while in two of those cases there is no note of the state of the lungs, and in one they were healthy in function. The breathing was quick, or there was cough, or pain in the chest, in four of the five cases with aortic, and in six of the seven with mitral-aortic valvular disease. More than three-fourths, therefore, of the cases of valvular disease with endocarditis had serious disturbance of the respiratory functions.

CLINICAL HISTORY OF ENDOCARDITIS IN CASES OF CHOREA.

The association of chorea with endocarditis has long been known, both clinically and from examination after death; and it has already received illustration in this volume, at pages 531, 532, where two important cases of chorea are alluded to that have been published by Dr. Broadbent and Dr. Tuckwell, in both of which there was endocarditis, and minute cerebral embolism; and in one of which there was acute rheumatism as well as chorea. I had also occasion, in this article on endocarditis, to give at page 635 a case which illustrates the association of chorea with endocarditis. I shall now give a brief account of the cases of chorea treated by me in St. Mary's Hospital, with especial relation to their association with endocarditis.

Clinical History of the Cases of Chorea, in relation to the presence of Endocarditis, observed by the Author in St. Mary's Hospital.—I find notes of 40 cases of chorea that were under my care in St. Mary's Hospital, and in 34 of them the signs of the heart are noted, while in 6 of them they are not so.

CASES OF CHOREA IN RELATION TO THE PRESENCE OR ABSENCE OF ENDOCARDITIS.

1.—Cases in which there was no endocarditis, heart sounds healthy			10
2.—Cases in which there was probably no endocarditis:—			
a. Slight prolongation of the first sound	5		
b. Anæmic murmur over the pulmonary artery	1		
		—	6
3.—Cases in which there probably was endocarditis:—			
a. Prolongation of the first sound	3		
b. Murmur, tricuspid or pulmonic	2		
		—	5
4.—Cases in which there was endocarditis:—			
a. With mitral regurgitation			
Ending in restoration of valve	2		
Lessening of murmur on recovery	2		
Mitral regurgitation established on recovery	8		
	12		
b. With aortic regurgitation	1		
		—	13
			34
Cases in which the heart was not observed			6
Total			40

Association of the Cases of Chorea with Rheumatism.—The well-established association of chorea with articular rheumatism, renders the study of the connection of rheumatism with these cases of chorea necessary before we consider the occurrence of endocarditis in chorea. Acute rheumatism, as we have just seen, is so very frequently accompanied by endocarditis that we must be careful, when ascertaining the frequency of endocarditis in chorea, not to attribute the internal inflammation of the heart too readily to chorea, when it may be caused by the rheumatism associated in certain cases with that affection.

Articular rheumatism, in a subacute form, was definitely present during the attack in six of the forty cases of chorea. In five of these cases the rheumatic affection immediately preceded the occurrence, and continued for a short time after the supervision of the attack of chorea. In one of the cases, in which there had been no previous rheumatic attack, the joints became inflamed in the course of the choreal affection.

In addition to these six cases of chorea with pronounced articular rheumatism, there were five cases of chorea in which there was pain in all the limbs (in 1), or in the shoulder and hips (in 1), or in the legs

(in 1), or in the hands (in 1), or there was stiffness of the arms and legs, and of the left ring-finger (in 1). In none of these cases, however, was there swelling or redness over the joints; but this does not apply to the redness which affected the wrists, elbows, and face in one patient from violent friction. There were also five cases of chorea that were free from rheumatism during the attack, that gave a history of antecedent acute rheumatism, occurring from two years to two or three months, and in one instance for an uncertain period, before the occurrence of the chorea.

The proportion in which endocarditis appeared in those cases will be given presently.

Proportion of Cases of Chorea in which Endocarditis was present.—In nearly one-third (10 in 34) of the cases of chorea in which the sounds of the heart were observed, those sounds were healthy; in one-sixth of them (5) there was slight prolongation of the first sound, and in one case there was a pulmonic murmur. I have classed the six latter cases among those in which there was probably no endocarditis, and I think we may infer that those sixteen cases, amounting almost to one-half of the whole, were free from inflammation of the interior of the heart.

In three cases in which there was marked, almost murmur-like, prolongation of the first sound, and in two with a tricuspid or pulmonic murmur, amounting to almost one-sixth of the whole (5 in 34), the presence of endocarditis was probable.

The remaining cases, amounting to fully one-third of the whole (13 in 34), gave complete evidence of the existence of endocarditis, in the presence of a mitral murmur in twelve instances, and of a diastolic-aortic murmur in one.

I think it probable that the majority of the six cases of chorea in which the heart was not observed, ought to be added to those in which there was no endocarditis.

Cases of Endocarditis with a Mitral Murmur.—The cases of choreal endocarditis with mitral regurgitation, considering the comparatively small number of those cases, offered as great variety in character, mode of commencement, course, and result, as the cases of rheumatic endocarditis with mitral regurgitation.

Endocarditis with mitral regurgitation ended more than twice as often in established mitral disease in chorea, than in acute rheumatism. Mitral regurgitation became permanently established in two-thirds of the cases of chorea with mitral murmur (8 in 12); and in less than one-third of the cases of acute rheumatism with mitral murmur of the first series (14 in 49), and in only one-sixth of those of the second series treated by rest (3 in 20). The integrity of the valve was restored in one-sixth of the cases of chorea with a mitral murmur (2 in 12), and in another sixth of them the murmur was becoming feebler when the patient left the hospital (2 in 12).

The mitral murmur in fully one-half of the cases (7 in 12) was situated in the region of the apex, and was not described as extending beyond that region; but was simply entered as a systolic mitral murmur, or a systolic murmur at the apex.

The five remaining cases, compared with those just dismissed, presented greater breadth of area; variety in intonation and volume of sound; and individual life.

In two of these cases the mitral murmur was very extensive, being audible over the back of the chest, above and below the scapulæ, and the greater part of the left side. One of them, when admitted, had been ill with chorea in a severe form for some weeks, but the affection was now but slight. A loud systolic murmur centred itself at the apex; and was audible along the sternum, and far to the right of its lower portion, though feeble at its upper part; from the third to the seventh left costal cartilages; in front of the epigastrium and the liver; and all over the dorsum, especially on the left side. The impulse of both ventricles was immoderately strong and extensive, the apex-beat being present an inch to the left of the nipple-line. These signs underwent little change after the admission of the patient, and it was evident that the endocarditis had ceased.

The other case came in with acute endocarditis, a mitral murmur being audible at the apex and over the right ventricle. A few days later it could be heard towards the axilla, and over the back, as high as the upper part of the scapula. At the end of the seventh week the murmur was grave and musical, and a fortnight later it appeared as a prolonged bellows sound. After this it was hardly so loud, but towards the end of the fourth month after admission it was grave and vibrating.

This case had an interest much broader than the simple relation of chorea to endocarditis; for it had interwoven with it from its commencement, and throughout an important part of the early period of its course, the relation of acute rheumatism to chorea, and of acute rheumatism to endocarditis also. It began with inflammation of the ankle, conjoined with chorea. Six weeks later, when admitted, the knee was inflamed, chorea being the most pronounced disease, and the two affections being accompanied by endocar-

ditis. Was this endocarditis the direct offspring of the subdued attack of acute rheumatism, or of chorea, or of the two conjoined affections, each taking its part in giving a combined birth to endocarditis?

During the third week the arms were slightly rheumatic, as well as the lower limbs, and the patient lay motionless in bed, apparently stilled by the affections of the limbs and joints, the chorea being almost or quite latent. After this the rheumatism insensibly disappeared, the chorea insensibly reasserted itself, and for the remainder of the patient's long history, the chorea, modified in form and severity, was the only apparent affection; accompanied throughout, however, by endocarditis.

The other three instances of which I have to speak were cases of chorea, unalloyed, during the attack, by rheumatic arthritis; but two of them had suffered some time before from acute rheumatism.

One of them, a girl, had been long ill with chorea, and had gone through a rheumatic attack two years before. She came in with a loud, smooth, systolic murmur at the apex, which was audible over the right ventricle. After this the murmur underwent minor transformations, being like a bellows sound on the 4th day, and almost musical on the 8th, when the apex-beat extended further outwards, the murmur being faintly, if at all, audible below the angle of the left scapula. The apex-beat extended a little beyond the nipple. This case came in with endocarditis, which was evidenced by the varying character of the murmur; but there is nothing to show whether or not this patient had acquired mitral disease from the old attack of acute rheumatism.

This last question does not complicate the next case, for though this patient, a girl, had twice been affected with acute rheumatism, yet she had no murmur, but a prolonged first sound, on admission. A murmur, however, appeared at the apex on the 4th day, which was grave on the 6th and the 8th, and was loud on the 14th day, when it extended towards the axilla. The apex-beat was strong on the 6th day, three and a half inches from the sternum; but on the 8th it could scarcely be felt.

The last case, a boy, was free from rheumatic taint, and presented no murmur during the first six weeks; but at the end of that time he had pain in the chest, and a week later a smooth bellows murmur appeared at the apex, which three weeks later spread upwards towards the axilla, and downwards over the stomach. After this, during a long period, extending from first to last over five months, the murmur underwent various changes, being a very smooth bellows murmur on the 62d day, audible upwards towards the axilla, and downwards over the stomach. On the 76th the murmur was very loud and superficial, being heard towards the axilla, but for a very short way below the heart. The first sound was very feeble, while the second was very loud over the pulmonary artery, in the manner already related at page 623. After this the mitral murmur underwent various modulations, being moderately loud on the 102d day, very loud at the apex on the 105th, but scarcely audible over the lung to the left, or towards the axilla; much weaker on the 126th; but on the 135th day it was loud below, especially on expiration, and was not heard outwards during inspiration. On the 146th day, and the last report, there was very slight fulness over the heart, the impulse of the right ventricle, which seven weeks previously was strong, extending from the third cartilage to the sixth, and from the sternum to the nipple, was on the last observation less strong to the right of the lower sternum, and extended from the second to the fourth cartilages. The mitral bellows murmur was not so smooth as before, and was again heard up to the axilla. The double impulse of the pulmonary artery, previously marked, was no longer perceptible. There was no murmur over the back. He went out comparatively well, being free from choreal movements.

In this case, as in that just related, during the attack of endocarditis, when the patient lay speechless in bed, the heart became enlarged, and the lung shrank away from before the heart, exposing its increased impulse over a large area; and the mitral murmur was heard extensively over the region of the contracted lung, and that of the stomach. At a later period, however, with returning health, strength, and exercise, the lung expanded freely, and interposed itself between the greater part of the heart and the walls of the chest, so as to cut off the extended border of the area of impulse, and to lessen that of the murmur by damping and silencing its sound.

Case of Endocarditis with a Diastolic-Aortic Murmur.—This patient, a boy, came in with a second attack of chorea, which began three weeks previously with pain in the legs of a rheumatic character, followed, a week later, by choreal symptoms, which became gradually more severe. On his admission the heart sounds, so far as they could be made out, were healthy, but on the 3d day a diastolic murmur was audible over the centre of the sternum. Ten days later this murmur was heard, very prolonged and loud, over the whole length of the sternum; being audible to the right of the upper

part of the bone, and to the left of its lower portion, but becoming weaker towards the apex of the heart. On the 86th day the diastolic murmur was still loud, and maintained its ground everywhere; and it was joined by a systolic murmur, loudest at the sternum and not mitral. Three weeks later the diastolic murmur was inaudible at the middle of the sternum, and was feeble at its upper and lower portions; but on the 79th day, the last observation, it had apparently resumed much of its loudness and extent, and the systolic murmur was silent.

In this case, as in one of those just told, the question must be put, Was the endocarditis caused by the primary articular rheumatism, or by the resulting chorea, or by the combined influence of the two affections?

ENDOCARDITIS IN PYÆMIA.

There was only one instance among the 71 cases of pyæmia or secondary inflammation examined after death in St. Mary's Hospital in which the appearance of endocarditis was observed and reported. That case, a man, who was under my care, presented a spot in the right lung, an inch long, consisting of pus, and apparently broken down lung-tissue, and superficial to this a patch of dry fibrinous deposit on the pleura; and numerous spots, similar but smaller, through the back of the middle and lower lobes of that lung. There was also a large globular and fluctuating tumor on the upper and inner part of the left kidney three inches in diameter. On cutting into it highly offensive blood-like fluid escaped, and on laying it freely open there was a clot of blood and a little pus. The sac was lined with a delicate, highly organized, chorion-like membrane, with numerous prominent bloodvessels ramifying on its surface. There was a large black spot of apoplectic effusion in the substance of the kidney near the membrane. The structure of the kidney was healthy.

The heart was of natural size, and there was a patch of recent roughness on the surface of the left auricle. Several nodules, from the size of a split pea to that of a millet-seed, were situated on the free margin of the mitral valve. The corpora Arantii of the aortic valve were enlarged. The patient was admitted in a state of great depression, his mind wandered, and mucous and sonorous noises were audible over the chest. The state of the heart was not observed.

The attack of endocarditis was in this case the marked secondary effect of the pyæmia, but the solitary occurrence of this instance with endocarditis in 71 cases of pyæmia shows that the inflammation of the interior of the heart, so common, as we have seen, in acute rheumatism and chorea, is rare in pyæmia, though less so, as we shall see, than in the fatal stage of Bright's disease.

The signs of the heart affection were not observed in this case of pyæmic endocarditis. I have had, however, frequent opportunities of examining a patient affected with pyæmia, in the course of whose very serious illness the signs of endocarditis appeared and held their ground. Pleurisy first showed itself, and the evidence of inflammation in both lungs; and after a time a systolic murmur became audible at the apex. This murmur was constant, but it varied in loudness, tone, and area during the course of the illness. After this patient's recovery a mitral murmur was established.

ENDOCARDITIS IN BRIGHT'S DISEASE.

I have only been able to find one instance with evidence after death of endocarditis in the whole of the cases of Bright's disease described in the post-mortem records of St. Mary's Hospital, amounting to 207, excluding those in which there was regurgitation through the mitral or the aortic valve, or through both valves, or obstruction of the mitral orifice. That case was one of fatty disease of the kidney in a man, aged 41, who was under my care. His heart was rather large, weighing 12½ ounces, and was dilated and flabby. The structure of the valves was healthy, with the exception of a patch of white deposit on the anterior flap of the mitral valve, which did not appear, after death, to interfere with the function of the valve.

This man, when admitted, presented a yellowish pallor and puffiness of face. He had been a healthy man until he took cold, nine months previously, after which he became gradually weak and pale, and had palpitation and frequent vomiting, symptoms with which he was still troubled. There was some albumen in his urine. The right veins of his neck were rather swollen and pulsating, and there was pulsation of the temporal artery. The heart's impulse was very feeble, and diffused over the cardiac space during expiration only, but it could be felt between the ensiform cartilage and the left seventh costal cartilage. The liver was firm and low, and presented a diffused pulsation in the epigastric space.

A soft systolic bellows murmur was audible at the apex, and a peculiar short double murmur between the nipple and the sternum, which was obscured by the natural heart sounds. These murmurs varied considerably from day to day, but

they were generally audible, though the diastolic noise was more or less obscure. About a week after his admission a peculiar humming venous murmur was heard to the right of the sternum when he sat up, but not when he lay down, which, sometimes, disappeared without apparent cause, when it could be brought back by pressure over the jugular vein.

On the 42d day he presented considerable general dropsy, and for the first time the murmurs were very faint and obscure, and two days later they were lost. After this the mitral murmur was sometimes audible, but was generally not so, and the diastolic murmur was only heard once, corresponding with a thrill near the apex. The last observation was made on the 77th day, when a faint systolic murmur was heard over the seventh cartilage, and feeble doubling of the first sound over the sixth cartilage. The urine was then scarcely albuminous, and it had been so during a considerable period of the history of this patient, who died on the 98th day.

I have ranked this case as one of endocarditis, because of the presence of a white deposit on the mitral valve, which was otherwise healthy, and of the history of varying murmurs, pointing to changing affection of the mitral and aortic valves. The long duration of the case, and the small amount of change to which the valve had been subjected, make it doubtful whether the endocarditis was present in more than its effect, the white deposit on the mitral valve, at and before the time of death; but if we take that appearance, and the varying signs of double regurgitation into account, I think we may infer that this case was one of endocarditis. It is true that both mitral and aortic regurgitation may be present in Bright's disease when there is very great tension of the arteries, and great hypertrophy, with dilatation of the left ventricle; that in such cases those murmurs usually vary in character, according to the varying intensity of the causes that gave them birth; that they may be suspended, restored, and again lost, even permanently; but this case did not present those conditions, for the heart, though dilated, was not greatly enlarged, and was not hypertrophied, since it only weighed 12½ ounces.

Admitting, then, that this was a case of endocarditis occurring in a patient affected with Bright's disease, it is evident, that as this was the solitary instance of that kind that was noticed among so many cases of Bright's disease without disease of the valves, that although endocarditis may occur in that disease, yet that it is rare. This becomes more marked when we compare the cases of acute rheumatism, and of chorea, with those of Bright's disease; for in the two former affections, from one-half to one-third of the cases were affected with inflammation of the interior of the heart.

The frequent presence of thickening of the mitral valve, and occasionally of the aortic valve; and the large proportion of cases of valvular disease without a previous history of acute rheumatism; perhaps point to the occurrence of endocarditis in those cases during the earlier period of their history. If so, endocarditis, and pericarditis, behave very differently from each other in Bright's disease, for while pericarditis is common towards the fatal period of this disease, especially when the kidney is granular, and is rare during its earlier history, endocarditis is very rare towards its fatal period, but is not very infrequent during its earlier history; that is—if the thickening of the valves, and especially of the mitral valve, and complete valvular disease, have their origin in the Bright's disease itself.

CLINICAL HISTORY OF ENDOCARDITIS OCCURRING IN CASES OF VALVULAR DISEASE OF THE HEART.

The influence of previous valvular disease in rendering endocarditis more frequent and severe in cases of acute rheumatism has been already seen at page 648. We then observed that the presence in that affection of disease in the valves of the heart, by adding to the labor of that organ, and by rendering its internal apertures more rough and irregular, increased the danger of the occurrence of internal inflammation of the heart, and intensified that inflammation when established.

So great, indeed, is the influence of valvular disease in exciting and intensifying inflammation of the diseased valve, that we find that endocarditis is apt to occur in such cases, even when free from acute rheumatism, chorea, or any other general disease.

I would refer here to some interesting remarks by Dr. Moxon on this important subject.

The accompanying table (p. 656) will show at a glance the proportion in which endocarditis was present at the time of death in the cases of valvular disease of the heart treated in St. Mary's Hospital.

Pathological Evidence of Endocarditis in cases of Valvular Disease of the Heart.

It is difficult, even impossible, in every case to say, from the appearances presented after death, whether or not endo-

carditis is present on the affected valves, and the adjoining surfaces of the ventricle and auricle. This is due to the readiness with which, in certain cases, a deposit of fibrin from the blood as it streams backwards and forwards through the mitral and aortic apertures, attaches itself to the surfaces of the imperfect valves, roughened by disease. This is equally the result, whether those surfaces be roughened by the slow degeneration of the diseased fibrous tissues, which, although they may have been generally inflamed at the starting point of the disease, yet they may have long ceased to be so; or whether the surfaces of the valve be inflamed by a recent and renewed attack of local endocarditis. In many instances, however, it is self-evident that inflammation actually affects the valve, for the appearances presented are precisely those that are found in cases of recent endocarditis, owing to acute rheumatism, chorea, or pyæmia. Those appearances in these cases are to be confided in, for the diseased valves have been described, without, however, as a rule being defined as being inflamed, by a succession of able and careful pathologists, including the distinguished names of Dr. Markham, Dr. Burdon Sanderson, Dr. Murchison, Mr. Gascoyne, Dr. Charlton Bastian, and Dr. Payne.

Table showing the number of cases with established valvular disease, among those not affected with acute rheumatism, in which endocarditis was present at the time of death.

		Affected with Bright's disease
I.—Cases with established mitral regurgitation:—		
a. Cases with endocarditis, not affected with Bright's disease	9	5
b. Cases with fibrinous concretions on the valve, probably not affected with endocarditis	2	3
c. Cases in which no description of the valve was found	1	2
d. Cases without endocarditis or concretions	22	19
I.—Total	34[1]	29[1]
II.—Cases with aortic regurgitation: (A)—from disease of the aortic valve:—		
a. Cases with endocarditis, not affected with Bright's disease	5	1
b. Cases with fibrinous concretions, endocarditis doubtful or absent	5	5
c. Cases in which there was no description of the valve	2	2
d. Cases without endocarditis or concretions	13	12
Total	25	20
(B)—From great dilatation of the aorta, the flaps of the valve being healthy but insufficient	5	1
II.—Total with aortic regurgitation	30	21
III.—Cases with mitral-aortic regurgitation:—		
a. Cases with endocarditis, not affected with Bright's disease	5	3
b. Cases with fibrinous concretions, endocarditis doubtful or absent	4	0
c. Cases in which there was no description of the valve	3	0
d. Cases without endocarditis or concretions	16	16
III.—Total	28	19
IV.—Cases with obstruction of the mitral orifice:—		
a. Cases with endocarditis affected with Bright's disease	1	1
b. Case with roughness and ulcer at edge of valve	0	1
c. Cases with vegetations or concretions on valve, endocarditis doubtful or absent	2	1
d. Cases without endocarditis or concretions	18	6
IV.—Total	21[2]	9[2]

[1] I am not certain that these numbers include the whole of the cases with mitral regurgitation, since most of the original copies of those cases have been lost or misplaced, and I have taken them from a detached tabulated abstract of those cases.

This note applies also to the cases of mitral regurgitation given in the Table at page 651.

[2] In 5 of these cases the size of the mitral aperture is not described; in 5 it was contracted to a moderate extent, and in 19 to a great extent; and in 1 it was almost closed by a ball of organized fibrin.

Among the cases of *mitral regurgitation*, five presented "fringes" and one a ring of small papillary elevations or granulations around the free edges of the valve, and two others had warty or rough excrescences, and another had nodules of lymph on those free edges; and in one of these, the auricular surface of the valve was roughened. One of those instances described, I think, by Dr. Payne, presented also yellow succulent elevations, almost resembling a false membrane, but seated under the epithelium. I have also included among the cases of endocarditis four instances with vegetations on the auricular surface of both flaps of the mitral valve, and one with extensive ulceration of its anterior flap, in which case the adjoining surface of the ventricle was inflamed; five other cases presented large excrescences, or concretions and smaller vegetations, but these I have not included among those with endocarditis, although some of them may have had that affection. This may be said also of a doubtful case in which the posterior flap of the valve was attached to the wall of the ventricle by adhesions readily separated.

Five of the fourteen cases that I have classed among those with endocarditis were affected with Bright's disease, and nine of them were not so.

Forty-one cases with mitral regurgitation were free from vegetations, and of these, nineteen had Bright's disease, and twenty-two were free from that affection.

The cases with *aortic regurgitation* presented comparatively few instances or severe, with endocarditis, but these presented great variety in their features. One of them showed deposits of red vegetations towards the edge and centre of each flap of the aortic valve. In another, the flaps of the valves were cemented together, and then free margins were roughened, by fibrinous deposit. In a third the aortic aperture was converted into a mere chink by adhesions; and there was an irregular deposit of lymph, forming vegetations, about the basis of the conjoined flaps, some being hard, some cheesy, and others apparently quite recent. The united flaps projected like a funnel into the aorta in the fourth instance, and a little above the valve, and therefore on the inner surface of the aorta, was an oval patch, half an inch long, with a red highly vascular flocculent surface. The aortic valve, in the fifth case, was enlarged but soft. One of the flaps had ulcerated away at the sides, and a large nodular mass was appended to its sesamoid body. The sixth case was one of great interest, with contraction of the descending aorta below the subclavian artery so as scarcely to admit a probe, and embolism, blocking up the left brachial artery. The valve was universally red, soft, pulpy, and formless, and the aperture was contracted. I had originally only ranked five of these cases as being affected with endocarditis, but I think that the whole six may safely be so classed. Only one of these six cases with endocarditis had Bright's disease, the remaining five being not so affected. Ten other cases presented concretions of various size, some being large, one like an alpine strawberry, attached to the aortic valve; these cases being affected, and unaffected, by Bright's disease in equal numbers. Twenty-five of the cases with aortic regurgitation were free from contraction, and of these, thirteen had Bright's disease, and twelve were free from that affection. In six cases, aortic regurgitation was due to great enlargement or dilatation of the ascending aorta, the flaps of the aortic valve being healthy in structure, but of insufficient size to close the widened orifice of the aorta.

It will I think be sufficient if I state the proportions in which the cases with *mitral aortic regurgitation* were affected with endocarditis, presented concretions, without distinct evidence of endocarditis, and were free from concretions, without entering into details. I consider that eight of those cases had endocarditis, five being free from, and three being affected with, Bright's disease; four of them had concretions on the valves, none of which had Bright's disease; and in thirty-two there was no concretion on the valves, one-half of these being free from, and the other half affected with Bright's disease.

I shall deal with the cases with *obstructed mitral orifice* in the manner that I have just dealt with those having mitral-aortic regurgitation. Two of them had endocarditis, one being free from, and one affected with, Bright's disease, and another case having that disease presented roughness and ulceration of the edge of the contracted mitral valve; three had vegetations, one of those only having Bright's disease, and twenty-five of them had neither endocarditis nor concretions in any form on the obstructed mitral orifice, only seven of which cases had Bright's disease.

It is evident that while cases with mitral regurgitation are affected in a rather large proportion, or nearly one-fourth (14 in 63), with endocarditis, only one, or at most two, in twenty-nine of the cases with obstruction of the mitral orifice gave evidence after death of that affection. Cases with aortic regurgitation occupy a middle position between the two classes just considered, 6 in 51 (or 1 in 9) of these cases being affected with endocarditis. The cases of aortic regurgitation that were free from Bright's disease were much more frequently affected with endocarditis (5 in 30 or 1 in 6) than in those that were affected with that disease (1 in 21).

Cases with mitral-aortic regurgitation have endocarditis rather more frequently (8 in 47 or 1 in 6) than those with aortic regurgitation (6 in 51 or 1 in 9), and less frequently than those with mitral regurgitation (14 in 63 or 1 in 4½).

Valvular disease was less frequently attacked with endocarditis in those cases that were affected with Bright's disease (11 in 78 or 1 in 7) than those that were free from that affection (20 in 105 or 1 in 5·2); and, as we have seen, this tendency in Bright's disease to lessen the frequency of the occurrence of endocarditis in cases affected with valvular disease, prevailed through the whole of the varieties of disease of the valves that we have been investigating, excepting in cases with mitral obstruction.

The Signs and Symptoms of Endocarditis affecting Cases with Valvular Disease.

The signs and symptoms of endocarditis when it occurs in cases of valvular disease of the heart, not affected with acute rheumatism, do not differ essentially from the signs and symptoms of endocarditis, when it attacks cases of acute rheumatism affected with valvular disease of some standing. I have already given a brief clinical history of a series of cases of that class at pages 648–651, and it will, I think, be sufficient if I here refer to the narrative and *résumé* of those cases. As in those cases so in these, the two great distinguishing features of the supervention of endocarditis upon valves already affected with regurgitant or obstructive disease are (1) the great variability of the valvular murmurs, and of the size of the heart, as indicated by the alternate extension and contraction of the area of the impulse, and the alternate increase and diminution of its force; and (2) the great general illness with which the patient is affected, an illness not marked by dropsy, but by elevation of temperature, over-action or failing power of the heart, and pain in the cardiac region, side, or chest, hurried, difficult, and labored respiration, connected often with a congestive affection of the lungs, showing itself sometimes in the form of bronchitis or of pulmonary apoplexy with its attendant pleurisy. I would again refer to the illustrations I have given with regard to those vital symptoms in a previous part of this article.

I would here remark that the occurrence of a special fever, such as enteric fever, may, as we have already seen, suspend a mitral or an aortic regurgitant murmur for a time; but this occurrence proclaims itself by its own distinctive symptoms.

I have not given any account of the temperatures of the body in the above clinical histories of pericarditis and endocarditis; for the thermometer was only employed in the later cases, and therefore in an insufficient number to enable us to arrive at general results.

Endocardit's affecting the Tricuspid Valve.

Endocarditis and structural disease of the tricuspid valve are admitted to be so rare in the adult, that there are few clinical or pathological records describing affections of that valve.

I have examined the whole of the cases of valvular and other diseases of the heart, and of Bright's disease, contained in the post-mortem records of St. Mary's Hospital, from 1851 to 1869–70, with the special object of ascertaining the frequency, extent, and character of any affection of the tricuspid valve that might occur in those cases, and the result is given in the accompanying Table.

Cases with Affection of the Structure of the Tricuspid Valve, not including instances in which the valve was incompetent owing to the great size of the tricuspid aperture; but including all those in which the edges of the valve were thickened, but the function of the valve was unaffected.

		Affected with Bright's disease.
a. Cases with endocarditis, not affected with Bright's disease .	1	1
b. Case with fibrinous concretion on valve	0	1
c. Case with contraction of mitral valve	0	1
d. Cases with thickening and corrugation, or roughness of valve (1 with mitral-aortic reg., 1 with mitral obstr.) . . .	2	0
c. Cases with thickening of valve, valve not incompetent . .	11[1]	7[2]
	14	10

The tricuspid valve was affected with endocarditis in two instances; one of these patients was a woman, aged 40, who had been subject to acute rheumatism when a child, and had palpitation on

[1] Of the 11 without Bright's disease, 2 had mitral, 2 aortic, 3 mitral-aortic regurgitation, 2 mitral obstruction, and 1 had no valvular disease.

[2] Of the 7 with Bright's disease, 2 had aortic regurgitation, and 5 had no valvular disease.

slight exertion. She had been a patient in the hospital ten months previously with dropsy, ascites, albuminuria, and a mitral murmur. The ascites and dropsy disappeared, but they were greater than before when she was readmitted, when the lips and nose were blue; and the urine was scanty and very albuminous. The mitral murmur was louder than before, and dyspnœa appeared in paroxysms. The heart was rather large (12 inches), and presented patches of lymph on its surface; the walls of the right ventricle were half an inch thick, being thicker than those of the left ventricle. Warty, rough, irregular fibrous excrescences were present around the margin of the mitral orifice; looking towards, and being entirely in, the left auricle; the ventricular surface being free from deposit: and there was a smooth fibrinous deposit on the (auricular) surface of the tricuspid valve.

The other case with endocarditis of the tricuspid valve, was a woman aged 42, who had contraction of the mitral orifice, which allowed of the passage of but one finger. The heart was of very great size, and its cavities contained twenty ounces of blood, although it only weighed 13½ ounces. The tricuspid valve had all its flaps thickened with excrescences along their margins, but the valve itself was competent. She became subject to palpitation twelve months previously after a shock or fright. Three days before admission, she raised half a pint of bright blood. The legs and feet were swollen, she had pain in the chest, the heart's action was violent, and there was a confused rumbling sound at the apex. There was no albumen in the urine. She became gradually worse, and finally palpitation and dyspnœa were superseded by drowsiness.

In both of these cases, the right side of the heart was excited to excessive and continuous labor by the diseased condition of the mitral valve, which in one instance was affected with regurgitation, and in the other with great obstruction.

In one remarkable case a large concretion was attached to the tricuspid valve. This patient was a man, aged 69. The heart was large, weighing 16 ounces, the tricuspid valve was universally thickened, and a fibrinous deposit, the size of a nut, was present on the anterior surface of one of the flaps. The tendinous cords were hypertrophied and atheromatous. One of the valves of the pulmonary artery was converted into a hard concrete mass. There is no account of the left side of the heart.

These were all the instances that I can find in which there was endocarditis of the tricuspid valve, or the presence of concretions on its flaps; but the inquiry into the number of other cases in which the tricuspid valve was affected may throw some light on the probable frequency of antecedent endocarditis of the tricuspid valve, as a probable cause of disease of the valve.

I may briefly state that in one case there was a contraction of the tricuspid orifice, so as barely to admit two fingers; and thickening round the margins of the valve; and although the other valves were stated to be healthy, a mitral murmur was audible during life. In another case, with mitral obstruction, the edges of the tricuspid valve were thick and corrugative; and in a third patient, who had been affected with acute rheumatism six months previously, which was followed by mitral-aortic regurgitation, the tricuspid valve, which was not seen, felt rough and thick. These are the only cases that permit definite evidence that in them the tricuspid valve had been previously affected with endocarditis. There were however eighteen other cases, as may be seen in the Table, in which there was some thickening of the tricuspid valve, in two of which it was stated to be atheromatous; but in none of these cases did it appear that the tricuspid valve was incompetent. Twelve of those cases had mitral, aortic, or mitral-aortic regurgitation or mitral obstruction; and of the remaining six cases that were free from valvular disease, five had Bright's disease.

It does not appear to me that any of these cases present definite evidence of the previous existence of endocarditis of the tricuspid valve as the cause of the thickening of its flaps, although it is probable that in some of them the valve had been originally inflamed, and especially in those cases that presented aortic, mitral, or mitral-aortic regurgitation, or mitral obstruction.

TREATMENT OF ENDOCARDITIS.

Endocarditis is so completely an affection associated with those important diseases, acute rheumatism and chorea, in which it is rare, with pyæmia and Bright's disease, in which it is common, and with established valvular disease, that the proper treatment of the parent affection must in all such cases be the proper treatment of the associated inflammation of the valvular structure of the heart. The treatment of those diseases, however, should be modified in the form of additional precautions when endocarditis appears; and the general treatment of acute rheumatism and chorea must, from the first, be mainly governed by the consideration that in both of them endocarditis is the most se-

rious natural complication of the general disease. What I have said with regard to the treatment of acute rheumatism in relation to the prevention of pericarditis, applies also to the treatment of acute rheumatism in relation to the prevention, if possible, and the alleviation of endocarditis. We have already seen that one-half of the first series of cases of acute rheumatism are affected with endocarditis (165 in 325); and that in one-half of the remainder (79 in 164) the occurrence of endocarditis is either threatened (in 63) or probable (in 13). This treatment may be summarized in the brief but effectual rules of (1) the absolute rest of every limb and joint, and of the whole body, during the attack of acute rheumatism; and the maintenance of this absolute rest, especially in the limbs and joints that have been most recently affected, for a period of several days after the complete disappearance of the local inflammation; and (2) the application of the belladonna and chloroform liniment, sprinkled on cotton-wool, over the affected joints, and the support of those joints by the application of flannel over the affected parts so equally adjusted as to give relief and comfort to the patient. We have already seen that the great cause of the inflammation affecting the interior of the left ventricle is the powerful exercise and overwork of that ventricle in maintaining the circulation through the vessels of the inflamed parts, which at the same time call for a greater supply of blood. The fibrous structures of the heart, in common with the fibrous structures of the joints, are prone to inflammation in acute rheumatism; and in the struggle to which the left ventricle is subjected, the valves of that ventricle readily become inflamed at their surfaces and lines of contact. When endocarditis threatens, or first discloses itself, and especially if there be pain in the region of the heart, the application of three or four leeches over that region may be of essential service in lessening the inflammation, and so perhaps permanently saving the valve. It will be well also to cover the region of the heart with cotton-wool, sprinkled with the belladonna and chloroform liniment.

The influence of the treatment of acute rheumatism by means of rest, and the employment of soothing applications and comfortable support to the joints, on the occurrence, severity, and permanent ill effects of endocarditis, will be best illustrated by comparing the clinical history of the 74 cases treated by rest, with that of the 325 cases not so treated.

There was endocarditis alone, or combined with pericarditis, in one-half (161 in 325) of the first series of cases that were not treated upon a system of absolute rest; and in two-fifths (34 in 74) of the series that were so treated.

Valvular disease became established in 43 of the 127 cases (or 1 in 3·1, or 34 per cent.) of endocarditis with a cardiac murmur, including those with pericarditis also (18 in 46), but excluding all those that had previous valvular disease, of the series not treated by rest; and in 3 of the 24 (or 1 in 8, or 12·5 per cent.) of the same kind of cases, of the series that were treated by rest. If we extend the comparison to the whole of both series of cases, excluding those that had previous valvular disease, we find that 43 in 281, or 1 in 6·6, of the series that were not treated by rest, and 3 in 61, or 1 in 20, of the series that were treated by rest, had established valvular disease, indicated by a permanent murmur after their recovery from acute rheumatism, and at the time of their last examination.

There was no murmur, and therefore no valvular disease, when the patient recovered from the attack of acute rheumatism, in 60 of the 127 cases with endocarditis, and without previous valvular disease (or 1 in 2·1, or 44·4 per cent.), that were not treated by rest; and in 17 of the 24 (or 1 in 1·4, or 71 per cent.) of the cases of the like kind that were so treated.

The murmur was lessening in intensity at the time of the last observation, when the patient had recovered from acute rheumatism, in 24 of the 127 cases just spoken of (or 1 in 5·4) that were not treated by rest; and in 4 of the 24 (or 1 in 6) of the analogous cases that were treated by rest.

We here find that, in the series of cases of acute rheumatism that were treated by a system of absolute rest, the proportion of those that were attacked with endocarditis was slightly less than that of those that were not so treated. Thus far the comparison is but slightly in favor of the treatment of acute rheumatism by a rigid system of rest; and this would seem to suggest that a certain, and a very large proportion of cases of acute rheumatism are habitually and intrinsically attacked by endocarditis. When, however, we extend the comparison, and ascertain the proportion in which those cases of endocarditis, not previously so affected, acquired permanent valvular disease, so as to injure health during the remainder of life, and to shorten life itself, we discover that the series of cases not treated by a system of absolute rest were thus permanently injured in a far larger proportion of cases, amounting to more than twice as many, or in the ratio of 8 to 3, than in those that were treated by rest.

If we pursue the inquiry further, so as

to discover the relative extent to which the interior of the heart was inflamed in the two series of cases, we discover that there was but one instance, or 1 in 24, of those with endocarditis and without previous valvular disease, of the series treated by a rigid system of rest, that gave definite evidence of inflammation of both the aortic and mitral valves; while in 19 instances in 127, or 1 in 6·7, of the same kind of cases that were not treated by a rigid system of rest, there was direct evidence of aortic regurgitation. In nine, or rather ten, of those cases that were not treated by rest, there was a mitral murmur, and therefore direct evidence of inflammation of the mitral valve; but in the remaining nine cases there was also evidence of mitral endocarditis in the shape of a tricuspid murmur, or prolongation of the first sound, with intensification of the pulmonic sound, and obstacles to the flow of blood through the lungs. The whole chain of evidence points then, I think, irresistibly to the conclusion that the extent, severity, and permanent ill effects of the endocarditis were much greater in the series of cases that were not rigidly treated by rest than in the series that were so treated.

Pericarditis, also, attacked a much larger proportion of the cases not treated by a system of rest, or 63 in 325, or 1 in 5·2, than of those that were treated by rest, or 6 in 74, or 1 in 12·2. Thus more than twice as many of the former series of cases, that were not treated by a rigid system of rest, were attacked with pericarditis, than of the latter series of cases that were treated by a rigid system of rest.

I am of opinion, however, from a careful revision of the clinical history of those cases, that the treatment by opium, which was pursued in a considerable proportion of the first series of cases that were not treated by rest, had some influence in increasing the frequency and severity of inflammation of the heart, and especially of its exterior. Taking this into account, however, I consider that the clinical evidence here afforded shows, that the severity and permanent ill effects of endocarditis, and the frequency and severity of pericarditis, are greatly lessened by a system of treatment by rest absolutely maintained; and combined with the use of local means in the shape of the application of the belladonna and chloroform liniment, and of equal and comfortable support to the affected joints, and the employment of leeches applied over the region of the heart, when that organ was attacked by inflammation, and especially on its exterior, and when accompanied by pain.

The clinical evidence in favor of the treatment of acute rheumatism by rest is conclusively supported on the pathological grounds stated at the commencement of this article (see page 618), and in Dr. Moxon's very striking, important, and convincing lecture on endocarditis, to which I have there referred. We have there seen that the surfaces or lines of contact, pressure, and friction of the valves, and chiefly of the mitral valve, are especially affected with endocarditis. Thus the overwork of the left ventricle of the heart, and the resulting friction, pressure, and tension of its valves, in cases of acute rheumatism and chorea, tend to augment the primary influence of the parent disease, and to excite and intensify the inflammation of the interior of the heart, and especially of the mitral valve.

CARDITIS.

By W. R. Gowers, M.D.

Synonyms.—Myocarditis; Interstitial Myocarditis.

Definition.—An acute affection of the walls of the heart, consisting in interstitial serous exudation or cell-infiltration, and degeneration of the muscular fibres. The latter may occur without any change in the interstitial tissue. This has been regarded as a "parenchymatous myocarditis." But this change, when general throughout the heart, occurs as the result of some general blood state, and is unassociated with other evidence of inflammation in the heart or remaining organs. Without denying the possibility of the occurrence of a general parenchymatous inflammation of the heart, it seems more consistent with the relations of the process to consider these cases as examples of acute degeneration. (See Art. "Fatty Degeneration.")

Varieties.—The inflammation may be general, affecting all parts of the heart; or it may be partial, being limited to a small area. When general it may be diffused uniformly through the heart; it may affect the superficial layers only (when secondary to pericarditis); or it may result in scattered foci of suppuration. Circumscribed inflammations may result in the formation of an abscess in the wall of the heart. Lastly, the varieties have been distinguished of primary and secondary inflammation; the former occurring apart from, the latter in consequence of, preëxisting disease, general or local.

Etiology.—In the consideration of the causes of the disease, the variety which is due to the extension of inflammation from the pericardium may be excluded from consideration, since it owns the same causes as the pericarditis to which it is due, and is commonly the consequence of acute rheumatism. Other forms of carditis occur in the male much more frequently than in the female sex; and at all ages, but rather more frequently before than after thirty years of age. As a primary affection, carditis is extremely rare: a few of the recorded cases have been ascribed to exposure to cold after severe exertion, or to blows on the precordial region. In other cases no exciting cause could be discovered. As a secondary affection it has occurred in a few cases of acute rheumatism, apart, it is said, from endo- or pericarditis, and also in various septicæmic affections. Its chief local causes are pericarditis, endocarditis in rare cases, embolism, and growths in the heart.

Pathological Anatomy.—The inflamed muscular substance is at first injected, and then swollen and softened. Points of extravasation are scattered through it; the tissue becomes paler, of a reddish-gray tint, and may break down into a pulpy mass, partly from the acute degeneration and destruction of the muscular fibres, and partly owing to their separation by an interstitial infiltration of serum, blood-corpuscles, and corpuscular inflammatory products, derived from the interstitial connective tissue-elements or from the blood. These may be in the form of pus cells, which may be disseminated through the heart in the tracts of connective tissue, or may be aggregated in minute abscesses. In the localized form of inflammation, softening and breaking down of tissue may occur without actual pus formation, and a pseudo-abscess may result. If pus cells are formed, a true abscess of the heart is the consequence, and the destruction of the muscular fibres may be so complete that only pus may be found in the cavity. The adjacent tissue is, however, softened and degenerated. Such an abscess may attain the size of a hazel-nut. This local inflammation is much more common in the wall of the left than in that of the right ventricle, and is very rare in the auricles. It is most common in the left ventricle near the apex, in the posterior wall, or in the septum. When softening, purulent or non-purulent, has occurred, the wall is bulged at the spot, and secondary pericarditis may be produced. When the inflammation is adjacent to the inner surface, it may invade the endocardium, and spread to an adjacent valve. Ultimately, in most cases, the outer or inner wall of the abscess or pseudo-abscess gives way, and the contents escape into the pericardial cavity or into the ventricle; causing, in the former case, purulent pericarditis, in the latter, an "acute aneurism of the heart," and septicæmia, usually fatal in a few hours. Both walls have given way at the same time, and "rupture of the heart" has occurred. An abscess in the septum has burst into both ventricles; from the upper part of the septum it has burst into the aorta behind the aortic valves, or into the right auricle. In this way a fistulous communication has been established between the two ventricles, between either or both ventricles and the aorta, or between the left ventricle and the right auricle. If the inflammation subsides without the formation of pus, the cellular products may develop into fibrous tissue. This often occurs in the superficial layers of the heart after pericarditis, and it may occur in the localized form of carditis, a circumscribed patch of fibrous tissue resulting. Less commonly caseation takes place, even after pus has been formed, and the caseated mass may shrink and calcify.

Symptoms.—The symptoms of acute inflammation of the heart are sometimes distinct enough, but are in other cases obscure or misleading. The local signs are those of cardiac weakness, suddenly developed, after, it is said, a transient stage of excitement. The impulse is weakened or imperceptible; the first sound toneless. A systolic murmur has been heard in some cases, due, perhaps, to incapacity of the papillary muscles. The cardiac dulness is normal, or sometimes widened, from acute dilatation. The pulse is feeble, frequent, and may be irregular. Uneasiness about the sternal or cardiac region has been an early symptom in several cases, increasing in some to acute pain. The general symptoms are those of heart failure, and those which depend on cerebral anæmia may be so pronounced as entirely to obscure the real nature of the case. Dyspnœa is the most

constant symptom, continuous or felt on the slightest exertion. Nausea and vomiting, collapse, with coldness of extremities, and clammy perspiration occur. Convulsions, delirium, and coma have been prominent symptoms in several cases. The central temperature is raised; in one recorded case it reached 107°. The symptoms of collapse rapidly increase, and death occurs usually in a few days. Friedreich found the average duration to be four days, the minimum being a few hours, the maximum a week.

Localized inflammation of the heart may be attended by similar but less urgent symptoms, or may run an entirely latent course until the occurrence of the grave symptoms which proclaim the rupture of an abscess, such as, on the one hand, those of acute pericarditis, or on other, those of systemic or pulmonary embolism. In one case a pustular rash occurred, it is conjectured from embolism of the cutaneous arteries.

Diagnosis.—The diagnosis is a question rather of theory than of practice, for the disease is extremely rare, and its symptoms are produced by many other causes. The sudden onset of symptoms of cardiac weakness and failure, less sudden than in cases of rupture, more sudden than in cases of acute degeneration, if coupled with considerable elevation of temperature, and especially if occurring in the course of a disease such as pyæmia, may give rise to a suspicion of the existence of carditis. Abscess of the heart is even more equivocal in its symptoms. The rupture of an abscess may be suspected if sudden symptoms of systemic or pulmonary embolism or of pericarditis, supervene on less urgent symptoms of cardiac failure.

Prognosis. — General carditis has hitherto only been diagnosed after death, and it is doubtful whether recovery has ever taken place. In the circumscribed form it is probable that subsidence of the inflammation has, in a few cases, permitted the continuance of the heart's action and the disappearance of the symptoms. The prognosis in the form which is secondary to pericarditis is much less grave, since a large proportion of the muscular tissue is not damaged, and, with the subsidence of the adjacent inflammation, recovers good functional power.

Treatment.—The treatment of carditis is necessarily symptomatic. Its existence can rarely be ascertained, and, if known, no means of direct treatment exists. Rest to the heart is the first point to be secured. Cold to the precordial region has been recommended; warm poultices would perhaps give more relief. Warmth should be applied to the extremities, to equalize the circulation and lessen the tendency to correlated congestion of internal organs. The heart's action must of necessity be sustained by stimulants which, with the recumbent posture, constitute the best treatment for the cerebral symptoms. For the cardiac failure in septicæmia, full doses of the perchloride of iron have seemed to the writer to be of distinct service.

HYDROPERICARDIUM.—HYDROPS PERICARDII.

By J. Warburton Begbie, M.D.

The occupation of the pericardial sac to a greater or less extent by serous fluid, a condition known under both of the terms mentioned above, or simply as Dropsy of the Pericardium, is not of unfrequent occurrence. Laennec indeed speaks of this condition as being very common. "L'hydro-péricarde," he says, "ou l'accumulation d'une quantité plus ou moins grande de sérosité dans le péricarde, est un cas extrêmement commun;"[1] but he qualifies this statement by the remark, that idiopathic effusion into the pericardium is very rare, that ordinarily but a few ounces of serum are found in the sac, and that this quantity is effused shortly before death, sometimes at the very moment of dying, or even immediately thereafter. The causes of dropsy of the pericardium are various, and some of them most obscure. Dr. Walshe recognizes an *Active* and *Passive* Hydropericardium; also, a third form dependent on *mechanical obstruction*.[1] The first of these three varieties is very rare. Dr. Walshe,

[1] Traité de l'Auscultation. Des Maladies du Cœur, chap. xxii.: De l'Hydro-péricard.

[1] Diseases of the Heart, p. 266.

however, refers to certain instances of Bright's disease, in which he has known the pericardium fill with fluid, the symptoms indicating an irritative state, while the signs of pericarditis were wanting. Examples of a precisely similar kind are familiar to the writer, in connection with the dropsy of scarlet fever. He has seen a sudden and copious effusion into the pericardium occur at the same time that dropsical swelling manifested itself in the more ordinary situations, and in such cases, found no evidence whatever of plastic formations either upon or within the heart.

Passive Hydropericardium is seen in connection with other dropsies, with anasarca and ascites, but especially with hydrothorax. The relation of the latter, however intimate, as in some cases it is, to pleural dropsy, is by no means constant. On two occasions we have found a very large Hydropericardium in cases of primary cardiac disease with great anasarca, but with little, if any, hydrothorax.

Mechanical Hydropericardium. — An effusion of serous fluid into the cavity of the pericardium has been found in connection with aneurism of the aorta, with cancerous disease seated in the anterior mediastinum, exerting injurious pressure on the great venous trunk, and thus preventing the due return of blood through the coronary and pericardial veins, and certain morbid states of the heart itself, in which the venous circulation is greatly embarrassed. In such instances the dropsy, evidently due to direct obstruction near its seat, may with great propriety be called mechanical.

The serous fluid which occupies the pericardial sac is sometimes colorless; at other times, although quite limpid, and without any admixture of albuminous floculi, it presents a lemon yellow, or even rose-colored tints; rarely is it sanguinolent. The quantity of fluid varies greatly. Usually it is not excessive, but, on the contrary, moderate. In Passive Hydropericardium, Dr. Walshe has stated the amount to be from eight to twelve ounces; more than the latter quantity he has never seen. Instances, however, are on record in which a very large accumulation of serous fluid has been found in the pericardium. Benisart has related one, in which there existed four pints, or eight pounds (*huit livres*). From twelve to eighteen ounces of fluid can be injected into the healthy pericardium of an adult, but there can be no doubt that the pericardium, contrary to what is stated in certain anatomical treatises,[1] is extensible; the fibrous, as well as serous nature of the membrane may impair, but does not prevent its extensibility. In all probability, those cases of enormous distension of the sac by fluid, which are described by Corvisart, Avenbrugger, and others, were examples of pericarditis. It is apparently when altered by inflammation that the pericardium becomes most capable of distension.

Dr. Stokes refers to a case published by Sir Dominic Corrigan, in which the heart was covered with a pulpy lymph, and there was a vast effusion of liquid into the sac,[1] and Dr. Graves, in describing a most interesting case of Hydropericardium, connected with malformation of, and recent deposition of lymph upon the pulmonary valves, makes the remark, "the pericardium was distended with straw-colored fluid, so abundant that we expected to find pericarditis;"[2] implying that this distinguished physician regarded pericarditis as the usual determining cause of *large* pericardial effusions.

The most important and reliable indications of the existence of Hydropericardium are furnished by percussion and auscultation, but independently of these, there are other particulars, the value of which is by no means small. A sensation of discomfort in the region of the heart is frequently complained of, and even a sense of weight—a symptom of pericardial effusion to which Lancisi attached great significance. Senac describes the undulatory movement of the fluid as visible between the third, fourth, and fifth ribs; and Corvisart, the sense of fluctuation in the same situations, as distinguished by touch. Dyspnœa, more or less urgent, is usually present in cases of Hydropericardium. It must, however, be admitted that there exists no small amount of difficulty in assigning the true share in the production of this symptom to the effusion within the pericardium, seeing that it may in most cases be in part likewise attributed to the visceral disease, on which this form of dropsy depended, or possibly to the hydrothorax, by which it is so likely to be accompanied. A feebleness of the pulse, and, not unusually, an intermittent or irregular condition of the pulse exists. By auscultation, the heart-sounds are feebly audible, and appear to be distant or remote. On percussion there is extended pericardial dulness, for the most part not rising so high, nor passing to the same limits laterally, as is the case in chronic, and even in some instances of acute inflammatory effusion within the pericardium. The dilatation of the precordial region, or even of the left lateral region, as noticed by Louis, the epigastric tumor described by Corvisart, and the ex-

[1] *E. g.*, Holden's Illustrated Manual of Anatomy, p. 98.

[1] Diseases of the Heart, p. 20.

[2] Clinical Lectures: Pericarditis, p. 578.

tension of the left lung upwards, of which Dr. Graves and Dr. Stokes have written, are rare but striking phenomena connected with large pericardial effusions, dependent, however, on inflammatory action. Besides the general symptoms to which reference has been made, it must be held in view that others of the same nature will be likely to show themselves, the latter, however, having a more distinct relationship with the visceral disease on which the dropsy depends. The Hydropericardium, moreover, will in all probability be connected with some other dropsical effusion, hydrothorax or anasarca, or it may be ascites.

The remedies most useful in the treatment of dropsies are seldom effectual in relieving the dropsy of the pericardium. The writer has known the repeated application of blisters over the region of the heart to produce a decided impression in one case. The stronger diuretics and hydrogogue cathartics, "will," as Dr. Walshe observes, "be tried, were it only for the removal of the usually concomitant dropsies." Paracentesis pericardii, which has been repeatedly performed in the treatment of pericarditis attended by large effusion, and in some instances successfully performed, is of course an available means for affording temporary relief in the truly dropsical affection, temporary because, although the heart be freed by the operation from the surrounding fluid, unless the disease giving rise to the dropsy be removed, the fluid must necessarily re-accumulate.

ANGINA PECTORIS AND ALLIED STATES; INCLUDING CERTAIN KINDS OF SUDDEN DEATH.

By Professor Gairdner, M.D.

The phenomena of the disease, or group of symptoms, termed Angina Pectoris by Heberden, are perhaps the most interesting in themselves, and the most deserving of study in relation to other forms of cardiac disorder, of any which we shall have to consider in this section. In treating of this difficult subject, we must separate with great care the essential facts of the disease from the various speculations, or associated ideas, that almost inevitably force themselves into the mind in considering the facts. And this separation is by no means easy; for in this instance the facts themselves are apt to be more or less withdrawn from exact observation; the phenomena characteristic of the disease being mostly *subjective*, i. e., present to the consciousness of the patient only, and only through his description of them made known to the physician. It may even be said with truth, that no one fact in a typical case of angina pectoris is necessarily other than subjective, with the exception of the awful terminal fact of sudden death. And when this is wanting, or when it is delayed, there is hardly any combination of the remaining symptoms that may not vary in individual cases, or be differently presented by the sufferer, according to the exactness and concentration of his habits of thought, the vividness and power of his imagination, or the degree and kind of his individual sensitiveness to morbid impressions.

Still, the fact of sudden death, superadded to the evidence of certain sensations preceding death, may be considered to afford the nearest approach we have to an accurate definition of this disease. What these sensations are we shall endeavor to indicate, in so far as the inadequacy of language will allow, from the consideration of such individual instances as have been minutely and carefully recorded either by the sufferers themselves, or by physicians simply giving expression to the spontaneous testimony of their patients. By following the ideal descriptions of those who have allowed themselves to be guided by theories of the disease rather than by the facts, we might easily add to the fulness without increasing the value of our description.

First on the list of symptoms, according to Heberden and the majority of authors who have followed him, is *pain*.[1]

[1] In his Commentaries (1796), Heberden treats of this disease under the general title "De dolore pectoris" (Sec. lxx.). In his first communication on the subject to the College of Physicians in 1768 (Medical Transactions, vol. ii. p. 59), he merely terms it "A Disorder of the Breast." The two descriptions do not differ in essentials, but a few details of difference which seem to be of more or

How far pain, in the ordinary sense of the word, is essential to the idea of angina pectoris, we shall afterwards consider; for the present it may be sufficient to observe that pain, or at least a sensation of local distress amounting in certain cases to pain of a peculiarly overwhelming character, is in this disease closely associated with the symptoms immediately preceding death.

This peculiar anguish, or, as it might justly be called, agony of suffering, is paroxysmal; it frequently reaches its climax within a few minutes, and is relieved or disappears entirely within a like period of time, or at most within an hour or two; it recurs at uncertain intervals, sometimes without any obvious exciting cause, at others manifestly determined by exertion, and especially by too rapid walking up-hill, in which case it often ceases, especially in the earlier attacks, almost immediately on standing still: it is instinctively associated in the mind of the patient with the idea of a particularly severe form of oppression or suffocation; or rather to be more exact, with some indefinable sense of impending danger, to which he is unable to give expression, and which he endeavors to convey to others by similitudes that do not satisfy his own mind. A frequent expression is that recorded by Dr. Latham in the case of a very eminent man of the highest intellectual power; after an attack he said he "could scarcely bear it if it were as severe as it had been;" and shortly afterwards, "One can bear outward pain; but it is not so easy to bear inward pain."[1] This essential *unbearableness* of the suffering is most characteristic of angina pectoris, and it is quite independent of the degree of severity of the pain in other respects. And further it is to be observed that the intolerance here alluded to is not the mere impatience of the nerves, which can be mastered by a strong will and a calm heroic self-restraint; it is the sense that the very springs of life are implicated, and that under a prolongation or increase of the pain the whole machine must suddenly give way.[2] It is from this sense of impending death (rarely thus expressed in words by the patient), and from the fact that sudden death actually occurs during the paroxysm in a certain number of cases, that the pain, or special sensation, of angina pectoris derives almost all that it has of a distinctive character; and therefore Dr. Latham has justly elevated this most important but almost indescribable symptom to a co-ordinate rank with the pain itself, in his description of the disease as a whole. Angina pectoris, according to his admirably succinct definition, "consists essentially of pain in the chest and a sense of approaching dissolution." "The subjects of angina pectoris report that it is a suffering as sharp as anything that can be conceived in the nature of pain, and that it includes, moreover, something which is beyond the nature of pain, a sense of dying."[1]

Such, then, are the most important or essential facts which clinical observation teaches in reference to angina pectoris. Let us now consider them separately, and more in detail.

The *pain* of angina is usually felt at the lower sternum, but sometimes also under the middle or upper sternum, inclining, however, towards the left side.[2] Sometimes the pain extends to both sides of the chest in front, and perhaps more frequently into both shoulders, and into the back. Very specially characteristic is a "pain about the middle of the left arm,"[3] sometimes present in the right, or in both arms, which, according to Dr. Heberden, occasionally precedes,[4] but more commonly follows the pain in the chest. This, together with a degree of numbness of the left arm, may be described as present in the majority of cases in which the pain extends beyond the thorax.[5] The pain and numbness to-

less importance will be noticed below. The eminently careful and exact use of language by Heberden in his singularly condensed clinical studies, whether in Latin or in English, tends to invite attention to even the minutest discrepancies between his earlier and later statements.

[1] Latham, "Diseases of the Heart," vol. ii. pp. 375–76. It is no secret, that the case was that of the late Dr. Arnold, of Rugby.

[2] "Qui hoc morbo tenentur, occupari solent . . . ingratissimo pectoris angore, vitæ extinctionem intentante, siquidem augeretur, vel perseveraret."—Heberden, *Comm.* loc. cit.

[1] Op. cit. pp. 366, 364.

[2] "*Always* inclining more to the left side." (Heberden, Med. Trans.) "*Non raro* inclinatior ad sinistrum latus."—*Comment.*

[3] Heberden, Med. Trans. uti supra. "Dolor sæpissime pertinet a pectore usque ad cubitum lævum. . . In nonnullis vero . . . ad dextrum pariter ac lævum cubitum pertigit, atque etiam usque ad manus; sed hoc rarius evenit; rarissimum autem est, ut brachium simul torpeat ac tumeat."—*Comm.* loc. cit.

[4] Med. Trans. vol. iii. p. 3.

[5] The group of symptoms here alluded to, though first clearly indicated by Heberden as characteristic, was described long before by Morgagni in the case of a woman, forty-two years of age who died suddenly during a paroxysm, and was found to have a dilated and ossified aorta. The description is worth quoting, from the fact that it is probably one of the first clinically exact records existing in medical literature of a case of this kind. The patient had been "diu valetudinaria, diuque obnoxia paroxysmo cuidam ad hunc modum se habenti. A concitatis corporis motibus

gether, or the pain alone, may extend down to the fingers, or may stop short at the elbow, usually at the inner side of the arm; and painful sensations, more or less definite in character, may be felt also in the neck, or in one or both lower extremities; but these are exceptional, and there is reason to think that in some cases the local symptoms connected with aneurismal tumors implicating the nerves may have been confounded with those more specially characteristic of angina pectoris.[1] At all events, these local varieties of pain are not to be regarded as essential elements of the disease, although from their occurrence and their distribution they may lead to more defined conceptions of the nervous plexuses involved, and thus occasionally to the detection of an organic cause, or of something tending to throw light upon the peculiarities, or to guide the treatment of an individual case.[2]

Local tenderness on pressure is an occasional but by no means a constant symptom of angina pectoris. Sometimes, on the other hand, the pain is decidedly relieved by pressure, or by rubbing, as well as by counter-irritation of the parts affected. The pains are aggravated by movement of the whole body, and especially by severe or even moderate exertion in walking, which indeed becomes impossible during a severe paroxysm. Very marked relief is often afforded by the eructation of wind from the stomach, whether spontaneously or under the influence of carminatives. Rest of body, and warmth to the extremities, are among the more obvious of the physiological conditions which have been observed to have a well-marked effect in relieving the pains of angina, in their less extreme varieties.

The *peculiar sensation which culminates in the sense of impending death*, has been very variously described,[1] and indeed seems from its very nature to be almost indescribable. Among the uninstructed, or in the case of persons unaccustomed to observe and analyze their own sensations, nothing is more common than to find the term "breathlessness," or "want of breath," applied to every kind of thoracic oppression, and the sense experienced in angina pectoris of constriction, or in other cases of repletion in the chest, accompanied as it usually is by gasping or irregular respiration, is undoubtedly often called a want of "breath," or "suffocation," by persons who are simply feeling about, as it were, for an expression whereby to represent an uncommon and intensely oppressive sensation. A similar confusion lies latent even under the more technical language of Heberden, in his use of the Greek

ingruebat molestus quidam angor intra superiorem thoracis sinistram partem, cum spirandi difficultate, et sinistri brachii stupore: quæ omnia, ubi motus illi cessarent, facile remittebant. Ea igitur mulier, cum circa medium Octobrem A. 1707 Venetiis in continentem trajecta, rheda veheretur, lætoque esset animo, ecce tibi ille idem paroxysmus; quo correpta, et mori se, aiens, ibi repente mortua est." The examination after death showed disease of the aortic orifice and aorta, and Morgagni regards the sudden death as due to the sudden excitement of carriage exercise ("insolitum in Veneta fœmina rhedæ motum") operating upon a circulation weakened and obstructed by chronic disease, as to lead to ultimate failure in the power of the heart to propel the blood ("ut sanguis restitans promoveri amplius non poterat").—*De Sedibus et Causis Morborum*, ii. *Epist.* xxvi. 31 et seq.

[1] As, for instance, in several of the cases recorded by M. Trousseau in his interesting chapter on the subject. (Clinique de l'Hôtel-Dieu, vol. ii. p. 434 et seq. deuxième édition; Paris, 1865.) English Translation, 1868, vol. i. p. 596 et seq.

[2] In one very exceptional case, recorded by Heberden in the "Commentaries," there was no pain complained of in the chest, but only in the left arm, having, however, in other respects the characters of angina. After fifteen years of occasional and increasing suffering, the patient died at seventy-five years of age.

[1] It is difficult to judge from Heberden's descriptions how far the "angor pectoris, intentans vitæ extinctionem," was regarded by him as a simple pain. In his first memoir he speaks of the "sense of strangling, and anxiety with which it (the disorder of the breast) is attended," and applies the name Angina Pectoris on account of these characters rather than on the ground of pain. The anonymous patient who described his own case in the third volume of the "Medical Transactions," apparently discriminates very sharply, on the one hand between the pain in the left arm and chest, coming on "when walking, always after dinner, or in the evening;" and on the other, the "sensations which seem to indicate a sudden death;" which he describes as being like "a universal pause within me of the operations of nature for perhaps three or four seconds," and afterwards "a shock at the heart, like that which one would feel from a small weight fastened to a string to some part of the body, and falling from the table to within a few inches of the floor." This distinction of the sensation of impending death from the pain was unfamiliar to Heberden, who says he does not remember to have heard it mentioned by any other patient; and thinks that the sudden death of this patient, which came to his knowledge afterwards, was connected more with the pain than with this peculiar sensation. Dr. Parry speaks of the first symptom in angina pectoris as "an uneasy sensation, which has been variously denominated a stricture, an anxiety, and a pain." Dr. Latham was probably among the first to define the sense of impending death as being distinct from the pain.

term Angina,[1] which, according to its etymology, signifies a *strangling*, and according to its actual and primitive use was applied chiefly to certain affections of the throat, occasionally leading to sudden death by laryngeal suffocation, and giving rise to a sense of choking, or of constriction in the fauces. Yet Heberden, in using this term, had thoroughly realized the fact that angina pectoris is not really a suffocation or a breathlessness, in the ordinary acceptation of these terms. At most it is a sensation which by its urgency and oppressiveness recalls the impression of suffocation, and which may in certain cases be associated with true dyspnœa, or still more frequently with orthopnœa. In many instances, however, careful examination shows, and the patients themselves may be easily convinced, that respiration is really not impeded; that inspiration and expiration are alike free and noiseless; that the air is taken into the chest in full measure, and (in so far as the evidence of stethoscopic examination goes to prove the fact) that the mechanical renewal of the air in the vesicles of the lungs is perfectly accomplished. In this sense, the observation of Heberden is profoundly exact, that in the beginning of this distemper the patients "nulla tenentur spirandi difficultate, a qua hic pectoris angor prorsus est diversus."[2] And yet it might possibly be maintained that in a more transcendental sense respiration, *i. e.*, the chemistry of respiration, is usually impeded; that the transit of the blood through the pulmonic capillaries is for the time suspended or impaired, that the right heart is perhaps unduly loaded, and that the sensation of "breathlessness" is therefore not without a physical equivalent in the state of the blood, for the time restricted in its supply of oxygen. In the more advanced stages of angina pectoris, indeed, especially when in connection with organic disease, it rarely happens that some positive evidence of real dyspnœa does not exist, at least as a complication, if not as a part of the disease. Even in such complicated cases, however, it is usually easy for the experienced clinical observer to detect a difference of habit and aspect from cases in which the breathing is primarily impaired, *e. g.* as in aggravated cases of emphysema with bronchitis, or of double pneumonia, or extensive pleuritic effusion unconnected with a cardiac cause.

We are obliged, therefore, under these circumstances, to accept the necessary limitations of ordinary language in conveying extraordinary or almost indescribable impressions. It is certain that the patient in angina pectoris has a sense of obstruction in the thorax so overwhelming and so full of apparently imminent danger that he instinctively likens it to a suffocation;[1] yet it is equally certain that in many cases impeded respiration, in the ordinary sense of the term, is not present. This remarkable sensation, which is sometimes represented as a tightness or constriction, sometimes, on the other hand, as a fulness or over-distension of the chest, contributes even more than the pain to the indescribable *anguish* of angina pectoris; and it is this sensation especially which gives to the pain its peculiar character of "unbearableness" already noticed; this also, which carries with it in its graver forms that impress of immediately impending death, by which the real danger, and the ultimate probable event, are rendered so vividly present to the consciousness of the patient.[2]

[1] From ἄγχω, *strangulo*, whence also the compound words Cynanche and Synanche, and the Latin verb *angere*, which acquired the secondary sense of undefinable distress conveyed also by *anxietas*, and still more by our English word *anguish*.

[2] Heberden, Comment. loc cit. "Have no *shortness of breath*." (Med. Trans. uti supra.)

[1] "A *sense* of dissolution, not a fear of it," said one of the most gifted men I ever knew, and one most competent to analyze sensations.—J. R. R. Editor.

[2] A recent medical observer, himself a sufferer from angina, whose case will be referred to again in the section on treatment, has contributed what is perhaps the only really exact description in medical literature of one form of the constrictive sensation: "The front of the chest seemed to be bulged out in a convex prominence, which suddenly terminated at the lower end of the sternum in a sharp and deep depression towards the spine. This was a purely subjective phenomenon. There was no contraction of the diaphragm, and no retraction of the abdominal walls. But though the hand laid upon the parts convinced my mind of their normal condition, it in no way modified the sensation." (Dr. W. Herries Madden, in the Practitioner, vol. ix. 1872, p. 334.) In the case of John Hunter, to be cited below (a case of instruction in detail as to many phases of disease included in the present article), the sense of thoracic constriction in one attack was preceded for a fortnight by symptoms of "nervous irritation" in the left side of the face and head, as well as down the left arm. The special sensation in the chest in this case was a "feeling of the sternum being drawn backwards towards the spine, as well as of oppression in breathing; *although the action of breathing was attended with no real difficulty.*" (See *infra*, p. 683.) The special character of the breathing in Hunter's case, elsewhere alluded to, will be found to be a most exact anticipation of what has since been called "ascending and descending," or by some, "suspirious" respiration; a form of disturbance frequent in cases of angina, though it seems to have escaped Heberden's observa-

The other symptoms of angina pectoris have been variously described; so variously, indeed, as to lead to a suspicion of inaccuracies of detail on the part of individual observers of the paroxysm. On all hands it is agreed that in the intervals the patient may have all the appearances of perfect health; his color may be good, his appetite unimpaired, his breathing apparently natural, the action and sounds of the heart perfectly normal. It is equally certain that the paroxysm itself is unattended by fever, and that in uncomplicated cases it has none of the characters, as it has none of the consequences, of an inflammatory seizure.[1] But it is difficult to accept without hesitation the statement of some authorities, that throughout the attack the pulse may be entirely undisturbed either as to its rate of frequency, or as to its characters.[2] In most of the cases in which details have been carefully given, the pulse, at the height of the seizure, has been found small, often imperceptible or irregular in rhythm, but not necessarily accelerated, and sometimes morbidly slow; the countenance has been pale as death, the features pinched and anxious, the extremities cold; there has been often a cold sweat on the brow, sighing or interrupted respiration, and other signs of approaching syncope. On the other hand, it must be admitted that in a few instances the heart has been heard beating in the very midst of a paroxysm without appreciable alteration in the character of the sounds and impulse, and the pulse has been also said to be regular, and neither rapid nor weak. The senses and the consciousness have also been observed to be frequently quite entire in the midst of the paroxysm, though this fact also, like some of the others above mentioned, must be held as subject to numerous exceptions. On the whole, the strict analogy between the phenomena of angina pectoris and ordinary syncope cannot be unreservedly maintained, notwithstanding the arguments of Dr. Parry,[1] who, however, has undoubtedly marshalled a strong array of facts and reasonings in favor of this view of the case. The paroxysm of angina pectoris remains, after all, a mode of morbid function *sui generis*, although in some instances the manner of death in the paroxysm is more or less allied to syncope.

The condition of the nervous system, and especially of the brain and spinal cord, in angina pectoris, opens up many very difficult, and at present even insoluble problems connected with its ultimate pathology. For practical purposes it is sufficient to state the facts established by clinical observation. While it is quite certain, as stated above, that integrity (in a practical sense) of the nervous functions may be maintained up to the very instant of death in certain cases of angina, it is equally well ascertained that in other instances giddiness, vertigo, disorders of the special senses, spasms, tonic and clonic, and almost every kind of disorder of the general sensibility and consciousness may occur, and may also be the distinguishing features of particular paroxysms in persons in whom at other times paroxysms may occur devoid of all such phenomena. It is probable that in some of these forms of the disease the cerebro-spinal complications may be determined by special derangements of the circulation within the cranium, or even by disease of

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tion. See also the remarks on the case of Seneca, below; and at page 683, *note*.

[1] Dr. Latham has admirably *modernized* Heberden's arguments on this point. (Op. cit. vol. ii. p. 383.)

[2] "The pulse is, *at least sometimes*, not disturbed by this pain." (Heberden, Med. Trans.) "Arteriæ eorum, qui in hoc dolore sunt, naturaliter prorsus moventur. . . . In ipsa accessione pulsus non concitatur." (Comment. loc. cit.) Several authors have followed Heberden here without observing that his real meaning is not that there is no alteration of the pulse, but that there is no excitement of it, *i. e.* that the pulse is not *quickened* ("non concitatur") as in inflammation. Dr. Parry, regarding the disease as a syncope, speaks from another point of view, and has no difficulty in showing that the pulse, though not always greatly disturbed, "becomes more or less feeble according to the violence of the paroxysm." Such personal experience as I have on this point leads me to agree with Dr. Parry. The recent experiments and sphygmographic tracings of Dr. Lauder Brunton will be discussed in connection with the pathology of the disease further on.

[1] His expressions are as follows: "From the preceding observations, I think it evidently appears that the Angina Pectoris is a mere case of syncope or fainting, differing from the common syncope only in being preceded by an unusual degree of anxiety, or pain in the region of the heart, and in being readily excited during a state of apparent health, by any general exertion of the muscles, more especially that of walking." (Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris, &c., p. 67.) To the points of difference here noted must be added the persistence of the sensibility up to the very instant of death in many cases of angina pectoris, and the incomplete extinction of the pulse; while in ordinary syncope (as for example from emotion, or from hot rooms) the most absolute temporary insensibility, with a radial pulse which cannot be felt, and respiration just sufficient to maintain life, may occur as symptoms and be maintained for some minutes, with almost no danger to life.

the arterial system extending to the brain; but there are very rarely any permanent changes, either of structure or of function, tending to throw light on these attacks. On the other hand, it seems premature to infer, with Trousseau, the existence of any distinct relation between epilepsy as a predisposing cause, and angina pectoris; still more premature to affirm that "in certain cases, and perhaps in a considerable number, the *angor pectoris* is one expression of this formidable and cruel disease, a phase of its vertiginous form, or in two words an *epileptiform neuralgia.*"[1] The extreme rarity, on the one hand, of true angina pectoris among the countless multitudes of confirmed epileptics, on the other of genuine and well-formed epileptic attacks among the subjects of angina pectoris, seems to oppose a considerable difficulty in the way of accepting M. Trousseau's hypothesis. That the relation, however, between the occasional cerebro-spinal symptoms in these cases, and the cardiac disorder, is more than a coincidence, is shown by the fact that a very similar series of symptoms is observed in some cases of fatty heart; and the author of this article has in more than one instance observed like phenomena in connection with large aneurisms within the thorax.

In certain cases of angina pectoris, more especially when perfect rest cannot be obtained during the attacks, they are apt to be attended by more or less of sickness, and even of vomiting; but these symptoms are rarely obstinate. Flatulence has been already noticed as a frequent accompaniment of the paroxysm, the discharge of the imprisoned air by the mouth usually giving marked relief. In some instances the close of the paroxysm is accompanied or followed by a copious discharge of watery urine, as in hysteria. In one case Dr. Walshe has observed tetanic spasms, with complete opisthotonos, followed by local tonic spasms continuing for some hours after the paroxysm.

The *diagnosis* of angina pectoris is not very difficult in severe cases, except in so far as difficulties may arise from the inability of the patient to express his sufferings in words, or on the other hand from the too fluent and misleading descriptions of comparatively insignificant pains referred to the heart, by persons either unduly frightened or unduly sensitive. Persons who have lost near relatives or even intimate friends, by sudden death of cardiac origin, are extremly apt to be terrified by nervous symptoms of this kind; gouty and rheumatic sufferers are frequently a prey to flying pains which now and then occupy the habitual seats of angina pectoris, and which sometimes give rise to alarms not justified by the event, all the more when suspicion has been once aroused, and when, as happens not unfrequently, the physician as well as the patient may be for some time in doubt as to the cause of the symptoms. Disorders of the stomach, and still more notably of the uterus, frequently lead to pains in the left side, which may pass for cardiac angina. Intercostal neuralgia may have many causes, and not unfrequently radiates towards the left arm. In hysterical and romantic girls, pains about the heart are often associated with palpitation and irregular sighing respiration, sometimes also with well-marked irregularities of cardiac rhythm, or with murmurs requiring care in their discrimination, though not, on the whole, very apt to lead into serious error. Each of these cases requires its own special diagnosis, with reference to the cause of the symptoms; and it should always be remembered that the number of persons presenting themselves on account of such symptoms immensely exceeds that of the sufferers from genuine and dangerous angina pectoris. Moreover, the urgency of the symptoms is usually far less in these affections than in the true angina. The pains, in the milder disorders, are usually much less defined in character, and are never, or hardly ever, accompanied by so grave a sense of impending dissolution. The diagnosis requires tact and judgment rather than any elaborate rules of investigation, to save the physician from error.

A much more difficult diagnosis, and one in which in many cases it is impossible to arrive at more than a proximate conclusion, is the determination of *how far* any organic disease, and *what kind* of organic disease, may have had to do with the symptoms present in any particular instance of angina pectoris. Clinically speaking, it may be said that, as a question of pure experience in the living patient, the formidable prognosis of true angina is not necessarily relieved by the knowledge that after careful examination no organic disease can be discovered; for, in the first place, organic disease may exist without the possibility of discovery; and, secondly, they are precisely the forms of organic disease most difficult of discovery that have been shown to be most frequently associated with death from angina pectoris. Given, therefore, a very perfectly characterized instance of angina in repeated paroxysms *nearly* fatal, and tending to increase in severity, it cannot be said that the special diagnosis of organic associated lesions has any very immediate practical significance. The

[1] Clinique Méd. de l'Hôtel-Dieu, t. ii. p. 444. Paris, 1865; and in the English translation, vol. i. p. 602.

prognosis in such cases is emphatically grave in the highest degree, and remains so even after the most careful examination of the organs of circulation has given only a negative result. In cases of minor urgency, however, and in cases where the diagnosis of the angina paroxysm is not perfectly clear and well defined, or where one or two such paroxysms only have occurred at long intervals, it becomes a very important question for the physician, and still more for the patient, whether or not there is any organic lesion of the chest forming a barrier to ultimate recovery, and in case any such lesion exists, whether it is of a kind likely to be rapidly and inevitably fatal, or the contrary. These considerations give an importance to the details of diagnosis in angina pectoris which at first sight they might not seem to possess, as bearing on prognosis and treatment.

Dr. Latham has very truly said that in this respect at least the paroxysm of angina bears a certain resemblance to the paroxysm of epilepsy. In the attack itself we are obliged to act by routine, and are unable to discriminate. It is in the intervals that the physician tries to advance beyond the mere name that has guided him in dealing with the most urgent symptoms, and by careful examination of every organ and every function to discover how the whole organization can be most effectually strengthened against the enemy that is at the gates—nay, that is threatening the very stronghold of life itself. Such a complete investigation, and no other, constitutes *diagnosis*.

It needs scarcely be said that in the first instance the attention of the physician must be concentrated upon the heart, arteries, and great veins. He will inquire with the utmost care into the whole details connected with the circulation, both during the paroxysm and during the intervals. He will carefully look for evidences of hypertrophy, dilatation, valvular disease. But above all, and even in the absence of these, he will endeavor to estimate the probabilities of structural disease in the fibre of the heart itself, or of disease in the coats of the arteries leading, it may be, to induration and obstruction, or to aneurism.

Dr. Jenner, the discoverer of vaccination, was the first to make a decided advance in the pathology of angina pectoris. He did not himself publish anything on the subject, but communicated his information to Dr. Parry,[1] by whom his views were substantially adopted and brought before the public. A very remarkable series of facts appeared to these observers to show conclusively that angina pectoris was dependent in many, if not in most cases, on "ossification," or some other form of obstruction by disease, of the coronary arteries of the heart. Subsequent researches have proved that this view cannot be exclusively maintained, although according to Lussana[1] this condition has been found present in twenty-one out of thirty-six fatal cases. The statistics adduced by Sir John Forbes[2] show that in twenty-four out of forty-five cases examined after death there were found diseases and degenerations of the aorta; in sixteen cases the coronary arteries were diseased, and in a like number the valves of the heart; while in ten cases there was positive disease, and in twelve cases preternatural softness, of the heart itself. Many authors, from Morgagni downwards, have recorded cases of thoracic aneurism having in a more or less perfectly developed form the characteristic symptoms of angina pectoris; and we have already alluded to M. Trousseau as confirming by his large and carefully-watched experience the view that such cases may very closely resemble, and may, in fact, for a lengthened period, and after careful observation, be undistinguishable from what he regards as the truly idiopathic forms of angina. The author of this article is able from personal experience to say that no organic disease has appeared to him more frequently to assume the symptomatic characters of angina than aneurism; and he is also prepared to state as the general result of inquiries pursued over many years, and particularly directed to this subject, that even small aneurisms, arising very near the heart, and especially such as project into the pericardium, or compress in any degree the base of the heart itself, are much more apt to give rise to angina-like symptoms than much larger tumors in more remote positions. The attention of the physician in cases of supposed angina pectoris should therefore always be very minutely directed to the state of the arterial system as a whole, and more especially to any evidences that may exist of irregularities in the sounds or impulse of the arteries near the heart, or of the aorta in its ascending portion. The careful examination by percussion of the substernal region, and especially of the upper sternum; the comparison of the sounds of the heart with the arterial sounds, as heard at different points of this region; the detection of even slight traces of abnormal impulse, or of evidences of arterial obstruction at the root of the neck; the comparison of the radial pulses, and the

[1] Op. cit. p. 3.

[1] Gazzetta Med. Lombard. 1858–9 (ref. by Friedreich in Virchow's Handbuch, vol. ii. p. 422).

[2] Cyclop. of Pract. Med.; art. Angina Pectoris.

thorough investigation even of remote parts of the arterial system, may lead to inferences favorable, or the contrary, to the idea of an organic cause for the symptoms of angina pectoris.

Not less important, could it be obtained with reasonable precision, would be the evidence, in any case of angina, of a permanently weakened or disorganized state of the muscular fibre in the heart itself. We have seen that in twelve of the forty-five dissections collected by Sir John Forbes, there was found preternatural softness, and in ten positive disease of the heart, apart from valvular lesions. That many of these must have been cases of fatty degeneration of the ultimate texture of the organ is rendered extremely probable, if not absolutely certain, by the results of later inquiries,[1] which show that in a large proportion of cases of sudden death such changes in the tissue of the heart have been revealed by the microscope. On the other hand, it must be admitted that fatty heart has been often observed to be present to a very great degree when no symptoms at all resembling angina pectoris have been recorded during life, and when death, too, has not been sudden, but has occurred in the course of ordinary and sometimes of acute disease, having no apparent connection with the state of the heart. This subject will come under consideration hereafter, but in the mean time it may be stated in general terms that while a degenerated state of the cardiac muscular fibre is with great probability to be inferred in angina pectoris, there are few positive criteria which can be applied so as to ascertain the fact of the degeneration, much less its pathological character, or the extent of fibre involved in any particular case. Only after careful and repeated examination of the heart under various conditions of activity and comparative repose, will a careful physician venture an opinion as to the soundness of the organ in this respect, and even then it will be prudent to express his opinion with some degree of reserve. The practical inferences, moreover, which can be safely founded on such an opinion, either in relation to prognosis or treatment, are far from being clearly established.

Having as far as possible investigated the condition of the heart and arteries, it will be the duty of the physician to complete his diagnosis by a survey of the condition of the other organs and functions. Although in many of the most extreme cases of angina pectoris the lungs seem to be perfectly healthy, yet a certain amount of pulmonary congestion or obstruction may attend the disease in particular cases, especially in those complicated with dilatation of the heart, or with valvular disease. Such cases usually present more or less alteration of the complexion in the direction of lividity, and are also attended by cough, or by true dyspnœa. And it must not always be concluded that the effect of a pulmonary or bronchial complication is to give a more dangerous or hopeless character to the symptoms of angina. On the contrary, the pulmonary disease being frequently of a manageable kind, the application of the proper treatment will sometimes extricate the patient from a state of the greatest apparent danger, and allow of the return of the heart to a state either apparently normal, or nearly so. The author has a most vivid recollection of one case in particular, where, on numerous occasions during five or six years, he had to attend a patient manifestly suffering under complex diseases of the heart and lungs, with distinct paroxysms of angina, and physical signs of dilatation of the heart. In the worst attacks there was always a nearly or absolutely complete disappearance of the pulse at the wrist; the complexion was livid, and the expectoration was of the character usual in hemorrhagic condensation of the lungs, which was also indicated by dull percussion at both bases; yet from this formidable state the patient again and again rallied under careful treatment of the pulmonary disease, and although the state was evidently one of hopeless character as regards the ultimate termination, he was able in the intervals to pursue a rather laborious occupation. In other instances, the symptoms of angina pectoris are associated with enlargement or disease of the liver, and it is not easy to say whether the hepatic disorder is of primary or of secondary origin; but here also the cautious use of remedies is often very effective in removing the obstruction to the portal circulation, and thereby in restoring the heart to a comparatively sound condition, in which the threatening symptoms of angina may disappear.[1] Re-

[1] Dr. Quain has stated the argument with reference to the older observations of soft flabby heart, with great force and conciseness in his paper on "Fatty Diseases of the Heart;" Medico-Chir. Trans. vol. xxxiii. p. 129.

[1] It occasionally happens that the very intense and sickening pain of biliary calculus presents a degree of resemblance to angina in its accessories; and the author has even observed cases in which the diagnosis remained doubtful until the yellow tinge of the conjunctiva, appearing after an interval of hours, relieved the apprehensions of the physician. The remarks in the text, of course, apply not to this condition of pseudo-angina, but to the combination of true angina with hepatic congestion. But the admission of the existence of such a combination is not to be taken as a confirmation of the view of Brera, and of the elder Latham, that angina pectoris may be

nal disease forms a very serious and often unmanageable complication, attended by most distressing sickness, or by violent dyspnœa or orthopnœa, and requiring great caution in the use of internal remedies, but perhaps not altogether beyond the scope of treatment. Dyspeptic complications are usually of secondary importance, and cannot be said to be characteristic. They are most frequently associated with gouty angina.

Among constitutional states, gout is unquestionably the one which is most frequently related to angina pectoris; indeed, it would scarcely be too much to say that a large proportion of the suddenly fatal endings of gout in its irregular and atonic forms, more especially in the forms popularly termed "gout in the stomach," or "gout in the heart," are of this character.[1] No doubt the pathology of the states indicated by these terms is very uncertain, and the terms themselves vague and unsatisfactory to the last degree; but enough remains after every deduction to show—1. That gouty persons, and especially those who have had regular gout, degenerating after repeated attacks into the irregular and atonic forms, are subject, in an unusual degree, to the causes of sudden death; 2. That not only is death in such persons apt to be extremely sudden, but, further, the course of the disease is apt to be disturbed by violent paroxysms of internal pain; 3. That in certain cases the pain has distinctly the character of angina, while in other instances it seems to be associated with dyspeptic suffering, and with disorders of the liver and kidneys—the latter, at least, distinctly represented by a special form of disorganization which can be discovered and recognized after death; 4. That in gouty subjects the heart and arteries are very prone to become disorganized, and that the disorganization is specially apt to assume the form which other observations show to give a predisposition to angina, viz., calcareous degeneration of the aorta, especially of its commencement, and of the coronary arteries; 5. That cerebral disorders of various kinds in the gouty have often a like orgin in disease of the arteries of the brain. From these various observations, which will be found amply supported by the experience of physicians, and illustrated in the treatises of best authority upon gout, it may be inferred that the so-called metastasis of gout to the heart is the result of gradual degenerative changes operating more or less throughout the organism, which, if not so distinctly related as has sometimes been supposed to the gouty paroxysm in its ordinary form, are at all events closely associated with the causes of gout, and therefore form part of its history as a disease of the constitution. So much may be fairly asserted here, without involving us in this article in a discussion of the complicated questions of pathology and diagnosis, as well as of treatment, which arise out of the general question of gouty metastasis.

As regards other constitutional states associated with, or tending to produce, angina pectoris, nothing is known of sufficient importance to find place here. But the careful physician will always endeavor in each case to discover all the causes of deranged general health which may be interfering with the normal state of the functions; and thus, with each new observation thoroughly and scientifically recorded, the diagnosis of the disease, and with this many questions bearing on its pathology and treatment, will probably be rescued from the obscurity that at present surrounds the whole subject.

What has to be said here about the *causes* of angina has been to a considerable extent anticipated in the preceding sketch of the diagnosis. All the associated diseases may be regarded as causes, or on the other hand, and sometimes with greater probable truth, as conjoined effects of one or more common causes. Thus, to take the last-mentioned instance, gout may be more or less directly a cause of the angina paroxysm; or gout and angina pectoris, each of them separately considered in relation to previously existing states of the constitution, may have grown out of like proclivities in respect of age, sex, inheritance, habits of life, &c. In following out this obscure subject, there is great danger of running into over-refinements, which may mislead, and at all events may not be supported by sound practical observation. A few facts, however, remain to be stated as to the predisposing causes.

In his classification of cases according to age, Sir John Forbes found that only one-seventh of the cases recorded (12 out of 84) were below the fiftieth year of age; and in respect of sex, only one-eleventh (8 out of 88) were in women. It is just possible, indeed, that these apparent facts may be greatly biased by the mode of collection of the instances.[1] In a disease the

simply a disorder of the liver and nothing more.

[1] On this subject see Dr. Brinton's thoughtful dissertation on "Gout in the Stomach," in the second edition of his work on Diseases of the Stomach, p. 354, 1864.

[1] Sir John Forbes, in giving the numbers in the text, expressly states that it is necessary to "make some allowance for circumstances connected with these recorded cases, before they can be received as grounds for fixing the statistics of the disease, taken

symptoms of which are so purely subjective, the deaths of men of eminence, or men of a certain force and decision of character, leading to clear and precise statements as to their symptoms and morbid history, will culminate, as it were, in the minds of physicians, and will be recorded prominently when others would pass unobserved, or at least unrecorded; and in this point of view it is worth while to remark that the Registrar-General's returns, bearing on sudden death, do not show anything approaching to this remarkable disparity of males and females, nor even the marked if not exclusive proclivity of the advanced ages to this form of death. On the other hand, the Registrar's returns no doubt include under the term "sudden death" a great mass of utterly heterogeneous cases, some of which have no natural alliance with the disease now under consideration; and the convictions of individual physicians of large experience tend more or less in the direction of Sir John Forbes's averages.[1] Another fact, of importance if correct, and so far corroborated by Dr. Walshe, is to be found in certain tables by Sir Gilbert Blane,[2] showing the rarity of Angina Pectoris in hospital practice. Both in hospital and private practice, however, perfectly typical instances of the angina of Heberden are rather rare; and Sir G. Blane's figures, supported as they seem to be by an appeal to so large a number of miscellaneous cases (3835 hospital, 3813 private), probably mean only that Sir G. Blane was too busy to know much about the internal sensations of his hospital patients, and knew only a little about a very few of his more distinguished private patients. Medical statistics are altogether perverted from their legitimate use when statements of this kind are put forward without qualification, as if numerically exact. It is certain that conditions at least closely allied to angina pectoris are not very rare in hospital practice, and the author of this article has seen enough even of typical instances in hospitals to neutralize the force of Sir G. Blane's remark. Still, it may be conceded as at least probable, that in the higher ranks of society cases of extremely sudden death, associated with the symptoms described by Heberden, and not of aneurismal origin, or connected with valvular disease of the heart, bear numerically a higher proportion to the whole field of disease than among the classes usually treated in hospital. The subject, however, is one still open to investigation, and one on which a really adequate contribution of carefully and impartially observed facts would be of great advantage to science. The facts above recorded, so far as they may be trusted in leading towards a conclusion, tend to support the theory of the gouty origin of true angina pectoris. It cannot fail to be remarked that the disease seems to be dominated by the same proclivities of age, sex, and condition in life as gout. And there is further a very general impression among physicians and among the public, not supported by exact statistical evidence, but not on that account to be disregarded, that sudden death from heart disease is frequently hereditary, or at least is found to cling as a tolerably well-marked characteristic to certain families, sometimes for several generations. On the other hand, it should be stated, in qualification of this impression, that there are numerous instances of eminently gouty families in which no such tendency has been observed.

The general result of the inquiry into predisposing causes has been stated by Sir John Forbes in terms which may well receive the assent of physicians, at least as a provisional conclusion, till further and more exact analysis of the facts becomes possible. "Like many other diseases," he writes, "angina is the attendant rather of ease and luxury than of temperance; on which account, though occurring among the poor, it is more frequently met with among the rich, or in persons of easy circumstances."[1] To this it must be added, that the influence of sedentary occupations is remarkably apparent in Dr. Quain's collection of cases of fatty heart, in many of which the death was sudden, and with symptoms more or less allied to angina. Thus, in twenty-four of the cases in Dr. Quain's memoir,[2] in which the habits of life were noted, they were found to be "sedentary" in twenty-two, "active" only in two cases; and in several cases the sedentary habits were obviously determined by injuries which had restricted the power of exercise, or by accumulations of external fat

without reference to its degree of severity." His idea is that the "more severe cases, particularly such as depend on organic disease of the heart and great vessels," occur chiefly in males; the milder in females. "The very severe cases naturally attract more attention, more particularly if they have been terminated by a sudden death, and followed by a dissection; and these are the cases that are usually recorded and published." (Art. Angina Pectoris, Cyclop. of Medicine, vol. i. p. 83.)

[1] Among authorities of the first class, Trousseau is almost singular in disputing this position. "I do not think it proved," he says, "that males are more subject than females to this singular affection." Op. cit., Eng. Transl., p. 603.

[2] Med.-Chir. Trans. iv. 133.

[1] Loc. cit., vol. i. p. 83.

[2] Med.-Chir. Trans., vol. xxxiii. p. 194.

amounting to excessive corpulency. In some cases also, the disease itself has produced an aggravation of the tendency, by still further limiting the capacity for physical exertion, and thus allowing of fatty accumulation. Thus, in the well-known case of John Hunter, who certainly was not chargeable with any original sins of laziness, and who died of angina, it is recorded that after the tendency had been clearly declared, "the want of exercise made him grow unusually fat."

Thus far we have treated of Angina Pectoris as a distinct morbid form or group of phenomena, in which disorders of the circulation tending to sudden death are associated with local pain and other symptoms in the chest of a more or less definable character. But it must be added that many cases of sudden death, in which there is reason to attribute the ultimate result to disease of the heart, have occurred apparently without pain, sometimes without any, even the slightest, previous evidence of cardiac uneasiness, and certainly without any of the more characteristic and special symptoms of angina pectoris. It remains to consider these cases before proceeding to discuss the pathology of the whole subject.

Dr. Latham has justly remarked, in reference to the present subject, that "cases of sudden death often present themselves as mere fragments to our observation. Individuals are found dead. The mode of their dissolution and the circumstances preceding it are unknown." It can only be inferred remotely, as it were, and that only in some instances, from some casual and often very imperfect observation, that in these individuals the symptoms might possibly have been shown, had they been fully ascertained, to "hold a pathological kindred" with angina pectoris.

But again: cases not infrequently occur in which the symptoms observed during life resemble angina pectoris, but where certain of the characters attributed to that disease are either entirely wanting or imperfectly developed. It may be the pain that is wanting to the completion of the picture; it may be the sense of impending death, or it may be that sudden death does not actually occur, although most of the other symptoms of angina are present. Can we, with any degree of security, bring out of these nosological "fragments" such new combinations as may tend still further to throw light on the pathology of angina?

In this difficult inquiry, in which we are reduced to the study of "broken lights" and "fragments" of truth, we feel more strongly than ever the inadequacy of language, as between man and man, in treating of the mysteries of life. We are engaged upon what ought to be a strictly inductive clinical investigation; but the very elements of the induction are in great part withheld. Many patients, when threatened with death, refuse to speak about it, and remain, up to the very last, silent as to what is passing within. Many other patients throw out hints and indications, but are either unable or unwilling to enter into a detailed analysis of their sensations. A few describe their sensations with great minuteness, but in terms which are almost sure to mislead.

From these various causes it happens that sudden death may appear to occur absolutely without previous warning, or with very imperfect previous warning, and yet there may have been in reality a very decidedly abnormal state, fully present to the consciousness of the patient, but not *spoken out* by him; either because the symptoms were *unspeakable*, or because from one cause or other he was indisposed to speak. On the other hand, sudden death may not occur, and yet a patient may have lived days, or months, or even years, in the apprehension of sudden death, being warned by such internal sensations as have been described in reference to the paroxysm of angina. When, indeed, *pain* is the culminating symptom, the patient rarely omits, or refuses to speak out; he is then sufficiently explicit as regards the pain, but in many cases he leaves the other and less definable sensasations to be inferred. But where pain is not the culminating symptom, we are often reduced to inference altogether; and it is only in the case of persons whose outward lives and inner thoughts are much before the public, that an inferential diagnosis can be arrived at. Two cases of this kind, occurring in different ages of the world, and under very different circumstances, appear to afford in some degree the means of access to some of the information we are in quest of. One of these is the case of the Roman philosopher Seneca; the other that of the Christian divine Dr. Chalmers. The former case has been often referred to (though with some hesitation, the source of which will be immediately apparent) as one of angina pectoris; the latter has been recorded expressly as a case of sudden death from fatty heart.

The case of the philosopher Seneca was as follows:—

In early life he was apparently of delicate constitution. It is recorded of him by Dio, that but for the apparent probability of his early death spontaneously, Caligula would have had him destroyed. The supposed disease at this time was a *tabes*. He himself records that he was nursed with difficulty through a long illness by his aunt (*Consolatio ad Helviam*,

16). He further speaks in one of his epistles of having been extremely subject to catarrhal fluxes (destillationes), and in another he says that almost every form of bodily disturbance had affected him at one time or other.[1] It seems, therefore, extremely probable that Seneca was one of those martyrs to tubercular disease in early life, who, after a more or less protracted period of ill-health became somewhat more robust in constitution towards the middle term of life. He was, however, to the last more or less delicate, and at the time of his violent death at the instigation of Nero, he is said by Tacitus to have been "emaciated in body from scanty nourishment."[2] The peculiar symptoms, however, which have specially attracted the attention of writers as indicating angina pectoris, seem to have been confined to the last two years of his life, according to the opinion of Lipsius, who considers the epistles to Lucilius as having been written when he was sixty-one or sixty-two years of age. What gives a peculiar interest to the description, and at the same time may possibly make necessary a qualification of some of its expressions, is the somewhat affected and pretentious tone in which in these letters Seneca, a disciple of the Stoic philosophy, congratulates himself on the ease and freedom with which he could look death in the face, and maintain under severe illness, and in the prospect of sudden death, the calm, self-possessed, and cheerful spirit of the sage. His philosophy, under these circumstances, has in its details no important relation to the present inquiry; but the fact that his mental condition was such as is here described is important.

After a long truce from suffering, he says,[3] his bad health has returned upon him suddenly. He is as if given over to one disease, as regards which he adds: "I know not why I should give it a Greek name, for it may fitly enough be called *Suspirium*—a sighing, or want of breath." The attack is very brief and like a hurricane—it is over almost within an hour. As compared with any other disease it is like the difference between *being sick* merely, and *giving up the ghost*—so that the physicians themselves call this disease *meditatio mortis;* and sometimes death, which is always threatening in it, actually occurs. Knowing these things Seneca adds that he is by no means confident of recovery, even when relieved from severe symptoms. He considers only that he has got a respite; he is perfectly prepared for death; he does not at any time count even upon seeing out the day. He is, however, buoyant and cheerful, entertains himself with gladsome and strong thoughts, even in the midst of the stifling (*in ipsa suffocatione*). Death is, after all, not to be dreaded by the wise man; death may take him unawares, but he is nevertheless always ready to go. Even at the best, he adds, reverting to his own precarious condition, his state is not one of entire comfort; the breathing[1] is not quite natural; he feels always a degree of impediment (*hæsitationem quandam ejus et moram*). "Be that as it may be," he adds, "provided my sighing is not in sad earnest" (*dummodo nisi ex animo suspirem*). He holds himself as in the condition of one likely to be soon ejected, but yet not to be *ejected*, inasmuch as he is willing to go. "*Nihil invitus facit sapiens; necessitatem effugit quia vult quod ipsa coactura est.*"[2]

In this case of Seneca we have, in a highly developed form, the sense of impending death, associated with something which he himself calls a "suffocation," occurring in paroxysms, and causing daily and hourly uncertainty as to his tenure of life. But we have not the severe and peculiar pain of the angina of Heberden, nor have we the actual fact of sudden death, at least in the usual sense of the word; for, as is well known, Seneca was put to death by Nero, or rather was invited to put himself to death; and what we are able to gather from contemporary history as to his last moments would lead us to infer that death came by no means easily, but after a rather long and tedious struggle. Much doubt has been expressed accordingly, since this narrative was suggested to Dr. Parry by "a learned physician" as a case of angina pectoris, whether the symptoms will bear that construction. Dr. Parry himself inclines to consider it "rather a disorder of respiration than angina pectoris."[3] Sir John Forbes, on the other hand, says that "the case of Seneca, as described by himself, has been generally considered a case of angina, and we think most justly."[4] It is evi-

[1] *Omnia corporis aut incommoda aut pericula per me transierunt.* Epist. ad Lucilium, 54.

[2] The scanty nourishment here spoken of was not starvation, but probably deficient power of assimilation; for Seneca, as is well known, was enormously rich, and there is no reason whatever to suppose that his stoicism ever took the form of asceticism, or of voluntary fasting such as to injure bodily health.

[3] Epist. ad Lucilium, 54.

[1] "Non ex naturâ fluit spiritus." The double sense of *spiritus* in Latin, as of the Greek πνεῦμα, must be kept in view in interpreting this expression.

[2] Loc. cit. Compare also Epist. 61.

[3] Op. cit. p. 36.

[4] Loc. cit. p. 81. The opinion of Dr. Stokes, published in 1854, and founded mainly on the character of the respiration as implied in the word "suspirium" (which, as we shall hereafter see, he had himself occasion to de-

dent that materials fail us in attempting to decide the question; and they fail precisely at the very point where materials always must fail, unless the fact of actual death, and of sudden death with symptoms and signs referable to the heart, comes in to decide the point in favor of angina. True, the absence of recorded pain on the one hand, and the presence of something like a record of dyspnœa on the other, have been regarded as additional circumstances in favor of the view that Seneca's disease was spasmodic asthma. But in spasmodic asthma, however severe, there is rarely that vividly present sense of impending death so much dwelt upon by Seneca. Moreover, the noisy paroxysms of asthma would probably have provoked some more distinct allusion to the wheezing as a feature of the attack. Having regard to the idiom of the Latin language, indeed, the question as between some form of cardiac suffering and asthmatic dyspnœa must remain doubtful; but while the allusions to the breathing are of a very indefinite character, it must be remarked that the sense of impending death is the one obvious fact in the description.[1]

Turning now to the case of Dr. Chalmers, we find in almost every point the converse of that of Seneca. We have here the awful fact of sudden death in all its solemnity and mystery—not only without any adequate clinical history of chronic disease, but without any evidence of angina, or any other form of acute attack preceding the fatal event. And what adds to the mysteriousness of the result is, that the death took place, not amid any exciting crisis of passion, or of physical exertion, but in the darkness and stillness of the night, when body and mind alike had been undisturbed for hours. One indeed, who knew him,[2] has said of his conversation and manner the evening before his death: "I had seen him frequently in his most happy moods, but I never saw him happier." But this is not all. The narrative of Dr. Chalmer's death, and of the last weeks of his life, has reached us from two particularly well-informed sources. Dr. Hanna, who was his son-in-law and perhaps his most intimate friend, has given us the facts as known to his domestic circle. Dr. Begbie, who was his medical attendant, has recorded them with special reference to the observation, made after death, that the heart was in an advanced state of fatty degeneration, soft and friable, the muscular fasciculi barely traceable, without visible striæ, and everywhere containing fatty granules; the ventricles unusually thin, the "coronary artery loaded with calcareous deposit, much contracted, and in one place obliterated, presenting considerable resistance to the knife."[3] It

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scribe as characteristic of fatty degeneration of the heart), is too important for its details to be omitted here. We therefore give it entire, as it occurs in "The Diseases of the Heart and Aorta," p. 530. "We must agree with Dr. Parry in the opinion that the symptoms here detailed are not those of angina pectoris. It is remarkable that the occurrence of pain is not alluded to. But their similarity to that abnormal respiration, already described as the attendant on the fatty heart, is too obvious to be overlooked. For in this affection we see that special form of dyspnœa which may be described as a paroxysm of sighing. Seneca's words, 'Satis enim apte dici suspirium potest,' and again 'Brevis autem valde, et procellæ similis, impetus est,' are singularly expressive of a severe case of the cardiac sighing observed in persons laboring under fatty heart, for which, when the highest point of suspirious breathing has been reached, we can have no better comparison than that of a storm. And the words 'Deinde paullatim suspirium illud quod esse jam anhelitus cœperat, intervalla majora fecit et retardatum est et remansit,' well expresses the gradual ascent from what we may term the apnœal period to the extreme point of the paroxysm, and its subsequent decline." It is important to observe, that Dr. Stokes, in the chapter on Deranged Action of the Heart, expresses himself as follows with regard to angina pectoris in general: "The respiratory phenomenon which belongs to angina is some form of the sighing respiration so important a symptom in the fatty or weakened heart. . . . Upon the whole we may conclude that the special group of symptoms described as angina pectoris by Heberden, Parry, Percival, and Latham, is but the occurrence, in a defined manner, of some of the symptoms connected with a weakened heart." Op. cit., p. 487. These remarks of one of the greatest masters of modern medical observation will be found to have a very special importance in connection with what we have ventured to call, in a subsequent paragraph, *Angina sine Dolore*.

[1] Seneca particularly notes that the physicians called his disease *meditatio mortis;* a very unlikely and unusual form of medical expression for a disease so well known as ordinary spasmodic asthma. On the other hand, it must be admitted that *suspirium* was sometimes used as synonymous with asthma. Compare Cael. Aurel. Morb. Chronic. L. iii. 1; and Plin. Nat. Hist. xxiii. 7, 63, § 121. Celsus makes use of *difficultas spirandi*, and *spiritus difficultas*, but not of *suspirium*. The noise of the breathing is specially noticed by Celsus—"spirare aeger sine sono et anhelatione non possit" (L. iv. 8); and also by Cael. Aurel. "stridor, atque sibilatio pectoris." Loc. cit.

[2] The Rev. Mr. Gemmel, who was living in his house at the time. See Hanna's Life of Chalmers, edition of 1854, vol. ii. p. 775.

[3] Edinburgh Monthly Journal of Medical Science, vol. xii. 1851, p. 205. There were

is in the presence of these pathological data (given on the authority of Dr. Bennett) that we have to explain, if we can, the known facts of Dr. Chalmers's later life, and of its sudden and mysterious close. And the peculiar interest and value of the case in relation to our present inquiry consists in the following statements, which are carefully condensed from the two narratives above referred to.

Dr. Chalmers was a man not only of great genius and devotion, but of the most incessant and absorbing occupations. During a life extending nearly to the term of "threescore years and ten," he was scarcely ever withdrawn from public observation. He was eminently, in the highest and best sense, ἀναξ ἀνδρῶν—a leader and ruler of men; the "care of all the churches" was upon him, as on St. Paul, and the earnest and ceaseless labors of a life devoted to noble ends, were continued up to the very day before his death, in 1847, in his sixty-eighth year. In 1834, it is true, on the 23d of January, he had suffered a rather alarming attack of hemiplegia, from which, however, he soon recovered; and in June of the same year there was again a threatening; but with these exceptions his health appeared to have been always good, and equal to every ordinary exertion whether of mind or body. "He was hardly ever incapacitated by infirmity or loss of health from prosecuting his enterprise; and from early manhood to green old age, even up to his latest hour, he toiled, and spent his energies and strength." Probably no man in Scotland in the present century, with the doubtful exception of Sir Walter Scott, had led a life of such persistent literary activity, combined with so much and so various intercourse with men of all ranks in society. In his later years he retired more than previously from public business, but, as Dr. Begbie writes, "he was firm and robust. With accumulating years came a disposition to obesity; and with the silver-gray on the massive forehead came also the pallid and somewhat sickly look of fading health. Yet he seldom complained; or, if indisposed, it was only by some trivial ailment arising from indigestion. He was sometimes sick at stomach, but he was never faint, nor ever swooned away. . . . He had no præcordial pain or distress in breathing; no palpitation of the heart, or intermission of the pulse. He ascended heights with wonderful facility; he slept on either side, and his rest was calm and refreshing." Such was his state apparently, according to his physician, up to a period indefinitely near the fatal close.

It so happens that of the last month of Dr. Chalmers's life we have very exact records, including many memoranda, letters, &c., from his own hand. It was a month fraught with unusual excitement and exertion for a man in his sixty-eighth year. On Thursday the 6th of May, 1847, he set out for London to attend a committee of the House of Commons on a subject in which he was very deeply interested. He preached[1] in Marylebone Church on the 9th, and on the 12th submitted to a long, searching, and fatiguing examination, wherein Sir James Graham tried to "heckle" him (as he expresses it) for an hour together; but, he writes at the close of a lengthened description of the day's proceedings, "we concluded in a state of great exhaustion, but with an erect demeanor and visage unabashed." Such was his own humorous account of an event which obviously gave him much anxiety. In London, also, he made many visits and saw many sights, not sparing himself at all, or complaining in any way. On the 15th he went to Brighton, where he preached on the 16th, returning to London on Monday. On Tuesday he went to Oxford, seeing the sights of the place, and then going on to Bristol; the remainder of the week he spent there in excursions with great enjoyment, and among friends. He preached on Sunday

also traces of very chronic disease of the membranes of the brain, but probably not of such amount and character as to have much clinical importance as regards the fatal event.

[1] It may be worth while to remark here that *preaching*, with Dr. Chalmers, was something very different from the mere delivery of written words in an audible tone of voice. It was, in truth, a work into which he threw all his great energy of mind and body, and in its effect fully justified the remark of the old Scotchwoman who found it necessary to apologize for her favorite preacher *reading* his sermon, "Ay, but its *fell* reading *thon*." That Dr. Chalmers preached on every Sunday during this excursion is therefore a noteworthy fact, and the more so as he appears at this time to have been little in the habit of preaching when at home. In a more recent case, where death from heart disease was not sudden, but on the contrary very lingering, and where the very earliest symptoms, twenty-seven years before, had been such as to give warning of an impending danger, preaching had to be abandoned almost from the first; and although afterwards resumed, it became, in a second attack of ill health, the first duty that had to give way, from its manifest tendency to overstrain the weakened organ. (See the Autobiography and Memoir of the Rev. Dr. Guthrie, recently published; especially vol. ii. pp. 201–41, 215, 16, 18, 406–11.) It is to be observed that a very active use of the pen, and a great deal of work and enjoyment of life, continued possible to Dr. Guthrie for eight or nine years after the formal closure of his career as a preacher. He died in 1873, in his seventieth year.

at Bristol, and on Tuesday the 25th was at Darlington. In this interval he wrote a long and carefully considered note on the Education Question for Mr. Fox-Maule, and took a most affectionate leave of his sister, Mrs. Morton, with many effusions of pious feeling, but apparently without any despondency or personal misgiving as to the future; on Friday the 28th he returned home, as Dr. Hanna records, "bearing no peculiar marks of fatigue or exhaustion."

The next day (Saturday) was fully occupied in preparing a Report for the General Assembly, which he was to read on the following Monday. On Sunday morning (30th of May) he did not rise to breakfast, but, in answer to inquiries, said—"I do not by any means feel unwell; I only require a little rest." He conversed "with the greatest clearness and vigor;" attended church, and walked some distance afterwards with a friend on his way homewards; spent the evening in apparent good health and spirits, and among other occupations wrote to his sister at Bristol a hopeful and affectionate letter, expressive of perfect contentment and satisfaction. He retired to rest at the usual time, and the next morning was discovered dead and cold.

The separate accounts given by Dr. Hanna and Dr. Begbie leave no doubt that death took place long before the morning light, but at what exact period it was impossible to say. The body had an attitude of calm repose. "The bedclothes were scarcely disordered; on them rested a basin which had received the contents of the stomach."[1] This was the only evidence of anything like a death-struggle. Had it not been for this, it might have been supposed that Dr. Chalmers died in his sleep.

Cases like that of Dr. Chalmers (in respect to the suddenness of the fatal close) have often been recorded; but in very few of those in which the fatal result has been most sudden and startling have there been any such records of the incidents preceding death as are given above. In not a few of the cases observed personally by, or more or less intimately known to, the author of this article, there has been reason to believe that considerable suffering, or sense of disability, though not always of one and the same character, has been present; and in some of these it might easily, perhaps, have escaped attention had the individual been extremely reticent, or not surrounded by anxious friends, intent upon everything that appeared to affect the comfort of one dear to them, or the well-being of a family. In several instances, the first note of real alarm has been sounded on the discovery of an irregularity in the pulse; in one such case, sudden death took place within a fortnight, or at most three weeks, after this discovery.[1] In other cases there has been an indefinite distress felt on exertion, or on going up a hill; in a few, the more regular form of angina pectoris. One patient who had more or less of angina-like pain (so-called) breathlessness on exertion for at least some years, died at the last in bed, in the night, and at the side of his wife, who was not even awakened, or in any way made aware of his being at all uneasy, but found her husband motionless and half-cold, probably some hours after the event.[2] It therefore becomes exceedingly probable that the actual death was either painless, or at least that the duration of the suffering was so brief, as not to have given an opportunity for any expression of it. Thus a person may have been affected with angina pectoris once or oftener, and he may die suddenly, and yet it may not be at all clear that he has died in a paroxysm of angina. On the other hand, symptoms of a different order from the genuine, painful, angina pectoris, may become associated with angina-like paroxysms at a subsequent period; and yet, even then, the death may not be strictly sudden (in the sense above described), or even unexpected as to its occurrence, but rather the gradual culmination of days or weeks of sleepless agony. It is notorious among physicians that in valvular diseases of the heart, and even in aneurisms, in which the popular impression, derived from a few startling instances, is to the effect that sudden death is always to be expected, this mode of termination is in fact exceptional. One

[1] Monthly Journal, ubi supra, p. 205.

[1] In the case of Dr. Guthrie, above mentioned, a similar irregularity, with symptoms not very dissimilar in other respects, appeared to threaten sudden death in 1846, while death did not actually take place till 1873.

[2] This case was recorded with additional details, in Gout: Its History, Causes, and Cure, by William Gairdner (first edit. 1849, pp. 38–42), as a case of fatty degeneration of the heart, liver, and kidneys. The narrative there given of the symptoms is by my father, but I have a most distinct personal recollection, even at this distance of time, of all the essential facts, which both from intimate friendship, and from early professional studies, had more than usual interest for one who was just then engaged for the first time in minute pathological research; especially as occurring only a few months after the death of Dr. Chalmers. The patient became subject to the first symptoms of cardiac disease in 1841; had a smart attack of regular gout in 1846, followed by giddiness and cardiac pains, which were rarely altogether absent afterwards. He died suddenly, as described, in September, 1847, in the 63d year of his age.—W. T. G.

or two cases, widely reported, and taking possession of the imagination by their peculiar and mysterious close, become the types of a whole series, in which the incidents are only slightly or not at all removed from the ordinary course of fatal disease, as to the fact of the end being to a certain extent expected and foreseen. But even here we are beset by anomalies of experience arising from the extreme difficulty of realizing facts depending so much upon subjective impressions. For example, a young man intimately known to the author of this article went to Edinburgh many years ago to study medicine, it being known to himself and some of his friends that there was some internal flaw or weakness, in regard to the precise nature of which he always maintained a strict reserve. It was reputed (as in the case of Seneca) to be more or less of the character of "asthma;" but no regular asthmatic paroxysm was ever brought under notice. This young man pursued all his medical studies and took his degree without apparent difficulty; living in the main carefully, but often visiting the hospital at night and doing all the miscellaneous work of a hardworking student. He afterwards went to the Crimea and served through the whole campaign, up to the taking of the Redan fort, as an assistant-surgeon attached to a regiment; his letters at this time giving most minute descriptions of all his personal impressions of the scenes and great events around him, but being almost entirely silent as to his own physical sensations, if he had any, of chronic disease. He was afterwards affected with some of the current diseases of the service, and had also an attack of rheumatic fever, after which he was sent home, but continued with his regiment on its return, and finally died at Chichester in a time apparently of profound tranquillity, and with such startling suddenness that he had barely time to use some of the most familiar remedies and common external appliances before he was called away, his fellow-officers having had no previous note of warning whatever. A subsequent inquiry led to the discovery that the local applications which he had himself directed in the moment preceding his death were precisely those which he had learned in the Edinburgh Royal Infirmary to apply in several cases of angina pectoris, in the study of which he had interested himself. He had also, it appeared, confided to his mother the idea that he might possibly die suddenly, owing to some imperfection of which he was sensible at the heart. He died in his twenty-seventh year. The pericardium was found to be firmly adherent, and the heart rather small, its muscular fibres pale, and apparently altered in texture. In this instance it would seem probable that symptoms of an appreciable, but still of a tolerable kind, may have existed for many years, unreported, undescribed, and perhaps not even distinctly realized by the patient himself, though he was one carefully instructed in all that relates to this subject, and known to have taken a special interest in it from the point of view of medical observation.[1]

The cases adverted to above have been, with one or two exceptions, cases of sudden death in which the symptomatic history of the facts leading up to the fatal result is either imperfect, or altogether mysterious; and in which also the picture of angina pectoris as drawn by Heberden fails at some point or other to apply to the facts. But in cases of true angina of the most typical kind, and especially in those associated with a distinct organic lesion, such as calcification or other disease of the coronary arteries or fibre of the heart, it might easily be argued that the fact of a *sudden*, as opposed to a more ordinary mode of death, is not less mysterious than in any of those cases in which it has been preceded by no such typical symptoms. For, after all, what we know in cases of true Angina is simply the fact that pain of a certain order and of a certain degree of severity often brings death in its train; how the death occurs, and what precise conjunction of phenomena or pathological causes determines its occurrence at a particular moment, we know as little apparently in the painful as in the comparatively painless cases. It is plainly out of the question to suppose that a chronic, and in its very nature gradually advancing lesion, like fatty degeneration or disease of the coronary vessels in the direct and immediate deter-

[1] For additional details see the Edinburgh Medical Journal, vol. v. 1859, p. 95. Heberden's remarks in his first paper (1768) as to the association of angina with sudden death are important. He had at that time seen about twenty cases (four years later he notes fifty, and in his Commentaries about a hundred cases); of the twenty cases first observed he had *known* six to have died suddenly; and perhaps more may have so perished, without the fact being known. "But," he argues, "though the natural tendency be to kill suddenly, yet some of those afflicted may die in another manner" (unless such persons could be considered as exempt from all the other diseases proper to advancing age) "since this disorder will last, as I have known it more than once, near twenty years." Heberden had first become aware of the tendency to sudden death in angina, on mentioning the peculiar symptoms to a physician of great experience, who had told him that most of these cases had in his experience been suddenly fatal. The careful manner in which Heberden's own experience had been *matured* (so to speak) for publication appears very clearly in these incidental remarks.

mining cause of a death which occurs in a moment, or of spasmodic seizures which come on in the midst of comparative health, and pass away in many instances in a few minutes, or at most in an hour or two, leaving the patient with a quiet pulse, free from serious complaint, and (apart from certain forms of exertion) able for many of the ordinary duties of life. The cardiac fibre which carried Dr. Chalmers safely over the last three weeks of his life, with its harassing duties and active exertions in various places, cannot be reasonably supposed to have become *suddenly* so much more diseased (physically speaking) that it must needs be disabled to the extent of ceasing to act altogether, in the absolute quiet of an undisturbed night, after a day peacefully and happily spent in his own home, and an evening closed in a state of radiant satisfaction and joy, without any apparent trace of morbid misgivings. A like argument would probably apply to many or most of Dr. Heberden's cases of angina pectoris; to all cases, indeed, in which the element of spasm (so called) is a prominent feature; and in the elaborate argument, so well rendered and modernized by Dr. Latham, in which Heberden vindicates for his "dolor pectoris" a place among the spasms, as opposed to inflammation or organic disease, we are only seeking, with him, for a mode of reasoned description or of generalization for facts which are confessedly mysterious. The whole of the argument that has been raised since Heberden's time as to whether death in these cases is caused by *spasm* or by *paralysis* of the heart, and the small amount of actual information or real science which has emerged from the somewhat fruitless controversy, shows strongly how much we may deceive ourselves with the idea that in describing a mere association of symptoms with certain pathological lesions, we have fully explained the nature of the connection of the one group of facts with the other. From this point of view one more instance of sudden death, with all its preceding life-history, may be regarded as having a sufficient interest for us to be cited here with some detail.

The great comparative anatomist and profound physiologist John Hunter died, as is well known, with startling suddenness in the year 1793; and from all that has been transmitted to us of the circumstances of his fatal illness, and of the symptoms from which he suffered for twenty years before his death, it is evident that the opinion of one, at least, of his most intimate and confidential friends, as well as probably the secret convictions, in the end, of the distinguished sufferer himself, pointed in the direction of the angina pectoris of Heberden as the true nosological interpretation of his morbid state. The detailed posthumous narrative of the symptoms, coming, as it does, almost from the very lips of Hunter,[1] and characterized by all his restless activity of mind in the search after truth, forms unquestionably one of the most instructive chapters in the whole history of medicine. There is hardly a sentence in this wonderful narrative which will not repay the careful study of the physician; and although the substance of the whole is here faithfully preserved, the need for condensation will compel the sacrifice of many of the vivid touches which reveal the mind of genius intent, even amidst physical suffering, upon the mysteries of his own being.

How far these descriptive touches had been reasoned out into clear conceptions in the mind of Hunter himself does not appear from the narrative; it is certain, however, that his most intimate and congenial friend, Edward Jenner, postponed for many years the publication of certain highly original observations on angina pectoris (afterwards adopted and in part published by Dr. Parry), from the fear of compromising the feelings of John Hunter by a too obvious reference to his case.[2]

[1] "Each symptom," writes Sir Everard Home, "was described at the time it occurred, and either noted by himself, or dictated to me when Mr. Hunter was too ill to write. . . As the statement is made up from detached notes which were not written with a view to publication, it will appear in point of language extremely deficient; it was thought, however, best to leave it in its present form, lest by altering the language the effect of some of the expressions might be diminished, or misunderstood."—*Life of Hunter, prefixed to the Treatise on Inflammation*, 1794, p. xlv.

[2] The circumstances as delivered in writing by Jenner to Dr. Parry, are curious, and specially interesting as bearing on the early symptoms in John Hunter's case. "The first case I ever saw," writes Jenner, "of angina pectoris was that in the year 1772, published by Dr. Heberden, with Mr. Hunter's dissection. There, I can almost positively say, the coronary arteries were not examined. Another case of a Mr. Carter, at Dursley, fell under my care" (date not given); but in this case "the coronary arteries were become bony canals." "Soon afterwards Mr. Paytherus met with a case" . . . "At this very time, my valued friend Mr. John Hunter began to have the symptoms of angina pectoris too strongly marked upon him; and this circumstance prevented any publication of my ideas upon the subject, as it must have brought on an unpleasant conference between Mr. Hunter and me. I mentioned both to Mr. Cline and Mr. Home my notions of the matter; but they did not seem to think much of them. When, however, Mr. Hunter died, Mr. Home very candidly wrote to me, immediately after the dissection, to tell me I was right." In 1778, Jenner wrote a distinct

It is well established, also, that the case did, in fact, fulfil the anticipations of Jenner, both as to the fatal event, and as to the appearances observed after death. It has rarely happened, surely, that two minds so keenly alive to theoretic truth, and yet so observant of detail, have been applied to any even the most indifferent obscure case in medicine; for in this instance it is the author of the "Treatise on the Blood, Inflammation," &c., who is both sufferer and narrator, while it is the clear-sighted and eminently truth-loving discoverer of vaccination who forms and announces to us the diagnosis.

John Hunter "was a very healthy man for the first forty years of his life, if we except an inflammation of his lungs in the year 1759. In the spring of 1769, in his forty-first year, he had a regular fit of the gout, which returned in the three following springs, but not in the fourth." In the spring of 1773 (rather more than twenty years before his death) he had the first appalling attack of what may, from our present point of view, be fairly regarded as angina pectoris, though the pain (perhaps from some association of ideas with "gout in the stomach," the regular attack having, as stated above, not appeared at the expected time) was in this instance referred to the region of the pylorus. "While he was walking about the room, he cast his eyes on the looking-glass, and observed his countenance to be pale, his lips white, giving the appearance of a dead man; this alarmed him, and led him to feel for his pulse, but he found none in either arm; the pain continued, and he found himself at times not breathing. Being afraid of death soon taking place if he did not breathe, he produced the voluntary act of breathing by working his lungs by the power of the will."[1] The "sensitive principle" was not affected; for three-quarters of an hour he continued in this state, when the pain gradually lessened, and in two hours he was completely recovered.

The next attack was in 1776;[2] it was distinguished, however, by a very decided amount of vertigo, which was not present, apparently, in the first attack; he felt as if he had drunk too much, and was a little sick; on lying down it seemed as if he was suspended in the air; motion in a carriage gave the uneasy "sensation of going down, or sinking;[3] motion, either of the head or foot, was insufferable, from the idea it gave of ranging through vast distances. "The idea he had of his own size was that of being only two feet long." The special senses were extremely acute; the appetite indifferent; the pulse about sixty, and weak. In this state he con-

statement of his fears about Hunter's case, and of his views on the pathology of angina pectoris, intending it as a communication in private to Dr. Heberden; but, probably from the fear that it might lead to publication, the letter never was sent (see *Life of Edward Jenner*, by Dr. Baron, vol. i. p. 39). It is, moreover, certain that Hunter, in a fatal case recorded by Dr. Fothergill ("Medical Observations and Inquiries," vol. v. p. 254), had actually observed disease of the coronary arteries in connection with sudden death from angina pectoris as early as March, 1775; so that the presumption is exceedingly strong that Hunter not only was intimately acquainted with Jenner's views on the subject, but also had in part suggested them. There is thus a chain of evidence of no ordinary consistency tending to show that Hunter, who never formally identified his own symptoms with those of the angina pectoris of Heberden, was nevertheless cognizant of their real nature and probable termination, at least as early as Jenner's suspicions took origin, which, as we shall afterwards see reason to believe, was in 1777. The death of Hunter, in 1793, was in fact almost an exact reproduction of the very circumstances of Fothergill's case, viz., "in a sudden and violent transport of anger;" and the appearances on dissection were also strikingly similar.

[1] In this and other passages the mind of Hunter is very apparent. The speculations which follow may possibly be those of Sir Everard Home, and at all events they are not of much value as regards the present narrative.

[2] This date is probably a mistake, either of Hunter or the copyist; the true date was 1777, as appears from a letter to Jenner on May 11th, in which Hunter writes: "Not two hours after I saw your brother, I was taken ill with a swimming in the head, and could not raise it off the pillow for ten days; it is not yet perfectly recovered." During his convalescence Hunter went to Bath for three months, on the advice of his friends, who took a much more serious view of his case than he himself appeared to do. It was during his residence at Bath, apparently, that Dr. Jenner saw Hunter personally, and formed the strong views as to the character and probable issue of the case which he ever afterwards retained, and which he wrote out, as above mentioned, for Dr. Heberden in 1778.

[3] There is a characteristically Hunterian note here given in Home's narrative, which is valuable as showing how much these details of subjective phenomena interested John Hunter as a physiologist, while as mere personal matters he gave all his own sufferings extremely little consideration. "It is very curious that the sensation of sinking is very uneasy to most animals. When a person is tossed in a blanket, the uncomfortable part is falling down; take any animal in the hand and raise it up, it is very quiet, but bring it down, and it will exert all its powers of resistance, every muscle in its body is in action; this is the case even with a child as early as its birth."

tinued for about ten days; bleeding was of no service, purging and vomiting (by medicine) "distressed him greatly;" nothing appeared to be of the least use. From this severe illness he gradually recovered, but only after a long convalescence; and he does not seem to have been ever again perfectly well, having, it is said, grown much older looking in the interval between this and his next severe attack, which was in 1785.

The illness of April, 1785, may be said to have commenced with an ordinary attack of gout, followed by a great variety of anomalous nervous sensations which are minutely described, but over which it is not necessary to detain the reader.[1] Suffice it to say, that from this time onwards Hunter became increasingly subject to paroxysmal attacks, which assumed more and more the characters of typical angina pectoris. The nervous disturbance appears to have been at first peripheral, *e. g.*, "a sensation of the muscles of the nose being in action," an unpleasant sensation in the left side of the face, jaw, and throat, which seemed to extend into the head on that side, and down the left arm as low as the ball of the thumb, where it terminated all at once." After a fortnight these symptoms of nervous irritation "extended to the sternum, producing the same disagreeable sensations there, and giving the feeling of the sternum being drawn backwards towards the spine, as well as that of oppression in breathing, although the action of breathing was attended with no real difficulty; at these times the heart seemed to miss a stroke, and upon feeling the pulse, the artery was very much contracted, often hardly to be felt, and every now and then the pulse was entirely stopt." He had also pains in the heart itself, as well as the diaphragm and stomach, attended with considerable eructations of wind, "a kind of mixture of hiccough and eructation." In the most severe attacks "he sunk into a swoon or doze, which lasted about ten minutes, after which he started up, without the least recollection of what had passed, or of his preceding illness." The agonies he suffered were dreadful,[1] and when he fainted away he was thought to be dead.

As in other instances of angina, these attacks were at first brought on chiefly by motion, "especially on an ascent, either of stairs or of rising ground." The affections of the mind that were chiefly injurious were anxiety and anger; "it was not the cause of the anxiety, but the quantity of it, that affected him; the anxiety about the hiving of a swarm of bees, the anxiety lest an animal should escape before he could get a gun to shoot it," brought on an attack; "anger brought on the same complaint, and he could conceive it possible for that passion to be carried so far as to deprive him of life; but what was very extraordinary, the more tender passions of the mind did not produce it;" compassion, admiration, &c., might be carried to the extent of tears, "yet the spasm was not excited." "He ate and slept as well as ever, and his mind was in no degree depressed; the want of exercise made him grow unusually fat."

Mrs. Hunter, in writing to Jenner, called the disease, even at this stage, "flying gout."[2] We have already seen what Jenner thought of it several years before. Hunter himself was probably familiar with Heberden's description, and at all events had assisted in Heberden's inquiry by performing the examination of the very remarkable case recorded in the "Medical Transactions" in 1772. He himself began to suffer in 1773. That he had realized in some degree the danger of his position, therefore, can scarcely be doubted. He had indeed no unmanly fear of death, and was far too busy to occupy himself with what he would have regarded as weak sentimentalisms about himself. He probably avoided the subject deliberately,[3] and felt himself able to pursue all his various occupations with the same ardor as ever, in the intervals of suffering. But he was deeply sensible of the risk to which he was sometimes exposed by over-exertion, and still more by his uncontrollable temper; he was accustomed to say, that "his life was in the hands of any rascal who chose to annoy and tease him;"[4] a remarkable expres-

[1] Dr. Pitcairn elicited on this occasion, by special inquiries, that Hunter's mind had been much harassed, in consequence of his having opened the body of a person who had died of the bite of a mad dog, about six weeks before; in doing which he had wounded his hand. For a fortnight, it is added, his mind had been in continued suspense, from the idea that he might be seized with symptoms of hydrophobia; and it certainly seems very probable, as it was supposed, that the nervous symptoms alluded to may have been in some measure, at least, determined or produced by this accident.

[1] This is the personal testimony of Sir Everard Home, who witnessed this attack, having become Hunter's regular assistant in his practice, and acted for him during his illness. It is probable, but not expressly stated, that Home also was a witness to the first attack of illness in 1773, as he was then a young man living in Hunter's house.

[2] Palmer's Life of John Hunter, p. 96.

[3] In all his published correspondence there is only one brief allusion to his own illnesses, the one given above from a note to Jenner.

[4] Palmer, *ut supra*, p. 119.

sion, and a sad anticipation of the actual ending.

The close of 1789 brought with it a new set of complications, which may be briefly summarized as loss of memory, and various kinds of visual disturbance, especially the apparent deflection of objects from their true direction; some of the former subjective sensations, mentioned in the attack of 1776, returned upon him. "Dreams had the strength of reality, so much so as to awaken him; the disposition to sleep was a good deal gone, an hour or two in the twenty-four being as much as could be obtained. Neither the mind, nor the reasoning faculty, however, were affected; indeed he reasoned most acutely in regard to his own visual derangements, and pursued the questions suggested by them in physiology with a keenness which was quite characteristic.

At last the busy, ever-active mind was to cease from its labors, and the strong, much-enduring bodily frame, wearied out and spent in the service, was to give way. His recovery from this indisposition was less perfect than from any of the others; he never lost entirely the oblique vision; his memory was in some respects evidently impaired, and the spasms became more constant; he never went to bed without their being brought on by the act of undressing himself; they came on in the middle of the night; the least exertion in conversation after dinner was attended by them. Even operations in surgery if attended with any nicety, now produced the same effects.

The end is well known. There is reason to think it was almost foreseen by himself. A dispute of a painful, but not, after all, of a very serious or overwhelming character, had embittered his relations with the governors of St. George's Hospital. On the 16th of October, 1793, he determined to be present at a meeting, where, however, he apprehended a personal dispute. He expressed to a friend the feeling that such a dispute might be fatal to him, but went nevertheless. Something that he said in the Board-room was noticed, and flatly contradicted. He stopped, left the room in a silent rage, and had just time to gain the next room, when "he gave a deep groan, and fell down dead."

The appearances in the dead body were complex. The pericardium was very unusually thickened; the heart very small, its muscular substance pale; the coronary arteries were converted into open bony tubes; the valves of the left side of the heart also were involved in a similar degeneration; the aorta was dilated, in its ascending part, to the extent of one-third. The carotid and vertebral arteries within the cranium were also bony, and the basilar artery "had opaque white spots very generally along its coats." The structure of the brain itself was normal.

To these observations of what may be almost called historical cases, bearing upon the fact of sudden death and its associated symptoms, I will add only a few details gathered from a long and close observation of cardiac diseases in general.

Apart from what has been variously termed cardiac asthma, dyspnœa, or orthopnœa, which in many cases receives its clear explanation from the associated states either of the pulmonary circulation, or of the lungs, bronchi, and pleuræ, as disclosed by physical signs, there is often an element of subjective abnormal sensation present in cardiac diseases which, when it is not localized through the coincidence of pain, is a specially indefinable and indescribable sensation, almost always felt to be such by the patient himself. I make this remark deliberately, as the result of experience, and well knowing that it is liable to be brought into question in particular instances; that, in fact, a large part of what has been described under the titles given at the commencement of this paragraph, has been inextricably confounded by systematic writers with the sensation, or group of sensations, to which I refer.[1] To this group of sensations, when not distinctly accompanied by local pain, I have, in various instances, given the name of *Angina sine dolore*, recognizing, thereby, what I believe to be its true diagnostic and pathological significance, and its alliance with the painful angina of Heberden; the pain in which, however, as we have already seen, is an exceedingly variable element, both in degree and in kind. This painless, or at least not definitely and locally painful, angina, is found in connection with every kind of cardiac lesion which ends in death (whether sudden or not) in varying proportions; often associated with the other phenomena which make up the picture of a confirmed case of organic heart disease tending to death, but not rarely also under circumstances which admit of its being separately described. Among the valvular lesions of the heart, incompetency of the aor-

[1] "In considering this subject, we must not forget," writes Dr. Stokes, "that under the name angina pectoris, physicians have included, and still include, many examples of diseases which vary in their nature and combinations. Well-marked instances of the affection as described by Dr. Latham, are rarely met with; and the same may be said of the purely nervous cases noticed by Laennec. *I have never seen either of these forms.* The disease which in this country" (Ireland?) "most often gets the name of anginapectoris, might be more properly designated as cardiac asthma." Op. cit. p. 488.

tic valves is the one which most frequently gives rise characteristically to this peculiar form of suffering; and in the majority of the cases in which it arises early in the course of aortic valvular disease there is neither dropsy, or lividity, nor hæmoptysis; very often there is no disease of the lungs ascertainable by physical signs, and in particular no wheezing, even in very severe paroxysms of this truly cardiac anguish or indefinable distress. But there is, in variable degrees, a sensation which can only be called *anxiety* or *cardiac oppression;* the patient acquires a haggard, almost a frightened look, and from his habitual attitude and manner, as much as from anything he distinctly declares in words, it becomes evident that he is suffering from a sense of insecurity which he cannot possibly express. In the more aggravated cases the loss of sleep is a serious part of the suffering, and patients will sometimes declare that they are afraid to sleep, lest some other and greater evil than the loss of sleep should come upon them; obviously an experience actually acquired, that sleep is, in this state, sometimes the precursor, and apparently the cause, of a formidable increase in the symptoms. An intelligent patient in this condition recently put the question to his medical attendant, with respect to a very moderate dose of hydrate of chloral, proposed to be given after many sleepless nights, whether it would not be "dangerous," *i. e.* (as he afterwards explained to me), whether the sleep artificially induced might not be the means of determining an attack which might prove fatal. When sleep is obtained, it is brief and easily disturbed, often by frightful dreams; and when these occur they are mixed up with the sensations of an approaching paroxysm, so that the dream may appear to be the actual cause of the paroxysm. An assertion of the patient just alluded to was that he "woke up with the peculiar sensation on him, and it was too late to check it." In very extreme cases, which are often, however, complicated with true orthopnœa, dropsy, and other more recognized cardiac and respiratory symptoms of secondary origin, the patient may for weeks together be unable to lie down or to take ordinary rest, and on the other hand may be almost continually *half*-asleep; in such cases accidents are apt to occur, from the patient falling forwards in a fit of sheer exhaustion, or getting burned or otherwise injured while in a state of insensibility. Nor are more distinctly cerebral symptoms wanting. In some of these cases I have seen attacks closely resembling epilepsy, without any subsequent paralysis; when, however, hemiplegia or aphasic symptoms occur, it is most probable that they are due to more distinctly organic changes in the nervous centres; and usually to cerebral embolism. It would be vain to indicate the verbal expedients by which patients endeavor to describe their sensations, when found in an attack of this paroxysmal suffering. Palpitation, and breathlessness are often alluded to, separately or together; but still more often it is a sense of "oppression," or of "pressure," which is sometimes described as if the chest were actually being compressed from before backwards; one patient described it as a "kind of surging up," which came, as he thought, from the bowels, and was attended with the feeling of wind, and also, I suspect, with a degree of hysteric globus, rising, as he described it, to his throat, and causing him to pant for breath. The respiration is by no means necessarily or invariably disturbed in these cases, though it is frequently more or less quickened, and sometimes the opposite; in certain cases the respiration is alternately frequent and unfrequent; several rapid panting or gasping respirations are continued over half a minute together, and are gradually succeeded by a corresponding period of comparative quiescence, which at times culminates in a positive arrest or suspension, for a time, of a respiratory act (see the narrative of John Hunter's case above cited).[1]

[1] It is very remarkable that Dr. Stokes, who is undoubtedly entitled to the credit of having first distinctly realized, and clearly stated, the importance of this type of respiration as indicating cardiac disease (especially weakened action, or fatty degeneration, of the fibre of the heart) should have so completely overlooked the case of John Hunter, while fixing upon the symptoms described in Seneca's case as characteristic (see *note*, p. 676). The same remark applies to all the now numerous dissertations, in Germany as well as in this country, on the "Cheyne-Stokes respiration," as it has been called on the continent. "It consists," says Dr. Stokes (op. cit. p. 324), "in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations." Probably the first really exact description of this phenomenon was by Dr. Cheyne, in 1818 (Dublin Hospital Reports, vol. ii. p. 216). The peculiar interest and value of Hunter's case, however, for us consists in its giving the personal impressions, or subjective sensations, of that great physiologist in a way that no merely objective description could effect, and wholly apart from hypothesis. It is curious to observe how completely Hunter's description of his own sensations corresponds

This peculiar type of "suspirious," or irregularly sighing, respiration (as it has been termed), is so far characteristic of the "angina sine dolore," that I cannot but regard it as being in some way related to lesions involving the respiration through the cardiac nerves. Whether dependent necessarily on cardiac causes or not, however, it is certainly not necessarily associated with any organic lesion of the lungs or air-passages; it occurs, as Dr. Stokes has recorded, "without any *râle* or sign of mechanical obstruction." Frequently the irregularities of respiration do not go beyond a few quick gasps, or deep sighing inspirations, at a time, and the period of apnœa, or of rare and slow respiration, is correspondingly shortened; but when this condition of the respiration, even in its minor degrees, is associated with the peculiar look of indescribable *anguish*, the head thrown back, the arms extended or tossed about, and the whole frame showing by sheer muscular restlessness the terrible character of the *agony* (indicated often by cries, even when without local or positive pain), it scarcely requires the aid of a verbal description to make the diagnosis of angina clear to the observer. It is, however, important to remark that the character and peculiarly altered rhythm of the breathing are essentially distinct from the laborious but more regular and at the same time noisy respiration of true spasmodic asthma and of asthmatic bronchitis. I have also observed that organic and valvular deformities of the right side of the heart, even when complicated with great cyanosis, are only slightly characterized by the symptoms I have now endeavored to indicate; and, on the whole, the diseases of the mitral valve are less apt to be accompanied by this form of angina than those of the aortic, and the obstructive lesions less than the regurgitations. Dilatation of the heart in its more aggravated forms, however caused, and aneurisms (as already indicated) arising very near the heart, or projecting into the pericardium, are apt to be accompanied by considerable degrees of angina, as above described. And some of the worst cases I have seen have been those, in which the only lesion that could be fairly presumed to exist was fatty or other degeneration of the fibre of the heart, sometimes with, sometimes without, direct evidence of moderate or slight dilatation of the left ventricle.[1] As in the case of the locally painful, or neuralgic angina, the relation of the symptoms to the organic legion is by no means constant, even when the latter can be shown to be present, and to be presumably, in a certain sense, the cause of the symptoms. And it may further be affirmed, that the essentially paroxysmal character of this angina is such as to lead us inevitably to look for an explanation of it beyond the positive and permanent organic lesion of the heart or aorta, whatever that may be in the particular case.

We are now in a position to discuss, with such assistance as can be had both from clinical observation and from physiological pathology, the extremely obscure subject of the mode in which the innerva-

with Galen's commentary on a notable passage in Hippocrates, where a certain kind of "rare and large" respiration is described as "like a person who forgot for a time the need of breathing, and then suddenly remembered." See the very interesting account of the most ancient observations on this subject by Dr. Warburton Begbie, in his recent Address in Medicine (British Medical Journal, August 7, 1875, p. 166) in which there will also be found a brief but exact account of the more modern theories as to this kind of respiratory disorder.

[1] In one case of this kind, a much valued friend and a distinguished clergyman of the Church of Scotland, who died at the age of forty-one, after a gradually progressive illness watched with the greatest anxiety, and with full fore-knowledge of its character and probable termination, the beats of the heart frequently numbered as low as 22–24 in the minute; and I have counted them as low as 18, without any marked irregularity. The radial pulse was at these times exceedingly soft and small, but although the suffering was at times intense, it was not usually accompanied by positive definable pain, at least until the last few days or weeks of the disease, when (the patient not being at the time under my own immediate observation) I had the testimony of a well-informed medical friend as to the really angina-like character of the paroxysms. The suspirious respiration was always present in the more considerable paroxysms of suffering, and was usually not altogether absent. There were on several occasions very alarming pseudo-apoplectic or slight epileptic attacks, without permanent disorder either of the intellectual functions or of voluntary movement. Although this truly noble-minded and self-denying man pursued the work of his life up to the very verge of sudden fainting or death in the pulpit, yet his death in the end was by no means sudden, but rather a lingering agony. The entire duration of his fatal illness was under two years, and he continued at his post, with some interruptions, until about eight months before his death, which happened in January, 1865. Up to a few days before his death he maintained his pastoral connection with his congregation by means of letters, some of which have been published, and show all the power of a robust mind under the guidance of Christian principle and hope. Dr. Walshe, who saw this case with me in consultation, agreed with me in considering it one probably of fatty degeneration of the heart; but there was no *post-mortem* examination.

tion of the heart is affected in Cardiac Angina—in other words, the *ultimate pathology or pathogeny of the affection.* We have seen that the dolor pectoris, or angina pectoris, of Heberden was specially distinguished by him from all those pains in the chest which were regarded as due to inflammation, accompanied or followed by organic changes corresponding with the extent and severity of the inflammatory process. In other words, the essential pathology of angina, according to Heberden, was that of a *neurosis.* This we believe to be the only just rendering of the argument of this great physician, when he assigned to angina pectoris a place among the *distensiones*, or spasms. Later observers and pathologists have been much exercised in the attempt to resolve the question, whether sudden death, occurring under such circumstances, is from spasm, or from paralysis, of the heart; but we may safely conclude that no such question was, otherwise than remotely, involved in Heberden's argument. That argument was directed towards a very practical and real conclusion, and was not at all, we may well suppose, intended to foreclose questions of physiological pathology, which, according to all the evidence before us, were not before his mind, or, at least, not matured for discussion at the time at which he wrote. Angina pectoris had to be placed carefully apart from the *pyrexiæ* and the *phlegmasiæ;* had any doubt been left open on this subject, the *dolor pectoris* would have been considered as demanding the treatment of all so-called inflammatory pains in that day—large and repeated bleedings, vomitings, purgings, &c.[1] Hence the anxious care with which Heberden insisted on the paroxysmal and non-febrile character of the pain, and on the collateral circumstances which led him to bring it into the great group of the spasms; *e. g.* "*subito accedit, et recedit*"—"*in ipsa accessione pulsus non concitatur,*" &c. It is needless to pursue the argument in detail; possibly, indeed, the details might be open to question in some instances. But on endeavoring, as Dr. Latham has done, to grasp the essential principles of the argument, as seen through a somewhat obsolete phraseology, we may readily assent to them, even if we should suppose that Heberden, in his desire to prove angina pectoris a *neurosis*, may have somewhat neglected the evidence of its being often associated with organic disease.[2] He found in the suddenness of the paroxysms, in the apparent good health of the intervals, in the relief often afforded by stimulants and by opium, the basis of his pathology of angina; and we may easily admit that some cases, at least, of the typical angina of Heberden must have been fairly open to the construction of being cases of spasm, and nothing more. But we now know that this typical angina is only the culminating form of a group of symptoms, which, in their less pronounced, less definitely painful, and more complicated forms, are found to permeate the whole field of cardiac pathology and diagnosis. The angina which consists purely of a paroxysm of pain, and of a paroxysm which kills suddenly and instantaneously, is rare; but the angina which consists of a tendency to paroxysmal aggravations (not always purely of pain), superinduced upon, and complicating, the other symptoms and sequelæ of cardiac organic diseases, is matter of every-day experience. In both forms there occurs occasionally a paroxysm which ends in death; but in the second form death is less frequently instantaneous and unexpected, both because the paroxysms are individually less intense, and because the fatal result,

[1] Angina pectoris, quantum adhuc illius naturam intellexi, ad distensionem, non autem ad inflammationem, videtur pertinere. . . . Sanguinis missio, vomitus, et purgantia mihi visa sunt aliena.—*Comm. uti supra.*

[2] He refers, however, to several cases which seemed to him to imply the existence of organic change; and to one only, in which "a very skilful anatomist could discover no fault in the heart, in the valves, in the arteries, or in the neighboring veins, excepting some small rudiments of ossification in the aorta. Nor were any indications of disease found in the brain." There is no doubt that Heberden's personal experience of angina was almost purely clinical, not pathological; but it has the advantage, for us, of being stated in language singularly terse, exact, and free from the suspicion of prejudice. Heberden claims, in his Commentaries, to have seen nearly a hundred cases of angina pectoris, of which three were in women. One was a boy twelve years old, "who had something resembling this affection." All the rest were in men near or past the fiftieth year of their age. At the time of his first paper, in 1768, Heberden had "never seen one opened, who had died of it. Most of those," he adds, "with whose cases I had been acquainted were buried before I heard that they were dead." The case specially alluded to above was almost certainly that of the "Unknown," who, in April, 1772, wrote to Heberden a minute account of his symptoms, and dying suddenly about three weeks thereafter, was found to have left in his will express instructions that Heberden should be informed of his death, with the view of having his body examined. This was accordingly done by John Hunter, and it is this case to which Dr. Jenner alludes, when he says that he can almost certainly affirm that the coronary arteries were not examined. The case was recorded in the third volume of the Medical Transactions.

when it arrives, is brought about in part by other causes than the immediate causes of the paroxysm. And even if we should maintain that *fatal* angina is always more or less dependent upon organic changes,[1] there would still remain to be explained these unquestionable facts, viz. : 1. Pain, suddenly coming and going; 2. The paroxysmal character of the symptoms, other than pain; 3. Absolutely sudden death in a few cases. On these grounds, now as in the time of Heberden, we may assuredly claim for angina pectoris a place among the *neuroses*, even while the admission is freely made that the element of *neurosis* is often superinduced upon organic, too often indeed incurable, disease in the heart itself, or in its nutrient vessels, or in the first part of the aorta.

Certain authorities have treated of angina pectoris as a form of visceral neuralgia, or "hyperæsthesia"[2] (Romberg) of the cardiac plexus. The latter term (as Dr. Anstie has well pointed out) is essentially a bad one; the former, in the case of typical angina, is perhaps admissible, viewing the disease from the side of the pain alone; but it errs both by excess and by defect, inasmuch as, on the one hand, pain of the severe form implied in the term neuralgia is not always the central or exclusive phenomenon, even of the cases ending in sudden death; while, on the other hand, a form of cardiac pain, or *pseudo-angina*[3] (as it has been termed) is not infrequent, which has most of the attributes of a neuralgia in the highest possible degree, and which, though eminently paroxysmal, is by no means apt to lead to sudden death or to any grave consequences whatever. This admission, which is very candidly and fully made by the late Dr. Anstie[1] in his interesting dissertation upon the subject, appears to me a very cogent reason for maintaining, rather than consenting to forego, the now well-known term angina pectoris, for which he entertains so strong an aversion, but which is, nevertheless, quite indispensable to us, as carrying the impress of a long line of personal observations, extending back to that "molestus quidam angor," which Morgagni has described as having suddenly terminated the life of a Venetian woman in 1707. And if it must be admitted that the name "angina pectoris" has sometimes been used in ignorance, or rather (from disregard of purely clinical experience) in a way really objectionable and tending to confusion, it is equally certain that the term "neuralgia" is beset with theoretical interpretations which tend to bias both clinical and pathological research. We have endeavored in the preceding pages to give an impartial statement of a wide range of phenomena, into which a neuralgic element enters in various proportions. A consistent theory must take account of that element, but will not allow it to take possession of the entire field.

Another question that requires consideration is, the nature of the disorders in connection with motor nerves which unquestionably occur in angina pectoris. Here, again, we find ourselves in the presence of vague and often quite fruitless discussions, indicated by the general terms spasm, paralysis, hyperkinesis, &c., and, among the older authors, asthma convulsivum, stenocardia, syncope anginosa, &c.

A third department of the inquiry, less generally entertained, inasmuch as the phenomena to which it refers are less constant, is the nature of the connection between the cardiac symptoms in angina

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[1] Eulenburg refers to Desportes, in Lartigue —"De l'Angine de Poitrine," p. 78, Paris, 1846; Surmay, L'Union Médicale, xxxi., No. 80, p. 34; for evidence of angina without disease of the heart. Anstie, in his Treatise on Neuralgia, pp. 69, 70, details, briefly, a fatal case, in which "not the slightest organic heart mischief could be detected, either during life or after death." Latham has also recorded cases where the appearances after death were, at least, of very questionable and doubtful character. But it is difficult to prove a negative by isolated instances which are opposed to the general results of pathological research. [I met with a marked case, in 1879, in which autopsy showed no disease of the heart, except a comparatively slight fatty degeneration in some portions.—H.]

[2] "Pain has been described by some of the most distinguished writers on nervous diseases as a *hyperæsthesia*. Yet there is really very little difficulty in convincing ourselves, if we institute a thorough inquiry into the matter, that pain is certainly *not* a hyperæsthesia, or excess of ordinary sensory function, but something which, if not the exact opposite of this, is very nearly so."—Anstie on Neuralgia, p. 2 et seq.

[3] "Genuine angina pectoris is undoubtedly a very rare affection. On the other hand, I *almost daily* meet with a form of complaint combining in a minor degree many of the characters of angina; and to this imitation of the true disease I propose to give the name of pseudo-angina. I believe that herein lies the explanation of Laennec's notion (so discordant with the experience of English observers) that angina pectoris is of very frequent occurrence."—Walshe, Diseases of the Heart and Great Vessels, 4th edit., 1873, p. 208. Compare the observations on Diagnosis in p. 670, of present chapter.

[1] On Neuralgia, and the Diseases that resemble it, by F. E. Anstie, M.D., 1871; pp. 63, 64. The first sketch of this most valuable treatise, contributed by the much-lamented author to the present work in 1868, contains no detailed reference to angina pectoris.

pectoris, and those cerebro-spinal manifestations which sometimes occur, and which we saw well illustrated in the case of John Hunter.

Is it possible to give any account of these three orders of phenomena which shall be consistent and intelligible, which shall be founded on positive facts and well-ordered experients, and shall thus fulfil the purposes, even provisionally, of a reasonable theory of angina pectoris? In endeavoring to answer this question, it will be necessary to refer to physiological researches which are still very incomplete, and even to clinical facts which have not as yet been tested by a sufficient number of independent observers. But it certainly seems as though some large and fruitful lines of research had recently been opened up amid much darkness and confusion.

We owe to Dr. Lauder Brunton[1] the clinical observation of a fact which, besides its therapeutic consequences (to be afterwards considered), may be regarded as shedding a new light upon the pathology of angina pectoris. In investigating a case of rheumatic disease of the aortic valves (obstruction and regurgitation), with dilatation of the aorta, and considerable hypertrophy of the heart, he found that during the angina-like paroxysms of pain to which the patient was subject, the sphygmograph invariably showed a great diminution in the amplitude of the pulse-wave, with blunting of the apex, slow or greatly postponed recoil, and obliteration of the dicrotic wave; the ordinary pulse of the individual (at least in the right radial artery) being characterized by a very ample and instantaneous upstroke, a pointed apex, a rapid recoil, and a distinct though not exaggerated dicrotic wave. Repeated experiments convinced Dr. Brunton that these altered characters of the pulse were due to an increased tension in the systemic arteries during the paroxysm, and that this increased tension was chiefly, if not solely, owing to "contraction of the small systemic vessels, so sudden and so great as to deserve the name of spasmodic."[1] Following up this line of observation, and being aware that Dr. B. W. Richardson[2] and Dr. Arthur Gamgee[3] had performed numerous experiments which showed that nitrite of amyl, when inhaled in small quantities, had the effect of remarkably lessening arterial tension by diminishing the contraction of the arterioles, Dr. Brunton was led to employ this substance for the purpose of relieving the symptoms in this case, and had the great satisfaction not only of finding that almost immediate ease was given in the severer paroxysms, but that the observations previously made on the relation of the paroxysm to increased vascular tension, were *enphasized* (so to speak) by the action of the nitrite of amyl. For when in the severest paroxysms the pulse was almost annihilated to the finger (though still regular and somewhat accelerated), thirteen drops of the nitrite of amyl inhaled from a cloth produced, in one minute and twenty seconds, a decided effect at once on the sphygmographic tracing and on the pain; while one or two smaller doses, repeated over sixteen minutes, restored the amplitude of the pulse-wave, and entirely removed the pain. It is, perhaps, unnecessary to multiply details, especially as regards doubtful points.[4] The experiment was

[1] Lancet, July 27, 1867, p. 97; Journal of Anatomy and Physiology, vol. v. p. 92; Trans. of the Clinical Society of London, vol. iii. p. 191. The case, which is fully recorded in the Clinical Society's Transactions, was that of a man aged twenty-six, admitted into the Royal Infirmary of Edinburgh under Professor Maclagan, on Dec. 7, 1866; and sphygmographic observations, begun at his instance, were continued under Prof. Bennett, to whom the case was transferred on Feb. 1, 1867. There were palpitation of the heart, and violent throbbing of the carotids, besides the angina-pain. The aconite and digitalis were ordered by Professor Maclagan; the small bleedings by Professor Bennett.

[1] Clin. Soc. Trans., *ubi supra*, p. 199. A lithograph, with eleven tracings in different states of the patient, is given, on which the description in the text is founded.

[2] Dr. Richardson's numerous and valuable reports of experiments on anæsthetic vapors, and on nitrite of amyl, from 1863 onwards (brought in successive years before the Brit. Association of Science), determined the power of this substance as an anti-spasmodic and paralyzing agent, and made numerous suggestions as to its probable curative value in tetanus, asthma, and other spasmodic diseases. Dr. Richardson also repeated, and investigated scientifically, Guthrie's accidental observation in 1859, as to its effect in dilating the capillaries; and he inferred that this effect was due to its paralyzing the arterioles through the vaso-motor nerves.

[3] Dr. Gamgee's (unpublished) experiments were made with the sphygmograph and hæmodynamometer, and led directly to Dr. Brunton's trials of the nitrite of amyl in angina, by demonstrating in animals and in man its action in lessening arterial tension.

[4] There is an ingenious attempt to show that a partial restoration of the original form of the pulse-tracing, which was shown to correspond to a remission, but not cessation, of the paroxysm under nitrite of amyl, was due to the persistence of abnormal tension in the pulmonary circulation, after the systemic had been relieved. The pain, under such circumstances, "disappeared from the greater part of the cardiac region, the neck, and the arm, but remained persistent at a point about two inches to the inside of the right nipple,

repeated sufficiently often to show that in this patient at least, increased arterial tension and angina-spasm were constantly associated, and that agents which produced diminution of the arterial tension always relieved the paroxysms. Among these agents, it is to be noted (though none was nearly so powerful as nitrite of amyl), small blood-lettings (of four ounces) were found to exercise a noted influence. Digitalis, on the other hand, appeared rather to aggravate the pain, and both digitalis and aconite made the pulse intermit, which was never the case with the nitrite. On the whole, it must be admitted, that notwithstanding certain unavoidable deficiencies, the experiment is as complete as can reasonably be expected in the evidence it affords of a correlation of some kind between angina-paroxysms and increased arterial tension, in at least one clearly-defined case of organic cardiac disease.[1]

Many other experiments, both on man and on animals, have been performed, which amply confirm the action attributed to the nitrite of amyl in this case. The therapeutical part of the subject will receive consideration afterwards; in the mean time it is sufficient to say that the relaxing effect of the vapor on the arterioles, and its efficacy, in some cases at least, in greatly and instantly relieving the breast-pang, have been placed beyond reasonable doubt.

The points still open to further investigation seem to be these: It is as yet not *proved* that all the forms, and all cases of angina, are characterized by increased arterial tension during the paroxysm. If, indeed, there be cases corresponding exactly with the original description of Heberden, cases in which (the heart being to stethoscopic and physical examination normal) "the pulse is not disturbed by the pain," it would be extremely desirable to have sphygmographic observations of such apparently uncomplicated angina-paroxysms. But we have already expressed doubts of the existence of such cases; at all events, the one recorded by Dr. Brunton is not such a case, but rather one in which the phenomena of the arterial tension must be regarded as wholly abnormal, being influenced by the fact of aortic regurgitation, a strictly mechanical cause of permanently and morbidly *lowered* blood-pressure in the arteries.

But again: Supposing it proved that a suddenly-developed and decided increase in the arterial tension is a characteristic, or even an essential feature of the true angina-paroxysm, we may still regard it as an open question whether the change in the blood-pressure is to be attributed entirely in such cases to contraction of the arterioles, or partly also to changes in the innervation of the heart itself, which would account at once for the pain and for the sudden death which sometimes occurs during the attack? Dr. Brunton has himself pointed out a fact which tells in this direction, notwithstanding the elaborate reasonings by which he supports the theory of vaso-motor derangement ending in spasm of the arterioles as the starting-point of the paroxysm. The experiments of Marey and others have shown that the effect of high blood-pressure in the arteries, *per se*, is to retard the pulse; while diminished arterial tension arising from relaxation of the arterioles (as in fever, or in capillary congestion from the effect of external warmth) increases the frequency of the heart's contractions. Now in the case alluded to, what actually took place was exactly the reverse of what might have been expected on the theory above mentioned. During the severest paroxysms, when arterial tension was at its height, the pulse was *small and rapid*, and when the pain and spasm had been subdued by the inhalation of the nitrite, the pulse diminished in frequency while regaining strength and volume. Dr. Brunton considers these phenomena as indicating "a derangement of the cardiac regulating apparatus, producing quickened instead of slowed pulsation." Further observations, therefore, seem to be required before it can be safely assumed that either vaso-motor derangement on the one hand, or disorder of the cardiac innervation on the other, is the primary or *essential* phenomenon of true angina pectoris; although we may probably take it as provisionally established that some law of intimate relation exists between increased blood-pressure in the arteries and certain forms, at least, of the angina paroxysm.

The peculiar interest of Dr. Brunton's observations, for us, consists not in his having finally settled the *nature* of this relation, but in his having shown that a remedy which has the remarkable power of instantly diminishing arterial tension

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. . . So long as this condition remained the pain was almost certain to return."—Clin. Trans. iii. p. 199.

[1] It is to be observed, that although the diagnosis actually made was that of aortic obstruction and regurgitation *without aneurism*, and although this was quite in accordance with the physical signs, and particularly the murmurs, described in the report, the remarkable difference in the sphygmographic tracings of the two radial pulses cannot but be regarded as leaving a doubt open as to the negative part of the diagnosis. On the other hand, aneurism, if present, may have been responsible in part for the definite character of the pain, which is usually not so well marked in cases of aortic regurgitation simply.

has also a corresponding and almost equally instantaneous control over those paroxysms of angina in which increased arterial tension is known to occur. We shall recur to this subject when speaking of treatment.

Meantime it seems necessary to observe that Dr. Brunton had been anticipated, in several quarters, in the merely speculative attempt to connect the symptoms of angina pectoris with vaso-motor changes. Thus Traube[1] had argued that the diminished volume and increased tension shown in the arteries in many attacks of stenocardia are to be viewed, in connection with the increased rate of the pulse and the feeling of anxiety (angstgefühl), as related to an increased stimulation of the nerve-centre of the vaso-motor system. Cahen[2] had treated at length of various neuralgic affections (including trifacial neuralgia, and various painful affections of the pelvic organs) as affections of the vaso-motor system of nerves attended by congestion; and he referred angina pectoris to the same category, and indicated arsenic as a valuable remedy for such cases, without, however, adding anything important to the symptomatology of angina. Landois[3] had made a somewhat similar generalization as to some cases of excessive nervous palpitation, which he regarded as being a vaso-motor angina pectoris. Finally, Nothnagel, in a very ingenious and interesting contribution to the clinical study of the "vaso-motor neuroses," devotes an entire article[4] to the special consideration of "Angina Pectoris vaso-motoria," upon the basis of five detailed cases (without special sphygmographic observations). But the details of Nothnagel's cases will show that, however closely some of the subjective symptoms of angina pectoris may be simulated by a purely vaso-motor lesion, there are some very striking differences between the disease so induced and the true angina pectoris of Heberden. For—1st, in the greater number of Nothnagel's cases the disease yielded easily to very simple treatment, and in none was there a fatal issue, or even, apparently, much real apprehension of immediate or urgent danger; 2dly, the sensations in the extremities (deadness, coldness, formication, *not* pain) were usually present in *all* the extremities indifferently, and *preceded* the palpitations and the cardiac uneasiness by some minutes; 3dly, the specially cardiac or other internal sensations were, a very distressing sense of palpitation, attended by anxiety, and sometimes by vertigo, or incipient faintness; 4thly, in one of these cases only was the pulse-rate decidedly altered, and in that case it was diminished from 84 to 64—60 during the attack; 5thly, pain was either absent, or assumed little prominence among the symptoms; 6thly, the sensation of impending death was evidently *connected with, and probably caused by, the palpitation* (in Heberden's most characteristic case above quoted,[1] as also probably in John Hunter's case, the very opposite of this was the fact; the feeling was of "a pause in the operations of nature for perhaps three or four seconds"). 7thly, several of the cases recorded were below the typical age (30, 38, 39, 46), and one only above it (63); that one being a woman. The lesson, therefore, taught by Nothnagel's cases is not, properly speaking, that typical, still less that fatal, angina pectoris is always to be regarded as due to vaso-motor spasm, but rather that, under certain peculiar conditions of the system, a sudden check to the circulation in the extremities, determined by vaso-motor spasm, may become cause of an increased action of the heart, palpitation, and *pseudo*-angina; the disease so induced, however, being devoid of the characteristic pains and the more aggravated phenomena of fatal angina; and that in such cases heat, and mild counter-irritation of the surface, have almost complete power to control both the external and internal manifestations; the prognosis being (according to N.) entirely favorable. At the same time, although we cannot admit that Nothnagel's cases were genuine cases of Heberden's angina, they are very instructive, and may, no doubt, afford some insight into the pathology of the true disease.

Leaving, for the moment, the line of inquiry suggested by these observations, we may revert to the *pain* of angina, which has been commonly regarded as a neuralgia of the cardiac plexus; the impressions of pain in the severer cases being radiated outwards through the numerous connections which are known to exist between the special ganglionic system of the heart, and the spinal nerves entering into the cervical and brachial plexuses through the cervical ganglia. It is difficult, from the very nature of the case, to prove this proposition; but there is no inherent improbability in it, unless, indeed, we should assume that the cardiac nerves of the ganglionic system are incapable of giving rise to acute pain; an as-

[1] Die Symptome der Krankheiten des Respirations- und Circulations-apparatus, p. 41. (Ref. in Rothnagel's article, *infra*.)

[2] Archives Générales de Médecine, 1863, vol. ii. p. 564.

[3] Correspondenz-Blatt für Psychiatrie, 1866 (quoted by Nothnagel).

[4] Deutsches Archiv. für Klinische Medizin, vol. 3, xiv. p. 309. Compare also vol. 2, p. 190, Case VII.

[1] See page 667, note.

sumption not in accordance with the facts of medical experience in the cases of gall-stone, colic, &c. Holding in view, moreover, the proved association of angina pectoris in many cases with disease of the coronary arteries of the heart, and with other lesions exclusively within the range of the ganglionic system of the heart and aorta, it is difficult to resist a bias in favor of the view that the nervous system of the heart itself is the origin or the chief seat of pain, in the great majority of the cases. To these arguments it may be added that in most cases the internal sensations (whether distinctly referred to the heart or not by the patient) are obviously first in the order of time and of degree; the brachial, intercostal, or cervical pains being sometimes altogether absent, and usually present only in the more severe and protracted attacks. It has, however, been plausibly maintained, notwithstanding these facts, that the spinal nerves are the true seats of the apparently cardiac pains of angina, and that all the apparently reflected sensations in the limbs, &c., are transmitted, like the external neuralgiæ, through a spinal centre. Dr. Anstie, who holds this view, adduces the unilateral character of the brachial pain in at least four cases out of five (?), as an almost irresistible argument against the radiation of pain outwards from the cardiac ganglia, through the peripheral nerves of communication. "It appears greatly more probable," he writes, "that angina is essentially a *mainly unilateral morbid condition of the lower cervical and upper dorsal portion of the cord;* liable, of course, to be seriously aggravated by such peripheral sources of irritation as would be furnished by diseases of the heart, and especially by diseases of the coronary arteries." This question is one which can scarcely be made less obscure by any arguments falling within the scope of this article.

We have already indicated some of the difficulties that have to be encountered in extending the group, or order, of the *neuralgiæ* so as to include angina pectoris; meaning by that term, of course, the formidable and fatal disease we have been chiefly describing, and not the very numerous, or rather innumerable, instances of pains referred to the heart, by hysterical women and others, which have no such significance. Referring chiefly to fatal cases of angina pectoris, Sir John Forbes and all the more considerable authorities from Heberden downwards concur in giving an immense preponderance to the male sex. Without insisting too much on the numerical details, which for reasons formerly indicated may perhaps be considered as somewhat biased by the mode of collection, it may be well to compare this overwhelming proportion of males who fall victims to cardiac angina (an excess on the male side greatly exceeding the greater proclivity of males to organic disease of the heart in general) with the numerical statements given incidentally in Dr. Anstie's work as regards the liability of the two sexes to neuralgiæ in general. "Eulenburg saw a hundred and six cases of neuralgia of all kinds, of which seventy-six were in women, and only thirty in men: my own experience is very similar; viz., sixty-eight women and thirty-two men out of a hundred hospital and private patients."[1] A difference so extreme as this is not to be accounted for "by supposing that as men take a much larger amount of strong physical exercise than women, they will furnish a much larger proportion of subjects in whom an ill-nourished heart will break down under its work, and be seized either with paralysis or cramp;"[2] and it seems scarcely necessary to do more than place these facts before the reader, in order to make it apparent that many of the arguments by which analogies drawn from the study of neuralgia in its more familiar forms are applied to angina pectoris, are questionable, if not altogether unsound. And yet I wound by no means be understood to deny that persons hereditarily predisposed to neurotic diseases, and especially to those of advanced life, may be specially liable, *cæteris paribus*, to angina in its more painful forms. Much care, however, is necessary in sifting facts and details of symptoms when recorded with a view to make good a general theory of this kind; and when we are called upon to accept a narrative of *epidemic* angina pectoris in a ship's crew, in which "numbers of men were simultaneously affected," while others were seized with "other forms of neuralgia, and severe colics,"[3] I cannot but infer that the limits of a safe induction have been considerably exceeded. In like manner, "remarkable" cases of "hysteria, the paroxysms of which were always accompanied by stenocardiac attacks," can only serve to give a doubtful character to the theoretic interpretations which Eichwald has obtained

[1] Op. cit. p. 156.

[2] Ibid. p. 72. This might be a valid hypothesis were it possible to affirm that the subjects of fatal angina are chiefly drawn from the class of men that take the greatest amount of strong physical exercise. The opposite, however, is notoriously the fact. We have already alluded to the generally received statement of Sir John Forbes, that angina pectoris is "the attendant rather of ease and luxury than of temperance;" and that it is comparatively rare (in its simple and typical form), among the laborious classes.

[3] Ibid. p. 74. The authority given is Guélineau, Gaz. des Hôpitaux, 1862.

from such a field of experience.[1] And even Eulenburg, notwithstanding the sobriety of his tone in general, and the great importance of his work as a magazine of valuable information and research, has shown how much a sound clinical observation has been subordinated to theoretical ideas, when he pronounces dogmatically that the disorders of respiration in angina are merely "consequences of the pain; the patient is afraid to inspire deeply, but if induced to do so, can generally accomplish it."[2] It may be doubted, I think, on the whole, whether much real knowledge has been gained by the classification of angina pectoris among the neuralgiæ; to which, nevertheless, the character of its pain shows a remarkable affinity.

Proceeding now to consider the motor derangements which form a part of the angina-paroxysm, and especially those which, affecting the heart itself, determine the fatal termination, it is impossible to overlook the facts brought to light by physiology as regards the influence of certain nerves on the movements of the heart. In particular, the remarkable inhibitory influence of the efferent nerves proceeding to the heart through the pneumogastrics, demonstrated by the brothers Weber[3] in 1846, and in 1856 shown by Waller[4] to be due to filaments from the spinal accessory nerves joining the pneumogastrics near their origin, has a peculiar interest for us in connection with this subject. It has been conclusively shown that by a galvanic current transmitted outwards through these filaments, or by galvanization of the centre in the medulla from which they are derived, the heart's action may be controlled, or even stopped, so that a true cardiac paralysis is the result of a strong current, while weaker galvanic action produces an indefinite retardation in the rate of the cardiac pulsations. Whatever theory be adopted as regards the so-called inhibitory influence, its results are too closely allied to the phenomena of syncope, pure and simple, to escape attention in treating of sudden death from angina. But it has been further shown by Cyon and Ludwig,[1] that a reflex influence may be so transmitted through nerves arising from the pneumogastrics (viz., the so-called depression-nerves), as at once to control the cardiac pulsations through the inhibitory efferent nerves, and to diminish tension through the vaso-motor system. As we have already seen reason to believe that in angina pectoris the vascular tension is usually increased rather than diminished, it may be inferred with great probability that if the pneumogastric nerve be implicated at all in the angina-paroxysm, it is probably more as an inhibitory or efferent, than as a reflex or efferent nerve. It must not be forgotten, however, that paralysis of the sympathetic nerve has the effect also of enfeebling and retarding, though not, apparently, of stopping, the heart's action; which, in a certain sense, may be regarded as not essentially dependent upon influence transmitted from any nerve-centre, though subject, as we have just seen, to control through the inhibitory or efferent cardiac filaments of the pneumogastric.

If we endeavor now to determine, in the light of these facts, what is the particular mode in which the heart's action is suddenly arrested in a paroxysm of angina, it must be confessed that no ultimate decision seems possible. Almost all the vague and unsatisfactory speculations formerly alluded to, as to whether *spasm* or *paralysis* is the prevailing condition in the fatal paroxysm, have proceeded on the assumption that these two conditions are essentially contrasted, or rather opposite to, and inconsistent with, one another; the former representing undue strength, the latter undue weakness, or absolute annihilation of contractile energy. Now this assumption can by no means be regarded as a legitimate, or even a probably correct one. At least it may be fairly affirmed, as a probable result both of physiological and pathological inquiries, that spasm (*i. e.*, irregular or abnormal contraction, whether painful or not) in a voluntary muscle is much more allied to weakness, or to deficient innervation, than to absolute excess of normal energy. And the frequency of the association of rigid or tonic spasm with paralysis, in the voluntary muscles, would tend to show that there is no absolute inconsistency, at least, in the supposition

[1] See Eulenburg, *infra*, p. 433. Perhaps the same remark applies to the presumed relationship between angina pectoris and spasmodic asthma, as indicated by Kneeland, Amer. Journal of Med. Science, Jan. 1850, and Anstie, op. cit., p. 68. It is to be remarked that Trousseau, in his vast and varied experience, has not recorded anything tending to confirm the relationship of these two neuroses, except in a case where both of them were dependent on aneurism of the aorta. See his Clin. Med., English translation, vol. i. p. 634.

[2] Med. Times and Gazette, March 26, 1870, p. 329. We have seen how emphatically this idea is contradicted by the specific statements in John Hunter's case, as well as by all the most exact clinical observations from Heberden downwards.

[3] Wagner, Handwörterbuch der Physiologie, Bd. iii., 2te Abtheilung, S. 42.

[4] Gazette Médicale, Paris, 1856, t. xi. p. 420.

[1] Journal de l'Anatomie, Paris, 1867, t. iv. p. 472.

that *both* spasm and paralysis may, in varying degrees, be present in the heart's arrested action which leads up to sudden death in the angina-paroxysm. As far as observation goes, in the case of spasm of the involuntary muscles (other than the heart), it seems as though abnormal, or painful, disturbance of rhythmic action were almost always an indication of weakened innervation, rather than of superfluous energy in the contractile apparatus as a whole. The spasm of colic, for instance, is associated with constipation, or deficient peristaltic action of the intestines; the false pains, or painful spasms, of the uterine muscles retard, instead of expediting, the process of delivery. We might, therefore, not unfairly argue from these analogies, that a painful spasm of the heart might be expected to interfere with its rhythmic or normal action quite after the manner of a paralysis, the abnormal being substituted for the normal action, and the whole sum of disordered effort being less than the sum of normal energy expended in healthy cardiac action. So that it might very well be presumed that painful spasm is by no means unlikely to be associated with a tendency to sudden stoppage of the heart's action, or virtual paralysis, whether from inhibitory nervous irritation through the pneumogastrics, or from disorders originating in the cardiac ganglia themselves, and allied in character to true paralysis of muscular energy. It must, however, be conceded to the advocates of the theory of paralysis, pure and simple, that nothing but the presence of severe pain in the angina-paroxysm, and the absence of this symptom, as a rule, in purely paralytic affections, tends to support the spasm-theory of angina. Post-mortem examinations have generally shown that the heart is found flaccid, rather than rigidly contracted; and the lesions found in the muscular substance of the heart itself are usually such as would confirm the idea of decidedly and permanently weakened energy, rather than a disposition to abnormal contraction. Rupture of the muscular bundles, so commonly observed in tetanus and other severe spasms of voluntary muscles, has never been recorded in sudden deaths from angina pectoris; while anæmia, fatty degeneration, and fibro-tendinous substitution, have been the predominating lesions of the muscular fibres itself. The question as between spasm and paralysis, therefore, is one of great difficulty, if not indeed practically insoluble in the present state of our knowledge.

While dealing with hypotheses of which no absolute or experimental proof can be obtained, we may remark that vaso-motor spasm, operating indirectly through the smaller arteries upon the muscular fibre of the heart itself, may possibly give a clue to some of the pathological changes which attend the paroxysm, and especially those which precede dissolution. Both Erichsen[1] and V. Bezold[2] have shown that as a result of deligation or occlusion of the coronary arteries, the heart's contractions become feeble or irregular, and ultimately cease; the normal action being restored again on removal of the ligature or of the compression. Now apart from the obvious bearing of these facts upon the case of organic obstruction or constriction of the coronary vessels (perhaps the most clearly established of all the permanent organic changes in connection with fatal angina pectoris), is it not extremely probable that a similar effect, or an aggravation of a pre-existing tendency to interrupted cardiac action, might occur, if in a case of disease of the aorta or coronary arteries, cardiac anæmia were aggravated for the moment by vaso-motor spasm of the smaller arteries within the heart itself? Even without such preceding organic disease it is conceivable that extreme vaso-motor spasm might affect the cardiac circulation directly through its smaller arteries, and so produce changes more or less similar to those observed in the experiments above mentioned. What has been already stated, however, in regard to Nothnagel's observations would seem to show that really fatal angina does not occur in this way; and that the first effects of general vaso-motor spasm upon the heart are more of the nature of palpitation, or excited action, than of interrupted or suspended pulsation.

On the whole, it must be admitted that the ultimate pathology of the angina paroxysm does not admit of being reduced to any very precise expression or definition; but various more or less probable conjectures may be made, in accordance with known facts and experimental researches, as well as with clinical and pathological observation, to account for the facts. Viewing the paroxysm as a *neurosis*, we might attribute its phenomena partly to vaso-motor spasm, and partly to inhibitory influence transmitted through the vagus nerve from the medulla oblongata. This latter influence would account more reasonably and probably than any other for those cases of angina in which mental causes and sudden shocks of any kind are known to influence the production of the paroxysm, without the intervention of peripheral changes such as can be attributed to vaso-motor spasm. In cases, again, resembling in their symptoms those described by Nothnagel, whether accompanied by organic disease or not—cases in which coldness of the surface, deadness

[1] London Medical Gazette, July 8, 1842.

[2] Centralblatt für die Med. Wissenschaften, 1867, No. 23.

of the extremities, and perhaps palpitation or increased rate of the pulse can be ascertained to precede the cardiac pain, there would be reasonable ground for presuming that the vaso-motor nerves were the earliest involved in the morbid circle, though it is still probable that, if such cases ever end in sudden death, it is through some more direct impression on the cardiac nerves, or on the coronary circulation. It is very doubtful, however, whether under any circumstances fatal angina pectoris can be viewed as a pure *neurosis*. Much more probably, the paroxysm is the expression in symptoms of sudden changes arising, indeed, from neurotic accidents, but only assuming grave importance in respect of their coincidence with a permanent cause of detriment to the circulation. Either the heart's fibre is permanently weakened, or its arteries are obstructed and diseased, or the general arterial circulation is disturbed through disease in the first part of the aorta, aneurismal or other. In certain cases it may be that the innervation of the heart is directly implicated in organic disease; at least in two cases of this kind[1] the cardiac plexus and cardiac branches of the vagus were found to be compressed in connection with angina-paroxysms which proved fatal; though probably the inferences which have been drawn from these rare instances may not be applicable to the general pathology of the subject. But whatever be the nature of the permanent change underlying the disease, its effect in the most characteristic cases is not much felt when the circulation is in a moderately tranquil state. In some of the very worst cases, indeed, it has been clearly ascertained that very shortly before a fatal paroxysm the patient has been in a state of entire comfort and tranquillity, with a regular and normally acting heart, and all the functions apparently so well-adjusted as to involve no appearance of any disease tending to shorten life. Usually there is an incapacity for sudden or severe exertion, and a liability to grave disturbance under strong emotion; but, on the other hand, a patient has been known to say, *within three days of his death in a paroxysm*, "I can walk with ease ten or fifteen miles, after I have been stopped three or four times at intervals of a hundred yards."[1] In such cases the paroxysms are plainly neurotic; but the disease is nevertheless not a pure neurosis. It is, on the contrary, obviously of a complex character, involving a permanent nucleus, so to speak, of organic change, together with a neuralgic element, more or less pronounced, and, connected with this, perhaps as a reflected neurosis in some cases, an element of motor disturbance in the heart's action, which may in some cases be of vaso-motor origin, while in others it may be more directly determined through the inhibitory filaments of the vagus. It is probably in the former class of cases that the action of nitrite of amyl is most immediately and surely productive of benefit.

There remains for remark only one obscure, and apparently non-essential, part of the pathology of angina pectoris, viz., the nature of the cerebral accidents we have indicated in the description of the disease as sometimes coinciding, sometimes alternating, with the more decidedly cardiac attacks. It is to be observed that among these accidents spasms, giddiness, temporary attacks of coma, associated with, or followed by, various disorders of the special and general sensibility, are common; while on the other hand, paralysis, either spinal or cerebral, is rare. These facts point strongly in the direction of a neurosis, and very probably a vaso-motor neurosis, of the cerebral circulation; and we know that in animals most of the symptoms above referred to may be induced artificially, by cutting off the arterial vascular supply of the brain and medulla oblongata, as in the well-known experiments of Sir Astley Cooper.

The *Prognosis* of angina pectoris is difficult to realize in individual cases, in proportion to the absence of clear lines of distinction between this and the various affections resembling Heberden's angina, which we have discussed in various parts of this article. Probably a critically exact, or absolute, prognosis, could only be founded on a knowledge of the nature and extent of the organic changes underlying the paroxysmal neurosis; and although we have already indicated a doubt as to whether the latter ever terminates fatally in the absence of such organic changes, yet it is beyond all question that the amount of organic disease which can be detected in any given case during life is a most insecure guide in estimating the

[1] Heine, in Müller's Archiv, 1841, p. 236; and Lancereaux, in Gazette Médicale, 1867, p. 432. In the former case the heart was at times observed to cease beating for several seconds, and at these times there was a feeling of indescribable anxiety, like that of angina pectoris; in the intervals of the paroxysms the patient felt perfectly well. The right phrenic nerve, the nervus cardiacus magnus, and the pulmonary branches of the left vagus were all involved in, or compressed by, calcareous deposits. In Lancereaux's case, the cardiac plexus was found vascular, and compressed by exudation; but the coronary arteries were also obstructed, and the aorta was diseased. The patient died of angina pectoris, in a paroxysm.

[1] Walshe, Diseases of Heart and Aorta, 4th edit., p. 199, *note*.

probabilities of death during a paroxysm in that particular case. "It is accordant with my experience," says Dr. Walshe, "that fatal angina is more to be dreaded in association with organic defects, either difficult or impossible to diagnose (such as slight fatty metamorphosis and calcified coronary arteries), than with those grave forms of structural mischief that are readily discoverable by physical examination."[1] Add to this that the mere inference from symptoms as to the gravity of the prognosis is likewise extremely open to fallacy; inasmuch as a series of comparatively mild or lessening attacks may sometimes (under apparently unchanged conditions) be succeeded by the most violent or dangerous, even fatal, paroxysms; while on the other hand, one or more attacks, very nearly fatal, may be followed by a long interval of comparative, or nearly complete, freedom. From this dilemma there is, in the present state of our knowledge, no escape; and all that we can do, therefore, towards the establishment of a guarded and limited prognosis in any case, is to study carefully its individual features, and particularly the relation of the symptoms to particular causes of aggravation, or of relief. Generally speaking, a form of disease which yields, gradually, to carefully pursued hygienic treatment, and in which the paroxysms are obviously under the control of the remedies about to be discussed, is relatively favorable; while the opposite indications justify the gravest prognosis. An absolutely favorable prognosis could only be justified by circumstances tending to place the disease in the category of pseudo-angina, as above indicated; and indeed it may be generally observed that the gravity of cases of angina in the experience of individual observers is often found to be in an inverse proportion to their estimated frequency, cases of hysteria, intercostal neuralgia, spasmodic dyspnœa, &c., being admitted by some more freely than others into the category of angina. There seems no reason to doubt, however, that a person affected with absolutely typical angina pectoris may survive for years, even after repeated paroxysms; and in some cases, apparently of the most threatening kind at one stage of their progress, the disease has been so far reduced in its frequency and severity that we may even, perhaps, speak of such cases as cured, in a practical sense. But cures of this kind are rarely, if ever, recorded with such minute attention to details as to inspire confidence, apart from the credit due to the reporters; and perhaps even the statements of Heberden as to the long survival of some of the cases mentioned in his first paper (see p. 680, *note*) may require qualification on the ground that clear evidence is wanting as to the absolutely typical character of the symptoms referred to.[1] Among cases actually ending by a fatal paroxysm, it has not occurred to me personally to have been informed of a longer duration than six or seven years, counting from the first well-defined seizure; but I have known more than one instance of survival for much longer periods, after attacks bearing so much resemblance to true angina as only to have required death to have occurred in a paroxysm, as a conclusive argument for considering them to be typical instances of the disease. In John Hunter's case, as we have seen (assuming the first attack of supposed gout in the stomach to have been really identical in character with succeeding seizures) a duration of rather more than twenty years, with numerous intervals of tolerable health and great mental activity, may be regarded as well established. Dr. Walshe has "met with an instance in which there was the strongest evidence that the first paroxysm had occurred *twenty-four* years prior to my interview with the patient."[2] And, in the general experience of physicians who have had occasion to see much of cardiac disease, it is by no means uncommon to find cases of valvular or other very positive and well-defined organic disease, in which symptoms of a dangerous or proximately fatal kind, probably more or less allied to angina, have preceded the fatal issue by an interval of very many years; sometimes, indeed (as in the case of the Rev. Dr. Guthrie, already referred to[3]) for more than a quarter of a century. Such cases, however, are rarely quite typical instances of Heberden's angina, and accordingly only a small proportion of them are characterized by the very sudden ending proper to the disease as described in the "Commentaries." It is difficult to obtain exact clinical histories of cases extending over so many years, but in one, in which I was consulted in 1872, and which terminated fatally some months ago, there was reason to suppose

[1] Op. cit. p. 201.

[1] It is at least worth noting (though the omission may be accidental) that in the Commentaries these statements are not repeated; and perhaps the language, though carefully guarded, admits of the inference that thirty years of additional experience had rather increased than diminished Heberden's sense of the gravity of the prognosis. "*Exitus hujus affectus est perquam memorabilis. Qui enim eo tenentur, siquidem, nullo casu interveniente, angina pectoris ad ἄκμην pervenerit, omnes repente corruunt, et fere momento pereunt.*" . . . "*Unicum vidi (ægrum), in quo hoc malum sponte suâ finitum est.*"

[2] Op. cit. p. 200.

[3] See *ante*, p. 686, *note*.

that the foundations of the aortic valvular lesion of which the physical signs were apparent, and of which the obvious symptoms had certainly existed many years before 1872, had been laid as early as 1852, when the patient had suffered from pulmonary hemorrhage. The threatening symptoms present on that occasion had been popularly attributed to a consumptive tendency, but Dr. Christison, who was consulted, had evidently detected some valvular lesion of the heart, and had carefully questioned the patient as to its possible rheumatic origin. It is not, indeed, certain, or even perhaps very probable, that well-marked and considerable aortic regurgitation existed at this period, nor is it possible now to ascertain at what precise interval after the first commencement of the disease the angina-like symptoms, which were notably present when I saw the patient, first became apparent. What I can personally affirm is, that in 1872 the symptoms and physical signs were those of old-established aortic regurgitation, with very considerable hypertrophy of the left ventricle, and all the usual concomitants; and notwithstanding this, the patient assured me that so late as 1870 he had explored the Aletsch glacier, and on other occasions, from about 1865 onwards, had been able to carry out walking tours in Switzerland, the Tyrol, and the Dolomite country, the character of which may be inferred from his having walked over the Monte Moro pass and the Gemmi, visited the Mer de Glace, and gone nearly to the Jardin, in addition to all the usual excursions about Chamounix. Moreover, this gentleman was in 1872 performing the duties of a parish clergyman in a populous place, sparing himself somewhat, indeed, in visiting, but preaching often more than once a day, and, as he affirmed, without any apparent injury or physical exhaustion; and the question most urgently and repeatedly pressed upon his medical advisers was as to his carrying out an engagement of marriage, entered into several years before, and maintained with full knowledge on both sides of the precarious condition of his bodily health. I need not say that no medical encouragement to this step could be obtained; but the marriage, nevertheless, took place in about a year after I was first consulted, and the death of this patient not long ago shows how real was his danger, and at the same time what a terrible burden of positive organic disease may be borne without apparently "giving in," by one whose objects in life are of sufficient importance to induce him to disregard the silent warnings of internal suffering. In yet another case known to me, in which, however, the symptoms were far more decidedly and typically those of angina pectoris, while the physical signs were much less manifest than in the preceding case, the patient was able to make numerous long journeys to the Holy Land, Egypt, &c., and always felt himself the better for them. This patient in the end perished suddenly.

The TREATMENT of angina pectoris resolves itself naturally into two departments, viz., that of the paroxysm, and that of the intervals. The former treatment is essentially palliative, and directed exclusively to the urgent symptoms then existing; the latter aims at being founded, in a wider sense, upon the diagnosis and prognosis of the individual case, after a complete examination into the state of all the bodily functions.

Heberden's views of treatment were limited to the first indication—the control of the paroxysm. "Wine and cordials taken at going to bed will prevent, or weaken the night fits; but nothing does this so effectually as opiates. Ten, fifteen, or twenty drops of the tinctura Thebaica taken at lying down will enable those to keep their beds till morning who had been forced to rise, and sit up, two or three hours every night for many months."[1] We have already seen that Heberden altogether repudiated the (so-called) antiphlogistic treatment as inapplicable to this disease, which he considered as belonging to the order of spasms, not of inflammations. In his later work he repeats in general terms the above recommendations, and adds to them a single phrase in favor of rest and warmth. He has seen an approach to a cure in one case, where the patient prescribed to himself the labor of sawing wood for half an hour every day. Beyond this, he has little or nothing to tell, and does not profess to have greatly advanced the cure of a disease, "qui vix ad huc locum, aut nomen in medicorum libris invenit." It may be fairly inferred from these expressions, that Heberden's views of the treatment of angina remained almost stationary for at least thirty years; and that here, as in the matter of prognosis, he does not appear to have gained confidence with his advancing experience. The treatment of the paroxysm by opiates and stimulants of various kinds has in fact been repeated by almost all the leading authorities, and is even now the only medical treatment which can be said to have received general assent. Latham, Stokes, and Walshe, among our more modern authors, concur in recommending from forty to sixty drops of laudanum, together with wine, brandy, or aromatic spirits of ammonia, repeated according to the violence of the paroxysm. Hoff-

[1] Med. Trans., vol. ii., *ut supra*.

mann's anodyne, or sulphuric ether in half-drachm doses, has been a favorite remedy with many; and musk, camphor, and other anti-spasmodics, have also been employed, though confessedly of less value than ether, which has also been followed by good results when administered by inhalation. Of late years, opium has been given hypodermically, and, it is stated, with more immediate as well as more successful results than when administered by the mouth. In so far as the principle of the treatment can be inferred from the success that has attended those remedies in some cases, it would appear that a rapidly induced narcotism, benumbing the sensory nerves and extending, perhaps, to the centre through which painful sensory impressions are reflected in the form of a paralyzing or inhibitory influence on the heart, by the motor fibres of the pneumogastric, is the first object to be accomplished in the presence of overwhelming pain, while the second and not less important object is to stimulate the heart's action by all the known excitants of the circulation. Warmth to the extremities and to the epigastrium, sinapisms to the thorax, and sometimes between the shoulders or at the back of the neck, may be regarded also as additional means of fulfilling the latter indication, and of assisting the cardiac contractions by their influence on the vaso-motor nerves. In my own experience, no remedial agencies have appeared more powerful than warm pediluvia with mustard, and fomentations applied at the same time to the arms and thorax, as hot as they can well be borne. With these, and with ether and other diffusible stimulants, I have often been able to dispense with the use of large opiates, in doubtful cases, or in cases where they seemed to be in some respects contra-indicated. It is well, if possible, to be informed of the condition of the kidneys, and of the lungs before prescribing opiates. Dr. Stokes[1] evidently looks upon large opiates as unsafe where fatty degeneration of the heart's fibre is suspected: and Niemeyer[2] discountenances narcotics altogether. The use of opium, however, is too valuable in typical cases of Heberden's angina, when apparently uncomplicated, to be readily given up. It should be given with discretion, its effects being carefully watched; and it should probably be withheld, or given in extremely moderate doses, wherever there is risk of uræmia, or of bronchial and pulmonary sudden congestion or œdema, or of the cerebral accidents that accompany angina in certain cases, especially those in which the cardiac fibre is the seat of degeneration. In these cases, too, it is not usual for the mere pain of angina to be so threatening, *per se* as to suggest opium in the same high doses as in the more typical instances where the paroxysm occurs in the midst of apparent good health.

Hydrate of chloral, from its well-marked sedative and anodyne powers, has been suggested as a substitute for opium in cases of painful angina;[1] but on the other hand, the depressing action of chloral-hydrate in large doses has been supposed to be a fatal objection to its employment in cases of weakened cardiac action. My experience of this remedy in severe cases resembling angina pectoris is limited to one case, but it is so remarkable as to deserve notice here. John McN., æt. 35, was subject to paroxysms of intense cardiac suffering, of a rather obscurely painful character, but with considerable orthopnœa, palpitation, sleeplessness, and frightful dreams. His symptoms are more particularly referred to in an earlier part of this article, and from a very careful consideration of them I arrived at the conclusion that they were essentially of the character there described as *angina sine dolore*, with slight bronchitic complication, and slightly albuminous urine—sp. gr. 1013–20. The heart's action was irregular, and the physical signs pointed unmistakably to hypertrophy of the heart and liver, with valvular and (probably) arterial disease. The details are too complicated to be introduced here, but my diagnosis was—Aortic insufficiency, with aneurism. The case was certainly not one in which extreme doses of any narcotic would have been regarded as expedient; but, guided by experience acquired before he applied to me, I allowed this patient to have thirty grains of hydrate of chloral to obviate the sleeplessness, and if possible to ward off the attacks. It answered well the first night, and on a succeeding occasion the same dose was ordered, and was to be given a little before midnight. By a misunderstanding of the directions three drachms of hydrate of chloral were sent instead of a like quantity of the usual syrup, and this being in one dose, apparently to be given as a draught, the patient took 180 grains at once of chloral-hydrate, from the hands of a night-nurse, after a restless and disturbed evening, at 11.30 P. M. Next morning I found him very drowsy, but not quite comatose, as he could be roused to give rational answers as to his own condition; the breathing was quiet, and only slightly stertorous. The pupils were, on the whole, contracted, but variably so; the pulse, which had been irreg-

[1] Diseases of the Heart and Aorta, p. 489.

[2] Text-book of Practical Medicine, American translation, vol. i. p. 371.

[1] Strange, Medical Times and Gazette, Sept. 4, 1870.

uiar in rhythm, was decidedly more natural than before; the face was a little congested, and the eyelids puffy, but the surface generally warm, and the whole appearances not such as to justify any very great alarm, especially as at the time it was supposed that only thirty grains of chloral-hydrate had been given, the mistake being found out afterwards. The patient gradually recovered from the effects of the overdose, and it is very remarkable that he always continued to attribute to this happy accident (as it might be called, speaking of the result only) a comparative immunity afterwards from the angina-like symptoms. The irregularity of the pulse recurred after the effects of the overdose of chloral had passed off, but under repeated doses of from thirty to sixty grains he became much better in all respects, and a course of iodide of potassium, with careful hygienic management, accomplished what, so far as the more immediately urgent symptoms are concerned, may almost be called a temporary cure of a very perilous condition. This man is now performing regulated duties as a railway servant, and is still occasionally taking hydrate of chloral, though warned not to allow it to become a regular habit. It is clear, therefore, that in some cases, at least, of angina pectoris chloral-hydrate might probably with advantage replace opium in the treatment, and that irregularity of the heart's action does not always prove a contra-indication to its use.

Inhalations of chloroform have been proposed, and in some cases employed, for the relief of painful angina; but, from the supposed tendency of deep chloroform-anæsthesia to paralyze the heart, this remedy has never been warmly supported or largely employed by physicians in such cases. The inhalation of ether seems preferable as attended with less risk; and chloroform, if given at all, should be in doses short of complete anæsthesia, whether by inhalation or by the mouth.

Of all the more modern additions, however, to the resources of the physician in the angina-paroxysm, the most important by far appears to be the employment by inhalation of nitrite of amyl, as first recommended by Dr. B. W. Richardson, and successfully carried out on a basis of careful clinical and experimental observation in angina by Dr. Lauder Brunton. We have already indicated in this article the nature of the scientific evidence on which this therapeutic suggestion rests, and have now only to consider the details of purely clinical experience in relation to this remedy, and the qualifications and cautions required in its employment. On this subject our knowledge is still very incomplete, but it is none the less necessary to place on record here whatever can be said to be well established as a guide to the practitioner.

My own experience, I may remark in passing, is certainly favorable to the use of this remedy, not only in positive angina pectoris, but also in many cases of cardiac asthma, and even of true spasmodic asthma without cardiac complication. In the very few cases of typical angina in which I have prescribed it, I have had distinct testimony as to the relief afforded, although my opportunities of close observation of the actual paroxysms have not been such as to enable me to add anything of real value to the statements of other observers. Looking to the practical aspects of the question, there is probably no single observation hitherto made which, as a simply clinical narrative, can rank beside the history of his own case by Dr. W. Herries Madden of Torquay.[1] We shall therefore give here some details of this remarkable personal experience.

Dr. Madden seems to have suffered from a temporary break-down in health at 24 years of age, "with obscure heart-symptoms, and threatened lung mischief." His father had died shortly before from angina pectoris—"the organic cause in his case being atheromatous obstruction of the coronary arteries." In the winter of 1859, at about 44 years of age, Dr. Williams detected slight mitral incompetency. In the spring of 1871, Dr. Madden records that he suffered from an attack of bronchitis, with great nervous prostration, but recovered in autumn, and was able to perform all his usual duties during the next winter and spring, in the midst of "a good deal of professional anxiety and much painful worry of a different nature." On July the 8th, 1872 (at 57 years of age), he had his first attack of angina, which occurred "suddenly, without the slightest warning," and was characterized by "pain extending across the front of the chest, along the inside of the left arm, and across the chin." In about ten days the frequent recurrence and increased severity of the attacks compelled him to desist from all professional duty. Notwithstanding the repose so obtained, the attacks, after a few days' interval, continued to increase in violence, lasting, for the most part, for a quarter of an hour or twenty minutes, and recurring frequently at intervals of about three hours. "Various remedies were tried, but with little or no benefit. Hypodermic morphia was the most useful, but it was impossible to employ it often enough without producing dangerous narcosis." At this period Dr. Madden was led, after considerable hesitation, to give a trial to the nitrite of amyl,

[1] The Practitioner, vol. ix. 1872, p. 331.

which he had previously supposed to be suitable only for those cases in which the face was pallid during the paroxysm. "As mine was flushed," he writes, "I dismissed from my mind all thoughts of trying it, and paid the penalty of hasty conclusions in the shape of a large amount of acute suffering." The result of the first trial of five drops, inhaled during a severe attack in the night, "was truly wonderful. The spasm was, as it were, strangled at its birth. It certainly did not last *two* minutes, instead of the old weary *twenty*. And so it continued. The frequency of the paroxysms was not diminished for some time; but then they were mere bagatelles as compared with their predecessors. Under these improved circumstances, strength gradually returned; the attacks became less and less frequent, and finally ceased. At the time of writing these lines (October 11, 1872) I have not had an attack for five weeks, and have resumed my ordinary duties, of course with care." It is most satisfactory to be able to add, from a private letter with which the author has been favored, from Dr. Madden, that his confidence in the remedy continues unabated, but that at this date (August, 1875) he has not required to use it for a considerable time.

As regards the more obvious effects of the inhalation of nitrite of amyl, Dr. Madden records that "the first effect was often bronchial irritation, causing cough; then quickened circulation; then a sense of great fulness in the temples, and burning of the ears; then a violent commotion in the chest, tumultuous action of the heart, and quick respiration. The angina pain died out first in the chest, next in the left upper arm, and last of all in the wrist, where it was usually extremely severe. . . . When the pain had ceased there was generally for some time a strong involuntary tendency to suspension of breathing, each prolonged pause being followed by a very deep inspiration. There was not at any time the slightest confusion of thought, or disturbance of vision, but occasionally slight and transient headache." The physical signs in Dr. Madden's case seem to have varied somewhat, and latterly had more the characters of aortic than of mitral disease. The description of the peculiar subjective sensations connected with the heart-pang in this case has been already quoted at p. 668, note 2.

It can be but rarely that, in a disease so paroxysmal and uncertain in its characters as angina pectoris, the conditions of a therapeutical experiment can be so perfectly attained as in this case. The hereditary predisposition, the age and sex of the patient, the proved existence of positive cardiac disease, and the vivid and personal narrative of the symptoms, combine in assuring us that the angina was of the most formidable kind, and all but typical, if not indeed absolutely so, in character. On the other hand, the relief was so marked, so strikingly instantaneous, and so frequently observed in repeated paroxysms, as to leave no doubt of the control exercised by the remedy. And further, the ultimate relief amounts to something more than a palliative remedial action; something, indeed, closely approaching the character of a cure. Further, as Dr. Madden has remarked, the relief is shown not to have been contingent upon the external evidences of vaso-motor disturbance during the paroxysm, although closely associated (as in Dr. Brunton's case) with the physiological action of the remedy in relaxing arterial tension. It is to be remarked, however, that beyond the more obvious facts, no very exact observations were made in Dr. Madden's case as to the connection between the attacks of angina and vaso-motor changes. "The presence of intense pain," he says, "is not favorable to the exercise of calm, philosophic analysis, and I can only tell what I felt."

But although this case, and others more or less resembling it which have been published, give the utmost assurance of the beneficial action of nitrite of amyl in the angina paroxysm as a fact ascertained by experience, yet the moment we proceed beyond the mere fact, we find the question of the *modus operandi*, indications, and contra-indications of the remedy surrounded with difficulties which have not as yet been resolved by scientific observation. It has been commonly supposed that the action of the amyl-nitrite is purely peripheral, *i. e.*, on the vaso-motor nerves of the vessels only, apart from the vaso-motor nervous centre; and that the relief caused in angina is in direct relation with the previously increased vascular tension, as suggested by Dr. Lauder Brunton in his first experiment. We had occasion to point out, however, when speaking of that remarkable case in its relation to the theory of the angina paroxysm, that the state of the heart's action corresponding with the period of increased vascular tension on the one hand, and with the relief through amyl-nitrite on the other, was different from what could be attributed to vaso-motor spasm and paralysis alone; and that there remain phenomena of the paroxysm which can be explained, in all probability, only through the innervation of the heart itself. A like difficulty still surrounds the explanation of the physiological and therapeutic action of the nitrite of amyl. Though unquestionably producing some of its well-known effects through vaso-motor paralysis, we are not

quite able to affirm with confidence that its action is purely peripheral, or even that it is quite uniform in all cases of angina. Thus in Dr. Madden's case it seems to have produced, as a primary result, "quickened circulation, tumultuous action of the heart, and quick respiration." This is, in fact, the usual effect of amyl-nitrite on healthy persons, in whom the pulse-rate may be raised in a few seconds from a normal state of about 70 to 120 or 140 pulsations in the minute; the flushing of the face, and the other distinctly vaso-motor effects following the rise in the pulse-rate. In Dr. Brunton's case, on the other hand, the pulse became slower when the spasm was being relieved. In a case published by Dr. Haddon, which, though rather imperfectly reported, appears to have been one of aortic incompetency with angina-like pain, the pulse was jerking, and 80 per minute at the commencement of the inhalation, and after only three drops were inhaled, "the pulse lost its jerking character and became gradually slower," but the face did not become flushed, and the pain was not relieved. In the course of a minute, "the pulse beat so slowly that I thought the heart would stop altogether; while the patient raised himself on his elbow, and with a pale face moved his head about, as if for breath. At the same time he seemed confused, and did not answer questions."[1] Under brandy and free ventilation the pulse recovered its former character and frequency, and the patient fell asleep in half an hour. In another case, which proved on post-mortem examination to be one of aneurism of the first part of the aorta, pressing on the right ventricle and pulmonary artery, and with universal adhesion of the pericardium, besides a degree of compression of the left phrenic nerve by a diseased bronchial gland, the paroxysms of coughing, which were among the most apparently dangerous symptoms in the case, were greatly aggravated on one occasion by the inhalation of five drops of amyl-nitrite, and a critical state of apnœa was induced. It is obvious that neither of these cases was one of typical angina, and it is quite possible that the phenomena may have been only accidentally connected with the inhalation; but Sander has recorded two cases, and Samelsohn one case,[2] in which alarming symptoms of collapse followed closely on the inhalation of amyl-nitrite. In the latter case there was not even a suspicion of internal disease, the inhalation being done experimentally, with a view to test its effects upon spasmodic closure of the eyelids in an anæmic young woman. The usual flushing occurred, but was in an instant "replaced by a deadly pallor; the pulse became thread-like and slow, the skin cold and clammy, respiration difficult, and gasping; consciousness was retained." These symptoms recurred again and again at intervals for an hour, and even up to next day the patient complained of feeling very cold. It is stated that she was menstruating at the time, and that on subsequent occasions she inhaled the nitrite without any such alarming incidents. It is quite possible that the effects of fright, or agitation, or some other accidental disturbing cause, may in these cases have complicated the action of the amyl-nitrite; but still they form a warning, not only that dangerous results may in certain circumstances follow its inhalation, but that the theory which regards its action as purely vaso-motor, and still more that which considers the vaso-motor nervous centres, and the brain and spinal cord generally, as not within the range of its direct influence, must be held in the mean time as subject to reservations to be afterwards ascertained by experience.

Generally speaking, the administration of nitrite of amyl in angina has been found to be free from danger, when used in doses of from two or three up to ten minims on a cloth or handkerchief, abundant access of air being allowed at the same time. The first effects of the remedy in healthy persons are, as stated above, increased frequency of the cardiac pulsations, with a feeling of palpitation, and throbbing of the carotids, followed in the course of thirty to forty seconds after the commencement of the inhalation by flushing of the face, warmth of the head, face, and neck, with perspiration; the latter symptoms being often general. Breathlessness and disposition to cough, giddiness, headache, slight indistinctness of vision, lassitude, and a feeling of intoxication, are among the variable after-effects. The actual thermometric temperature of the body does not appear to be much, if at all, affected; and consciousness is always preserved.[1] When given in angina the effects are similar, with the

[1] Edin. Med. Journal, July, 1870, p. 46.

[2] London Medical Record, March 17, 1875, p. 168; and Aug. 16, 1875, p. 479.

[1] Compare Goodhart, Practitioner, vol. vi. 1871, p. 12; and Talfourd Jones, ibid. vol. viii. 1872, p. 213. Dr. Wood (Amer. Journal of the Med. Sciences, new series, vol. lxi. 1871, p. 422) found that by poisonous doses in animals temperature was lowered "to a degree which is almost unheard of in the history of drugs." He also found that this substance has "the curious chemical property of checking oxidation." It prevents the change of venous into arterial blood, produces gradual paresis, depresses the action of the heart, and yet fails to affect consciousness and sensibility almost to the very last. Some of these results appear to require confirmation.

exception of the discrepancy formerly alluded to as regards the cardiac pulsations. The flushing of the face must be fully developed, in severe attacks of angina, before any relief is to be obtained; but in minor attacks the pain and sense of constriction give way before a very few drops; almost immediately on the first inhalations, or even after merely applying a bottle containing a little of the remedy to one nostril. Three to five drops on a small piece of lint, or on a handkerchief, may be said to be an ordinary, or experimental dose, as a commencement. When the patient has become thoroughly familiar with the effects of the remedy he may, if intelligent and conscientious, be entrusted with a quantity sufficient for ordinary use at his own discretion. One patient mentioned by Dr. Jones[1] had used about thirty ounces in six months; but the large quantity was accounted for by his belief that the remedy when kept in the pocket in a small stoppered bottle, became "flat," and required to be renewed. Dr. Jones believes that he was right in this impression. This patient discarded the lint, and always inhaled directly from the bottle, which he always carried about with him, containing about half a teaspoonful of the remedy. "One night his father found him sound asleep, with his hand hanging over the bed, and the bottle held firmly in its grasp." He declared that "he would not be without 'his bottle of drops' for a hundred pounds." This was a most remarkable case of relief, in what seems to have been aortic regurgitation, in a man of twenty-one years of age. It shows, however, that this remedy, like all others of the same class, is liable to abuse.

The remaining remedies of the angina-paroxysm are probably of small account in comparison with those already mentioned; but it is desirable to add a few words with respect to some of them. Notwithstanding the opinion of Heberden, blood-letting has been recommended, and in some cases, perhaps, successfully practised; the cases being probably those in which evident signs of cardiac venous congestion existed. In Dr. Brunton's case *small* blood-letting, of a few ounces only, appeared to give relief. Dry cupping between the shoulders is a more reasonable, or, at all events, less spoliative method of unloading the heart, and might in some cases co-operate advantageously with the use of warm stimulation of the surface as above recommended. Laennec first suggested the transmission of a magnetic current through the chest; but this suggestion may be said to have had no practical result, and the first apparently effective use of electrical or galvanic currents in angina pectoris is due to Duchenne, of Boulogne,[1] who professes not only to have relieved, but to have cured a typical case of severe angina of five months' duration, in a currier, aged fifty, "of a stout build and sanguine temperament, rather fat, and with a short neck," by treatment for a fortnight only with a strong faradic current passed through the skin of the nipple and upper region of the sternum. The description of the case is extremely striking, but its phenomena being purely subjective, there is not any absolute guarantee for its being more than a severe case of intercostal neuralgia, in which the extremely violent action of the "induction-apparatus graduated to maximum intensity, and working with very rapid intermissions," produced the effect of a strong and sudden counter-irritation. On any other supposition, indeed, the results are almost too wonderful for belief. The first shock produced excruciating pain, so that the patient uttered a loud shriek, and the current had to be arrested. This artificial pain, however, completely and immediately removed the angina pain, as well as the sensations of numbness and formication which accompanied it; and "the patient felt at once in his normal condition." Succeeding paroxysms were similarly arrested, and in a fortnight the patient was able to resume his employment. Another case, communicated by Aran to Duchenne, is especially cited by Trousseau (who records both cases in great detail) as "giving more value to the preceding considerations;" but this will probably not be the judgment of the reader of the preceding pages, when he learns that the subject of Aran's therapeutical experiment was a woman of thirty-two, who had been extremely hysterical, if not cataleptic, from intense grief, and had been for a long time a prey to a multitude of nervous disorders, the result of violently disturbing emotions.[2] Eulenburg has employed the

[1] The Practitioner, vol. viii. p. 219.

[1] De l'électrisation localisée et de son application à la pathologie et à la thérapeutique. 3ieme edit. Paris, 1872, p. 808. See also note Sur l'influence thérapeutique de l'excitation électro-cutanée dans l'angine de poitrine, Bulletin de Thérapeutique, 1853; and compare note below.

[2] It is, perhaps, worthy of remark, that the experience of twelve years after his first acquaintance with the facts of Duchenne's and Aran's cases had not enabled Trousseau to add anything of a more personal kind to his long citation from Duchenne's narrative, first published in 1853. See the 2d edition of Trousseau's "Clinique de l'Hôtel Dieu" (vol. ii. pp. 453–57), published in 1865, not long before his death. Duchenne himself, in the 3d edition of his well-known work (referred to above) published in 1872, and called in the preface "presque un nouveau livre,"

constant current, up to a strength represented by thirty elements of Siemen's battery, applying the positive pole with a large surface for contact to the sternum, and the negative to the lower cervical vertebræ; the successes which he claims, however, are rather equivocal, and it may be inferred from the method of his reasoning that he only employed the remedy in cases regarded as of vaso-motor origin.[1] I am not aware of any case in which angina pectoris of obviously organic origin has been, even temporarily, relieved by any form of electrical or galvanic application; but possibly further trials may still be desirable. Digitalis, aconite, and veratrum, have all proved either useless or injurious.

The *treatment of the inter-paroxysmal state* in angina pectoris depends essentially on the careful application to the individual case of all the practical suggestions arising from a very complete diagnosis, and from a consideration of the causes which have been observed or supposed to be chiefly at work in predisposing to, or in actually bringing on, the paroxysms. Generally speaking, tranquillity, both of body and mind; especially the suspension of all occupations, or even amusements, that tend to overstrain the heart, or hurry the breathing; very moderate daily exercise on level ground, and only to such an extent as is requisite for preserving the bodily tone, or for good digestion; the avoidance of all manner of food tending to flatulence, and the regular, but strictly moderate evacuation of the bowels, either spontaneously or by the mildest laxatives, are measures of hygiene so obviously suggested by simple prudence as hardly to require more than a passing allusion. It is not by any means certainly ascertained whether the subjects of angina ought to use alcoholic stimulants in any measure *habitually*, or to reserve them for the critical period of the attack. I incline to the latter opinion. Venereal excitement is probably in all cases an unfavorable influence. The use of tobacco in great excess has been specially investigated as a cause of angina by M. Beau;[1] but although I have frequently observed palpitation and intermission of the heart's action in smokers, it has not occurred to me to observe true angina pectoris thus produced. It will be obviously right, however, to discountenance any indulgence of this kind which is even doubtful as to its effects upon the heart's action. Beyond these simple measures of precaution, the treatment must vary according to the circumstances observed in each case, and it may even be said that there are cases in which no clear indication exists for any treatment beyond that of the paroxysm. But if it be discovered that gout, or congestion of the liver or lungs, or well-marked dyspeptic symptoms, or renal derangement, has concurred with, or alternated with, the paroxysms, or even that any of these disorders has been a marked feature of the case, without any obvious relation to the angina, it may be found that in undertaking the treatment of these apparently intercurrent disorders the cure or alleviation of the paroxysms may follow in due course. It is said, indeed, by some that gouty angina is peculiarly amenable to treatment, and therefore less formidable in its prognosis than other kinds; and although this is probably only an imperfect statement of the fact that cures of angina-like symptoms are sometimes obtained by remedies in the gouty habit,[2] yet as a practical question of duty there can be no doubt that we are bound to treat the constitutional disease, as the best means known of influencing the local symptoms. It will therefore be expedient to use all possible means for eradicating, or at least diminishing, the gouty predisposition, in cases of angina so characterized, by careful regulation of the diet and the use of anti-arthritic remedies, such as the carbonates of potash and lithia, or even in some cases small doses of colchicum; though it is very doubtful how far a well-marked attack of gout in the foot ought to be checked, either by colchicum or any other disturbing remedy, in those who have had angina and other internal manifestations of the disease. A holiday at Carlsbad, Vichy, or Töplitz, or, according to the fashion of last cen-

gives only one new case, with scanty and unsatisfactory details, in which, moreover, after "partial amelioration" under the method of electro-cutaneous excitation previously described, the patient died suddenly when entering M. Duchenne's consulting-room. He refers, however, to a case of cure by M. Boullet, and to "several cases of cure" communicated to the Academy of Sciences, in February, 1869, by M. Ed. Becquerel. These last I have not been able to discover. M. Becquerel simply reports M. Boullet's case without commentary, and with such brevity and want of essential details as to deprive it of all real clinical value. Evidently there is great inexactitude here, as well as a "plentiful lack" of trustworthy facts.

[1] Med. Times and Gazette, May 7, 1870, p. 490.

[1] De l'influence du tabac à fumer sur la production de l'angine de poitrine.—*Gazette des Hôpitaux*, 1862.

[2] On the other hand, a large proportion of the fatal cases of angina pectoris has been, as already shown, connected with gout, and between these two opposite sides of the question it is not easy to find a secure basis for the alleged relatively favorable prognosis of gouty angina.

tury, at Bath, may help to dispose of the remains of gout when its regular form threatens to pass into irregular manifestations. Fothergill and others have affirmed the cure of angina pectoris in this way.[1] If the urine shows persistently, or even frequently, a tendency to deposit lithic acid crystals, the treatment will, of course, be guided by this indication: and if acid dyspepsia is present, it will be necessary to use remedies at once antacid and tonic. If, on the other hand, the neuralgic element is highly pronounced, more especially if it is hereditary, or has been manifested in the individual patient in other forms, the angina pectoris being presumably a mere form of a more extended constitutional neurosis; we may probably look in such cases for relief to nervine tonics, but especially to iron, strychnine, and arsenic. I have seen in one or two cases very decided good results from the last of these remedies, given in the form of the ordinary Fowler's solution, ♏ v. for a dose, two or three times a day over a considerable period; and I can to this extent support the statements of Dr. Anstie, who in this country has chiefly advocated the use of arsenic in angina pectoris, and who refers to a case published by Philipp,[1] as having first strongly directed his attention to the subject. Anstie begins with three minims, and increases the dose gradually, if well tolerated, up to eight or ten minims three times a day; he has found, however, that some neurotic patients cannot tolerate arsenic from the irritability of their alimentary canal, and in such cases it must be discontinued, or perhaps some other form of administration might be devised. Anstie gives several striking cases, in one of which, at least, there had been a few slight attacks of gout, and a few small calculi; another was that of a woman, aged forty-six, who was still menstruating, though irregularly, and who certainly seems to have had extremely severe symptoms of the order of angina; she was cured by a six months' course of arsenic in doses gradually mounting to 21 minims daily; after eight weeks the patient abandoned the remedy, supposing herself cured, but had to recur to it from experiencing a renewal of her sufferings, which again yielded to a precisely similar treatment.[2] Arsenic is specially adapted for anæmic cases, and often exercises a favorable influence over the function of hæmatosis; but in cases where anæmia is well-marked it may be combined with iron, or the latter may be given with strychnine (ten minims of the sesquichloride tincture with $\frac{1}{10}$ gr. strychnine three times a day). Phosphorus has lately been recommended by Dr. Broadbent, in doses of from $\frac{1}{60}$ to $\frac{1}{40}$ gr. twice daily, but has not as yet been adequately tested. Zinc, silver, and most of the older remedies of this class, have been, on the whole, found wanting in true angina pectoris, though sometimes useful in pseudo-angina. A remarkable experience was that of Bretonneau (detailed by Trousseau),[3] who, following out a very crude chemical theory of the calculous origin of angina, professed nevertheless to have stumbled upon the practical result of treating cases of angina successfully by large doses of bicarbonate of soda, combined in certain cases with belladonna. The directions given are very complicated, but the essential part of the treatment seems to be as follows: The alkaline treatment is begun with two

[1] The case here specially referred to was mentioned by Fothergill in 1773, incidentally, in a paper on angina pectoris, as "the first case apparently of this nature that occurred to me, above twenty years ago." He adds, "the person is now, or lately was, living, and in good health . . . He was at that time about thirty years of age, and the youngest subject I have ever seen affected with this disorder." The symptoms are fairly described, considering the early date, and long interval between their occurrence and the publication, but can scarcely be looked upon as thoroughly characteristic. He "went to Bath several successive seasons, and acquired his usual health. This is the only instance that has occurred to me," writes Fothergill, "of a perfect recovery from this obscure, and too often fatal malady." We have seen that Heberden's experience also yielded only one case of apparently perfect recovery. In one other case, with distinct gouty complications, Fothergill prescribed Bath waters, with good results as regards the gout, but with no favorable effect on the angina. In another case the Buxton water appeared to be of temporary service. Fothergill seems to have been strongly impressed with the necessity of reducing exuberant fatty deposition in angina pectoris, and for this purpose recommended vegetable diet; though he did not anticipate in any respect later observations as to the connection of sudden death with fatty degeneration of the fibre of the heart itself. See Medical Observations and Inquiries, vol. v., 1776, p. 223, "Case of an Angina Pectoris, with Remarks;" and p. 252 of "Farther Account of the Angina Pectoris, by J. Fothergill, M.D., F.R.S."

[1] Berliner Klin. Wochenschrift, 1865. See, however, Cahen (*ut supra*) Archives Générales, 1863; and a much older case by Alexander, of Halifax, 1790 (History of a case of Angina Pectoris cured by the Solutio arsenici), Medical Commentaries, vol. xv. p. 373. This last case has, apparently, had very little effect on English practice, but is referred to by Desgranges, Trousseau, and other continental authorities.

[2] Anstie, op. cit. Compare pp. 78, 182–84, 226–27.

[3] Op. cit., Eng. transl., vol. i. p. 610.

scruples of the bicarbonate of soda, daily, in divided doses, rising gradually to eight or ten scruples, increasing and diminishing the dose alternately over intervals of ten days, and then suspending the treatment for fifteen or twenty days together; these various processes are repeated up to the end of a year or more, after which a pause of several months is allowed. At all stages of this lengthened treatment, belladonna may be given in gradually increasing doses, up to the point of relief to the spasms, or until symptoms of incipient poisoning occur, viz., "unpleasant dryness of the mouth, marked disturbance of vision, accompanied by a very striking dilatation of the pupils." Notwithstanding the great therapeutic reputation of Bretonneau, I have not been able to learn that any one else in France has personally succeeded with this treatment, and even M. Trousseau, his most distinguished pupil and follower, does not profess to do more than record his master's opinions. The facts as stated may therefore probably remain among the curiosities of medical experience; but as they have been generally referred to, it is necessary to make brief allusion to them here.

In cases of angina connected with positive organic disease, the treatment must follow the lines of that of the cardiac or vascular lesion which is discovered to be the cause of the symptoms. It is very doubtful, however, whether in cases of fatty heart, or of calcareous and other degenerations of the vessels, there is any positive special treatment which can be recommended with confidence. In cases of aneurism, on the other hand, the iodide of potassium, in large doses of 20 to 30 grains and upwards, will be found of great value in checking all the painful sensations, and even, in some cases, arresting or suspending the disease; and the bromide of potassium, or of ammonium, may be given in some cases along with the iodide, as a palliative. A late American writer[1] specially commends the bromide of ammonium, and gives two cases in which, in doses of 15 to 20 grains, it seems to have averted the paroxysms.

Note on the Literature of Angina Pectoris.—The leading authorities have been mostly referred to in the preceding pages, and will be found quoted much more numerously and in chronological order in the two great French dictionaries mentioned below,[2] under the head "Angine de poitrine." I have not in the text of this article referred to the letter of M. Rougnon to M. Lorry, in 1768,[1] which has been set forth by M. Jaccoud and others as constituting a claim on the part of France to priority, or at least to a simultaneous discovery of angina pectoris with that of Heberden. From the accounts given of this letter, as I have been

[1] Dr. Rufus K. Hinton, Philadelphia Medical and Surgical Reporter, March 6, 1875.

[2] Nouveau Dictionnaire De Médecine et de Chirurgie pratiques, tome 2ieme. 1865, p. 509 (Art. by Jaccoud). Dictionnaire Encyclopédique des Sciences Médicales, tome 5ieme. 1866, p. 65 (Art. by Parrot). Consult also the Bibliography in Forbes, Cyclopædia of Practical Medicine, vol. iv.; the essay of Wichmann, Ueber angina pectoris und polypus cordis (Ideen zur Diagnostic, vol. ii. 1801); Brera (Della stenocardia; saggio patologicoclinico, Modena, 1810); Desportes (Traité de l'angine de poitrine, Paris, 1811); Zechinelli (Sull' angina del petto e sulle morte repentine, Padova, 1814); Jurine (Mémoire sur l'angine de poitrine, Paris et Genève, 1815); Lartigue (De l'angine de poitrine, Paris, 1846); Lussana (Intorno all' angina pectoris, Gazetta Medica Lombarda, 1858–59), besides the great and well-known works of Senac, Corvisart, Laennec, Testa, Kreysig, Bouillaud, Hope, Latham, Stokes, Walshe, Friedreich (in Virchow's Handbuch), Bamberger, and others on Diseases of the Heart, the most recent being that of Dr. Hayden, Dublin (1875), which reached me after the first part of this article was written, but to which I have been indebted for several suggestions in the latter part of it, and some valuable references. The works of Romberg and Eulenburg, on Diseases of the Nerves, should also be consulted; and the articles in all the older systematic treatises and dictionaries, whether British or continental. With the exception of Rougnon, all the authorities quoted in any of these sources up to 1778 are English. In that year Elsner published at Königsberg a monograph, entitled, "Abhandlung über die Brustbraune," which was followed in 1782 by Grüner (Spicilegium ad anginæ pectoris . . .), and Schäffer (Dissertatio de angina pectoris, 1787). Several articles or treatises soon followed in Germany, Denmark, and Holland; but I do not know if angina pectoris is even mentioned by name in French medical literature prior to the paper of Baumés in 1808, "Récherches sur cette maladie à laquelle on a donné les noms d'angine de poitrine et de syncope angineuse" (Annales de la Société de Médecine pratique de Montpellier, 1808). The first Italian monograph was that of Brera in 1810, cited above. After this, the literature becomes much more copious; but the well-known article of Dr. Forbes, in the first volume of the Cyclopædia of Practical Medicine, 1833, will always remain, especially for the English reader, the chief source of exact information down to a comparatively recent date.

[1] Lettre à M. Lorry touchant les causes de la mort de M. Charles, ancien capitaine de cavalerie, arrivée à Besançon le 23 février, 1768 (Besançon, 1768, 8vo.) Rougnon described the paroxysms of pain, and ascribed these and the sudden death to ossification of the costal cartilages. He did not give any name to the disease, or indicate otherwise its pathological and clinical relations.

able to read them, it is manifest that M. Rougnon is in no just sense of the words a rival or competitor of Heberden; he is, however, probably entitled to the credit of having independently described a single case of sudden death, with symptoms more or less resembling Heberden's angina, as we have seen that Morgagni had done a century before. Without in the least degree desiring to detract from what is due on this account to Rougnon, it must be here pointed out that Heberden's position is entirely different. Instead of describing only one case, and reasoning inaccurately as to its pathology, Heberden founded a minute and exact clinical description upon the observation of not less than twenty cases, of which, he informs us, six had been known to him as having perished suddenly. Heberden's account of the "Disorder of the Breast," accordingly, soon became known to medical men in various countries as an accurate and comprehensive sketch of a new disease, while Rougnon's case passed into oblivion, without even in France exciting the attention that was perhaps due to it as an isolated observation. The claim advanced on behalf of Rougnon is evidently an after-thought, and cannot now be admitted. If sudden death from angina is to be recognized in any sense at all as a discovery on the strength of an individual case, the credit undoubtedly belongs to Morgagni rather than to Rougnon. It is right, however, to add that I make these remarks without having personally read Rougnon's letter, which I have inquired for in vain in the medical libraries of this country.

DISEASES OF THE VALVES OF THE HEART.

By C. Hilton Fagge, M.D., F.R.C.P.

The literature of diseases of the valves of the heart, as of all other thoracic diseases, is necessarily divided into two periods; that before, and that after, the discovery of auscultation. The earlier period, however, contains very few observations. Burns[1] quotes two cases of aortic obstruction, briefly related by Riverius and Willis respectively, towards the end of the seventeenth century. Dr. Gee[2] points out that Vieussens in 1715 recorded a case of disease of the aortic valves, in which the pulse was "*fort vîte, dur, inégal, et si fort que l'artère de l'un et de l'autre bras frappait le bout de mes doigts autant que l'auroit fait un corde fort tendue et violemment ébranlée.*"

Friedreich[3] is therefore not quite accurate in heading his list of papers and works on affections of the endocardium with Meckel's essay in the Mém. de l'Acad. Roy. des Sciences, published in Berlin in 1756. But it is in the second half of the eighteenth century that we find the first detailed observations of diseased cardiac valves. Among the most striking of these is one recorded by John Hunter, in his "Treatise on the Blood, &c.," which originally appeared in 1794. It is that of a Mr. Bulstrope,[1] who had "almost throughout his life had an irregular pulse and upon the least increase of exercise a palpitation at his heart, which was often so strong as to be heard by those who were near him . . . He of late years (about the age of thirty), took to violent exercise such as hunting; and often in the chase he would be taken ill with palpitations and almost a total suffocation. Some of these fits continued several days: at such times he became black in the face. Sometimes an universal yellowness took place; and then he could not lie down in his bed, but was obliged to sit up for breath. He consulted Dr. Heberden and Sir George Baker; the palpitation I suppose they thought either arose from spasm or was nervous, for they ordered cordials. I was sent for on the same day to give a name to the disease. My opinion was that there was something very wrong

[1] "Observations on some of the most frequent and important Diseases of the Heart." Edinburgh, 1809, pp. 175, 176.

[2] "Auscultation and Percussion," 1870, p. 260.

[3] "Krankheiten des Herzens," Virchow's Handbuch der speciellen Pathologie und Therapie, 1867, p. 198.

[1] The preparation from this case is still in the Hunterian Museum of the Royal College of Surgeons, which also contains several other specimens of diseased cardiac valves, preserved by Hunter himself. The passages cited in the text are from the "Catalogue of Path. Specimens," vol. iii. p. 197.

about the heart, that the blood did not flow freely through the lungs. . . . That the means to be practised were rest, bleed gently, eat moderately, keep the body open and the mind easy. . . . Eight ounces of blood were taken from him that day, which relieved him. . . . At last he became yellow, and his legs began to swell with water . . . and he died. The heart was very large . . . the valves of the aorta shrivelled up, thicker and harder than common. The diseased structure of the valves accounts for every one of what may be called his original symptoms; the blood must have flowed back into the cavity of the ventricle again at every systole of the artery. . . . We can easily trace the effects of this retrograde motion, which would only be a stagnation of the blood beyond the left ventricle, first in the left auricle, then the pulmonary veins, then the pulmonary arteries, next the right auricle, and in all the veins of the body; producing that darkness in the face, &c." Even earlier than this, Senac, in his Treatise on the Heart (the second edition of which appeared in 1783) had related a case in which the auriculo-ventricular valves were ossified, and remarked that the pulse was necessarily small, because the blood did not all pass into the aorta, but some of it flowed back into the auricle. Soon after the commencement of the present century, three works on diseases of the heart were published, in which valvular affections are treated of with considerable detail: that of Corvisart,[1] in 1806; that of Allan Burns,[2] in 1809; and that of Kreysig,[3] in 1815.

The study of these works is of considerable interest. Corvisart gives an admirable account of the anatomical appearances exhibited by diseased valves, which he distinguishes as undergoing calcareous or osseous induration, or as presenting excrescences (vegetations).[4] It is remarkable, however, that he seems to have had no conception of these diseases as causing regurgitation, or imperfect closure of the valves.[1] The tendency of valvular affections to cause dilatation of the heart (or, as he terms it, "aneurism of the heart") was well known to Corvisart. By Burns and Kreysig considerable advances were made. Both these writers recognize valvular lesions as producing two distinct effects, "obstruction" and "regurgitation," and trace many of the consequences of the latter condition. Kreysig lays special stress on inflammation of the endocardium as causing the lesions in question. Burns may even be said to hint at the occurrence of cardiac murmurs, for he speaks of regurgitation from the ventricle into the auricle as producing not only a jarring sensation but also "a hissing noise, as of several currents meeting. In all probability" (he goes on to say) "it is something of this kind which is described as audible palpitation in some diseases of the heart."

The history of the subsequent literature of valvular affections is involved in that of the auscultatory phenomena which they produce; and hereafter, when these are under consideration, I must endeavor to deal with the most important parts of it.

DESCRIPTION AND ANATOMY.—The pathological changes met with in the valves of the heart are naturally divisible into two groups; 1. Those which are acute: 2. Those which are chronic.

1. The acute affections are of an inflammatory nature, and come under the general head of endocarditis. Indeed, it has long been known that the membrane forming the valves is more liable to inflammation than any other part of the endocardium. And, as we shall presently see, recent observations have shown that this is true in a more absolute sense than had been imagined: and that when inflammation of the linings of the heart's

[1] "Essai sur les Maladies et les lésions organiques du Cœur et des Gros Vaisseaux." So far as diseases of the heart are concerned, however, this writer (who will always be remembered as having popularized Avenbrugger's discovery of percussion) is better known by his second edition, which appeared in 1811, and was translated into English by Mr. Hebb in 1813.

[2] Op. cit.

[3] "Die Krankheiten des Herzens." Berlin.

[4] The word "vegetation" is now commonly used in this country, but it may be interesting to note that Mr. Hebb, the translator of Corvisart, never employs it as an English term, but always incloses it between brackets, and uses "wart" or "excrescence" as its equivalent.

[1] This must be borne in mind in estimating the claims of different writers to priority in regard to the discovery of the presystolic thrill and bruit. Corvisart first mentioned the sign afterwards known as *frémissement cataire*. He speaks of it as "a particular rustling, difficult to describe, perceptible to the hand when it is placed over the præcordial region, and which doubtless proceeds from the difficulty which the blood experiences in passing through an aperture no longer proportionate to the amount of fluid to which it has to give vent." It might thus appear that Corvisart associated thrill with mitral stenosis. But it must be recollected that he summed up the effects of *all* valvular diseases in the contractions of the corresponding orifices which he supposed them to cause. He had no conception that the presence of thrill was of any value as regards a *differential diagnosis* of valvular affections.

cavities is met with, it has almost always been set up by a similar affection of one of the valves.

The microscopical anatomy of inflammation of the valves may, therefore, be dismissed in a very few words, as being the same as that of endocarditis in general. The minute bloodvessels, which recent observers have shown to exist in the valves, become gorged with blood, and the cells of the external tunic of these vessels undergo proliferation.[1] But this change is quite subordinate to that which occurs in the proper substance of the valve itself: in the connective tissue of which young cells are formed in large numbers, while the intercellular material becomes softened. The tissue is thus much swollen; and as the change in question is not at first general, but is confined to certain spots, the result is the formation of a number of small granulations, projecting from the surface of the valve. These granulations are very commonly limited, in the first instance, to a particular region in each valve, namely, that which lies immediately behind the line of closure. Thus, in a cuspid or auriculoventricular valve, the earliest swelling is found on the auricular surface, and a little above the free edge; in the case of the semilunar valves, it is on the ventricular surface, and along the delicate curved line, limiting the apposition of the valves, that stretches on either side of the corpus Arantii. In these positions the granulations are often pretty uniformly arranged, like a row of minute beads. The remembrance of their seat may be facilitated by imagining the valves to have been coated on their apposed surfaces with a layer of some soft substance (such as butter), which during closure of the valves would be forced into precisely the positions that the granulations occupy. And according to the view formerly entertained, that the granulations were formed of an exudation of plastic lymph, it was easy (with Sir Thomas Watson[2]) to refer their arrangement to this cause. But, as we have seen, the microscope shows that they are swellings of the tissue of the valve itself, and this explanation is, therefore, untenable. The granulations vary in appearance; being sometimes colorless, sometimes red (the latter perhaps from imbibition). Their consistence is different in different cases: sometimes they are so soft as to be detached from the valve by the slightest touch; sometimes they are so hard as to resist all attempts to remove them. They are much more often seen on the valves of the left, than on those of the right side of the heart: the former being in fact much more subject to endocarditis than the latter.

When acute endocarditis occurs as part of a rapidly fatal general disease, the presence of such a line of minute granulations is generally the only sign that inflammation of the valves had existed: and if (as is often the case) the affection be confined to the auricular surface of the edge of the mitral valve, it may be entirely overlooked, unless attention be specially directed to this spot.

But in certain cases, the changes are far more considerable. The granulations are very much larger, and become confluent, so as to form masses, which fairly deserve the name of vegetations. These bodies, projecting into the stream of the blood, necessarily offer a favorable surface for the reception of coagula; and thus colorless fibrin of firm consistence is deposited upon them, often in large quantity. This so closely resembles in appearance the swollen tissue of the valve itself, that it may be impossible to say where the one begins and the other ends. Indeed (as has already been mentioned) the older theorists (who thought that the valves, which were then supposed to be non-vascular, were incapable of inflammation) believed even the smallest granulations to have been thus deposited from the blood. When this opinion was shown to be incorrect, its opposite prevailed; and it is only after repeated and prolonged discussions that pathologists have come to the conclusion that the larger masses have the double origin just attributed to them. These, again, are often found to be still further increased in size by the deposition of dark red clots upon them, while the patient is in the act of dying, or during the post-mortem coagulation of the blood.

These vegetations necessarily float to and fro with the valve to which they are attached, and thus they are almost always brought into contact with the surface of another valve opposed to them, or with some part of the endocardial lining of the heart's chambers. For it must be added that they are not always sessile, but are often suspended by a pedicle of some length, allowing them to swing backwards and forwards through a considerable range of movement. The result is that they frequently set up inflammation in the parts against which they rub. This fact was, I believe, first pointed out by Dr. Moxon, who, in 1868 and 1869, exhibited to the Pathological Society several illustrative specimens.[1] My own obser-

[1] Rindfleisch, "Lehrbuch der pathologischen Gewebelehre." Leipzig, 1871, p. 205.

[2] "Princ. and Pract. of Physic," 4th edition, vol. ii. p. 294.

[1] Path. Trans. xix. p. 148, xx. p. 156. It will be shown further on that Dr. Hodgkin long ago described the effects of friction in the case of valves affected with *chronic* disease.

vations have convinced me that his statements are perfectly correct. A vegetation hanging from an aortic valve is often thrown upon the wall of the aorta during the ventricular systole, and sets up there a little ulcer, penetrating into the middle coat, or even down to the adventitia: during the diastole the same vegetation is carried downwards, and touches the anterior surface of the mitral valve, or the endocardial lining of the ventricle, and the spot touched is found, after death, to be precisely indicated by the presence of a little fresh mass of vegetations. The opposed surfaces of the aortic valves are often seen to be coated with vegetations, in such a way as to suggest that the one was affected secondarily to the other, although it may not be possible to say which was primarily diseased. Or, again, a button-like mass of vegetations projecting from one aortic valve has been seen to bore a hole right through the substance of the valve opposed to it. A cluster of vegetations growing from the auricular surface of the mitral valve often sets up inflammation in the base of the opposed segment of the valve, where the vegetations meet it during closure of the valve: and from this spot the inflammation spreads into the auricle.[1] Dr. Moxon has even expressed the opinion that vegetations attached to the lining membrane of the heart's cavities are scarcely met with, except as the result of friction, in the way just described, the valves being first diseased. And I would add my belief that there are few cases of acute inflammation of the valves, in which secondary effects of this kind may not be traced.

The rapid and extensive movements performed by these floating vegetations have probably much to do with the frequency with which portions of them become detached and carried with the blood-stream to distant parts, producing effects which we shall hereafter have to consider. But it must be added that they are also very liable to undergo a finely granular metamorphosis (according to Rindfleisch, not fatty), which renders them still softer than they originally were. In this softening process the inflamed tissue of the valve itself takes part; so that large portions of it often become disintegrated, and an ulcer is produced, which may destroy the whole thickness of the valve and perforate it. Such ulceration, for instance, may separate one or both edges of an aortic valve from attachment to the arterial wall; or eat away a large part of its substance. In the mitral valve, it is not uncommon for a hole to be pierced right through its substance. In this case, however, the edges of the aperture are always thick and raised, and covered with large vegetations; and these generally meet across it, so that there is no reason to suppose that by such a perforation the physiological action of the valve is in any way impaired. Indeed, these vegetations are often so massive, that the existence of a perforation, and even of an ulcer, may escape notice unless it be specially looked for. The records of post-mortem examinations at Guy's Hospital contain only one notice of such a perforation in the course of six years; but I have little doubt that it had really occurred more often. It must be added that the ulceration not rarely extends from the valves themselves into the adjacent parts of the muscular substance of the ventricle.

The same process of softening and ulceration, occurring in the chordæ tendineæ of the cuspid valves, leads to their rupture. This is by no means infrequent; in six years I find it recorded sixteen times in the reports of post-mortem examinations just referred to. Clinically it would appear to be of far greater importance than perforation of the valve: I imagine that it must invariably render the valve incompetent. The changes which precede rupture of the chordæ would appear to consist in a swelling and thickening of their substance. Generally they give way in about the middle of their length; but sometimes they are torn away from the musculus papillaris, which may then exhibit no trace of their original insertion into it. The left and the right chordæ of the mitral valve appear to be equally liable to rupture. Sometimes the laceration is confined to a single chorda; sometimes it affects nearly all those that arise from one musculus papillaris. The ruptured chordæ float to and fro with the stream of blood, which necessarily regurgitates freely into the auricle at each systole. Once I saw such a loose end tied neatly into a knot, which took some time to undo. In another instance three or four broken chordæ were twisted up spirally into a body resembling an uvula, being matted together by a deposit of fibrin. In a third case, recorded by Dr. Moxon, the free extremities of two such chordæ seemed to have become adherent to the surface of the mitral valve above, forming loops, beneath which a probe could be passed. Large masses of fibrin are whipped out from the blood, and deposited on the sides and extremities of ruptured chordæ, and often unite them together, so that it is impossible to say how many of them may have been torn through, until the superjacent mass of coagulum is removed.

[1] These statements are derived from the detailed reports of the post-mortem examinations at Guy's Hospital during the last few years, most of which were made by Dr. Moxon, but some by myself.

Another effect of this process of ulceration, especially in the mitral valve, is the formation of a so-called "aneurism of the valve." An ulcer having formed on its ventricular surface, the base of this yields before the pressure of the blood, and a pouch is formed, projecting from the auricular face of the valve. Such an aneurism is generally very small: I lately saw one which was of about the size of a percussion cap; it had on its summit a mass of small vegetations. This form of aneurism must be distinguished from that described by Dr. Thurnam,[1] which arises in the gradual yielding of all the coats of the valve, and which may attain a much larger size.

2. In *chronic* diseases of the valves the appearances vary greatly, not only in individual cases, but also according as one or another valve is affected. But they may generally be summed up as dependent on the growth of a firm connective tissue, which thickens the substance of the valve, and by its contraction leads to great alteration in its form, and more or less seriously impairs its functions. Calcareous matter also is often deposited.

This "*sclerotic*" change—if we may adopt the term sclerotic as equivalent to the *chronische sclerosirende Endocarditis* of German authors—may either be primarily chronic, or arise out of an acute inflammation of the valve. In the latter case, vegetations are sometimes found, showing that endocarditis had once occurred; and these may even be calcified. But they

[Fig. 97.

Fibroid thickening of Mitral Valve.]

are not necessarily present. In several cases of valvular disease, that had, in each instance, doubtless arisen in attacks of acute rheumatism which the patient had had some years previously, I have, on post-mortem examination, found that the mitral valve exhibited no trace of vegetations: it was simply thickened, with its chordæ; and its orifice was narrowed. Here the affection had been of acute origin: but the appearances were undistinguishable from those which are met with in disease that has from the first been chronic. The difficulty of determining the way in which valvular changes arise is further increased by the fact that thickened valves are very liable indeed to the supervention of an acute process, identical with that already described as belonging to acute endocarditis. It would appear that the elements of the morbid tissue in chronically diseased valves are apt to undergo a fatty change, analogous to that which gives rise to atheroma in chronic arteritis. The result is that the structure of the valves becomes softened and gives way, and thus that a process of ulceration is set up, precisely as in acute endocarditis. A very large proportion of the cases in which the autopsy shows the chordæ tendineæ of the mitral valve to have been freshly ruptured are cases of long-standing valvular disease, in which inflammation has thus supervened. Another common result is that masses of calcareous deposit, evidently of old formation, are found lying loose in the floor of recent ulcers.

a. In the cuspid valves the effect of chronic disease is generally to produce a stenosis or narrowing of the aperture. The wall of the valve, especially towards its free edge, becomes greatly thickened, and its segments cohere together. The morbid tissue is exceedingly dense and hard, so that by the older writers such valves were described as cartilaginous: it often contains masses of calcareous deposit, and these sometimes attain a very considerable size. The chordæ tendineæ undergo a similar change and coalesce, so that sometimes each musculus papillaris gives origin only to a single massive column, which may be more or less fluted, or pierced with one or two slits, indicating the lines of separation between the chordæ of which it was made up; at the same time the chordæ generally become much shortened, so that the edge of the valve is drawn down; and thus with its small aperture it has very much the appearance of a funnel, projecting far into the cavity of the ventricle. Dr. Douglas Powell,[1] has endeavored to distinguish two forms of stenosis of the mitral orifice, in one of which the valve presents this funnel shape, while in the other it is stretched horizontally, like a diaphragm, between the auricle and the ventricle. But it appears to me that no such distinction can be fairly drawn, and that in all cases a narrowed mitral valve tends more or less to assume the form of a funnel, although this is no doubt much more marked in some instances than in others. The orifice at the bottom of the funnel is sometimes circular: but in the case of the mitral valve it more often resembles a slit, of which the

[1] Med.-Chir. Trans., ser. ii. vol. iii. p. 250.

[1] Med. Times and Gaz., 1871, vol. i. p. 395.

axis corresponds with the line uniting the meeting angles of the original segments of the valve. The latter variety has long been known as the button-hole mitral. In either form the aperture may be so contracted as hardly to admit the tip of the little finger; and cases are often met with in which only two fingers can be introduced, instead of the three which can be passed through the healthy valve.

But the effect of chronic disease of the mitral valve is not always to produce stenosis. It may be the very reverse. It is said that sometimes one of the flaps of the valve becomes adherent to the ventricular wall, and so is rendered incapable of meeting its fellow. But this is a very rare occurrence; indeed I doubt whether it is ever met with. At any rate I have not been able to find a single instance of it in the recent records of post-mortem examinations at Guy's Hospital. These records, however, contain one case in which Dr. Moxon found the edge of the anterior curtain of the valve turned up on its ventricular surface, and adherent there, so as to form a ridge on this surface,[1]—a change by which the depth of the curtain must of course have been diminished. It might be thought that the same process of contraction which ordinarily causes stenosis of a thickened valve might (if acting in a direction at right angles to that in which it usually acts) draw up and shorten the valve without narrowing its orifice. Writers have in fact described such an appearance. But it is one which I have never myself seen, nor have I met with any recorded instance of it: and I am not sure that it ever occurs. Not uncommonly, however, some of the chordæ tendineæ become elongated, and do not properly tether the membranous part of the valve, which therefore becomes inverted into the auricle during the ventricular systole; and this result is often favored by the conversion of the corresponding muscularis papillaris into a dense fibrous tissue. Sometimes those chordæ which are inserted nearest the centre of the valve alone undergo this process of lengthening, and this part of the curtain is then found after death to be bent on itself and flaccid, having evidently been accustomed to yield before the pressure of the blood. Sometimes, again, the chordæ become shortened by disease, instead of being elongated, and thus tether the valve too closely, and prevent the apposition of its segments.

I believe that the preceding paragraph includes descriptions of all the chronic changes in the cuspid valves, by which regurgitation is produced, without obstruction to the onward current of blood. Each of these, however, is of infrequent occurrence. In six years, during which period sixty-seven cases of mitral stenosis presented themselves in the post-mortem threatre of Guy's Hospital, I find only twelve recorded instances of what I may term pure regurgitant disease of the same valve; in six of which the edge of the valve is stated to have been inverted into the auricle, in the manner above described.

The fact just stated will doubtless surprise many readers, who are aware of the frequency with which mitral regurgitant disease is clinically spoken of. The question must be discussed in detail further on; but it may be well here to state that, in a large proportion of cases, a reflux of blood into the auricle during the ventricular systole is probably independent of disease in the valve itself, and due to changes in the walls and cavity of the ventricle, destroying the due proportion between the auriculo-ventricular orifice and the valve by which it should be closed. It must be added that many cases are placed after death under the head of mitral stenosis, which had before been regarded as examples of regurgitant disease. For when moderate obstruction and regurgitation coexist, the latter is often clinically more noticeable, and (as I believe) is often alone discoverable before death; while at the autopsy the appearance of the valve may be exactly similar to that which is found in another case, in which during life obstruction had been the main feature.

β. In the semilunar valves the morbid appearances resulting from chronic disease vary much more than in the cuspid valves. First may be mentioned the adhesion of the valves together. This begins at the point where the corners of adjacent valves are inserted into the aortic wall, and gradually creeps along their free edges, uniting them together. All three valves may thus be fused into a single mass, projecting into the arterial channel in the form of an inverted funnel, with a central aperture, which is often of very irregular form, and may be extremely small, being, in most cases, further narrowed by the presence of rough calcareous nodules of greater or less size, deposited in the substance of the altered valves. While this process is going on, the natural attachment of the corners of the valves to the aorta often gradually becomes obliterated; two of the pouches, or even all three of them, become thrown into one; and three slight projections in the floor of the resulting funnel-shaped mass are often

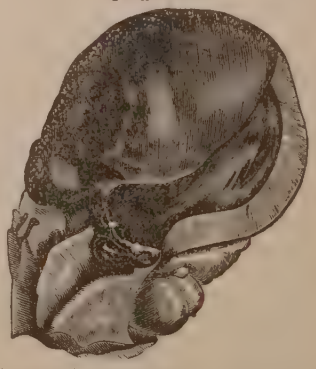
[1] This condition is analogous to one which is commonly seen in cases of perihepatitis, where the anterior thin edge of the liver is neatly folded over on to the convex surface, and bound down beneath the thickened capsule, the apparent rounded margin of the liver being thus really derived from the under surface of the organ.

all that is left to indicate the original lines of separation between the different valves.

In other cases the effect of chronic disease of the semilunar valves is, that they become puckered and shrivelled. Instead of forming pouches, they often rather resemble flat, narrow shelves, projecting a little way from the wall of the artery, the mouth of which they are quite unable to close. The corpus Arantii, with the thin curved borders on either side of it, disappears entirely; and all that is left is a thick, shapeless body, often with its rounded edge retroverted, and perhaps torn away from its aortic attachment on one side, so as to hang down like the lip of a jug, or a dog's ear. Or, again, the valve may contract, and its free border thus become tightly drawn across between its points of attachment, so that the open pouch is converted into a deep pocket with a narrow entrance, into which the tip of the finger cannot be made to enter. This result, however, is not always due to changes in the valves themselves. Sometimes it depends on chronic disease in the coats of the base of the aorta, attended with yielding and dilatation of its walls, by which the valves, although healthy, are unduly stretched. So considerable may this yielding be, that in one case recorded in the reports of post-mortem examinations at Guy's Hospital, the corners of adjacent valves had become distant from one another a quarter of an inch at their points of attachment.

When a valve, thickened, or retroverted by chronic disease, comes into contact with any part of the lining of one of the heart's cavities during the movements of that organ, further morbid changes are produced by the friction, as is the case in acute affections. There is, however, this difference, that the endocardium does not become ulcerated or covered with vegetations, but is thickened, opaque, and slightly roughened. This was pointed out by Dr. Hodgkin as far back as the year 1829.[1] In the case of Dr. Cox, one of aortic disease, in which a valve was stretched to upwards of an inch in length, "the coats of the aorta for about an inch and a half above the retroverted and distended valve, and against which it must have been carried during the systole of the heart, were considerably thickened, and presented an uneven surface. On the inner surface of the heart there were some irregular spots of opacity at the part where the diseased valve would have struck during the diastole." Dr. Hodgkin adds that "the partial thickening on the internal surface of the heart and vessels, in consequence of some unusual contact, is a morbid appearance, which has not been particularly pointed out by pathological anatomists, yet it does not appear to be a rare occurrence." Except Dr. Moxon, I do not find that any writer has since alluded to the appearance in question. But in another respect our knowledge has advanced greatly since the publication of Dr. Hodgkin's paper; for we find him relying on these effects of contact as proving that the blood had been subjected to two motions—the one progressive, and the other retrograde—a fact with which, of course, every one is now familiar. According to Dr. Peacock and Dr. Bristowe,[1] it is not uncommon in cases of disease of the aortic orifice for the endocardium below the valves to present a fibroid thickening in the form of bands or reticulations, due probably to the impact of the regurgitant stream of blood on that part. Of this I have lately seen a striking instance. The same thing may also occur in the auricle, as the result of mitral regurgitation.

[Fig. 98.



Calcareous Degeneration of Aortic Valves.]

It will be observed that in the semilunar, as in the cuspid, valves the effects of chronic disease are twofold. It may either cause obstruction to the onward flow of blood, or give rise to regurgitation and to a backward current. All writers, in fact, insist on this distinction, while admitting that both effects often exist together. Dr. Moxon, therefore, rather surprised me a short time ago by stating that in his experience the occurrence of aortic obstruction, apart from regurgitation, has been extremely rare. I at first supposed that he was referring to fatal cases only, among which pure aortic stenosis would naturally be infrequent, since this is generally said to affect the prospect of life less than any other form of valvular affection, and since, moreover, it is very apt to become complicated sooner or later by the development of regurgitation.

[1] "On Retroversion of the Valves of the Aorta," Lond. Med. Gaz. vol. iii. p. 439.

[1] Path. Trans. xxi. p. 105.

Thus, in looking through the records of post-mortem examinations for the last six years—during which time there have occurred sixty-eight fatal cases of aortic regurgitant disease—I find only two, or perhaps three, of pure aortic stenosis; and in at least one of them the patient's death was due not to this affection, but to coexisting mitral stenosis. But I afterwards ascertained that Dr. Moxon believed aortic obstruction, independent of regurgitation, to be rare clinically, as well as in the dead-house; and this opinion certainly appears to be confirmed by the fact that, during part of the period in which I acted as Medical Registrar at Guy's Hospital (within which period seventy-one cases of aortic regurgitation came under observation) I find only two recorded instances of pure stenosis of the orifice in question; and in one of these regurgitation became developed before the patient's death.

The changes to which the aortic valves are liable are not all included in the thickenings, and adhesions, and puckerings that have hitherto been described. In some cases the tissue of the valves undergoes atrophy; and they become so thin, that it is difficult to believe that they were capable of sustaining a column of blood. A striking example of this came lately under my notice. A young man, æt. thirty-two, had long suffered from asthma, and becoming anasarcous was admitted into Guy's Hospital under the care of Dr. Wilks. After death the aortic valves were found to be most remarkably thinned; they had no more substance than the most delicate tissue-paper, and no corpora Arantii could be felt in them. One of them was slightly fenestrated near its margin. They were very small, and, when left to themselves, fell back into the Sinuses of Valsalva. The pulmonary valves presented the same change in a minor degree; and the mitral valve was likewise unusually thin. The heart was much enlarged. The lungs were emphysematous; and it appeared to me that the thinning of the heart's valves was due to a process of atrophy, perhaps related pathologically to that which caused the pulmonary emphysema. I have since found, in the records of post-mortem examinations at Guy's Hospital, a similar case, observed by Dr. Moxon. It is that of a man, aged fifty-six, whose lungs are stated to have been senile and a little emphysematous, and to have contained much black matter. The heart was small; the right side was dilated, forming the apex; the pericardium was everywhere adherent; the mitral and aortic valves were very delicate in appearance.[1] This atrophy of the aortic valves is probably rare, and has not yet been shown to have any clinical significance. But there would certainly appear to be danger of the rupture of such thin structures, when strained in an effort of coughing or in any other way.

I have still to mention another abnormal appearance in the aortic valves, formerly supposed (by Corrigan) to render them incompetent, but now known to interfere in no way with their functions. I refer to the small openings which are sometimes found in the thin, crescent-shaped borders which extend on either side of the corpora Arantii. By some writers analogies have been found for such a fenestrated condition of the aortic valves in the normal state of the same valves in some of the lower animals; while others have regarded it as exhibiting an identity of structure between the semilunar and the cuspid valves; the filaments which remain above the apertures being supposed to answer to the chordæ of a mitral or tricuspid segment. It has also been doubted whether this fenestration of the aortic valves is the result of a slow atrophic change, or whether it is simply a defect of original development. And with reference to this point Dr. Wilks[1] states that he has seen it in young people, and has therefore always regarded it as congenital.

ETIOLOGY.—Before passing to the consideration of the other causes of disease of the cardiac valves, it may be convenient to discuss the views of certain writers, who have attributed to congenital malformation (or to intra-uterine disease) some affections that, as it appears to me, may arise at any period of life. These views are of considerable antiquity. In his account of Mr. Bulstrode's case, already referred to, John Hunter expresses a doubt whether the shrivelled appearance of the valves of the aorta was "a natural formation, or a disease." And of another specimen, in which there were two valves only instead of three, one of which had a kind of frænum or cross-bar attaching its middle to the side of the artery,—the catalogue of the Hunterian Museum says, "this malformation was in all probability congenital." Early in the present century, Mr. Burns described as a "species of mal-conformation of the heart," that condition in which the mitral valve, instead of being formed of two flaps, presents the appearance of a septum, with an aperture in its centre, stretching across the opening. More re-

[1] Since this was written I have (in November, 1874) met with a third instance of extreme thinning of the aortic valves, in another patient, who died of the effects of pulmonary emphysema.

[1] Pathological Anatomy, p. 93.

cently several observers have expressed similar opinions. Dr. Conway Evans,[1] in recording a case in which the mitral valve was funnel-shaped, says this condition was "evidently of congenital origin." Dr. Kelly has recently maintained the same view.[2] And Dr. Peacock devoted to this question a part of his first Croonian Lecture, delivered before the Royal College of Physicians in 1865.

The arguments for and against the opinion that certain affections of the valves are congenital require to be taken separately for the different valves.

And, first, with regard to the *mitral* valve. Great stress has been laid on the fact that mitral stenosis is very frequently associated with tricuspid stenosis. It is generally said that all the valves on the right side of the heart, which are so rarely subject to disease in after life, are much more liable to intra-uterine disease and to malformation than the valves on the left side. And congenital union of the pulmonary valves is really common, being indeed the most important malformation of the heart, and being generally attended with other evident malformations, such as an aperture in the septum, &c. Now, since the relatively higher function of the right side of the heart during fœtal life is supposed to be the cause of the greater liability to congenital disease in the pulmonary (as compared with the aortic) valves, writers have assumed that this liability must be shared by the tricuspid valve also. But, as a matter of fact, there is no proof that disease of the tricuspid valve before birth is otherwise than an exceptional occurrence, like disease of the aortic valves, or of the mitral valve at the same period. Friedreich,[3] indeed, says that in newly-born infants minute soft granulations are not rarely found on the auricular surface of the tricuspid valve; but, as they do not generally disturb the functions of the valve, he hardly regards them as morbid, considering them rather to stand on the boarder between physiological and pathological appearances. Dr. Peacock, however, has related[4] a case in which there was a thick exudation of recent lymph on the auricular surface of the tricuspid valves, in a cyanotic child, who died when about seven months old. It is stated that the cusps were thickened and adherent at their angles, so as to contract the dimensions of the orifice: but this still admitted a ball twenty-four lines in circumference, while the mitral aperture had a circumference of only eighteen lines. Friedreich has related a similar instance. Now it is certainly possible that tricuspid stenosis may have its origin in the inflammatory process described by these writers, as occurring in certain infants at or soon after birth. But in that case it might fairly be expected that the mitral valve, if affected at all, should be so in a slighter degree. Now I believe that it is invariably the case, that when both the valves in question are stenosed, the mitral is much thicker and much narrower than the tricuspid. For instance, it was so in Mr. E. Pye Smith's[1] case, which Dr. Peacock cites as of congenital origin.

Another argument in favor of the view that mitral stenosis may be due to malformation is based upon the fact that the patient has sometimes been in bad health for many years, or even from birth. Thus, in Mr. Pye Smith's case, the patient had been ailing all his life; and, although thirty-seven years of age, did not appear more than fifteen or sixteen, and had never presented any signs of puberty.[2] In 1870, Dr. Kelly exhibited to the Pathological Society[3] a heart with a button-hole mitral valve, taken from a woman aged thirty-three, who, even when a child, "could not run about well or indulge in any severe exertion on account of great shortness of breath and palpitation of the heart." But, so far as I can learn, such cases are quite exceptional. Patients affected with mitral stenosis generally state that they have had perfect health until a few months, or at most two or three years, before they first came under medical observation for their heart-disease. Moreover those who are practically conversant with the routine of morbid anatomy will, I think, agree with me, that in the bodies of those who have had rheumatic fever some years before death, the mitral valve is very frequently found presenting appearances which clearly indicate the gradual development of stenosis. In such cases the inferior edge of the valve is thickened, and harder than natural; its orifice no longer admits three fingers readily; its chordæ are beginning to cohere. Every stage of transition may be seen between a healthy valve and one presenting the most extreme degree of stenosis. My belief, therefore, is that it is needless to refer this affection of the mitral valve further back than a past attack of acute rheumatism, if the patient has had such an attack. And even when shortness of breath and other symptoms of cardiac defect have existed from childhood, it appears to me more likely that the stenosis is due to morbid changes arising in the years that may have elapsed

[1] Path. Trans. xvii. p. 90.
[2] Ibid. xxi. p. 91.
[3] Op. cit., p. 216.
[4] Path. Trans. v. p. 64.

[1] Path. Trans. iii. p. 283.
[2] Dr. Peacock, "Malformations of the Heart," 2d ed., 1866, p. 139.
[3] Path. Trans. xxi. p. 91.

since birth, than to malformation or disease occurring in the short period of intra-uterine life; especially since in the fœtus the left side of the heart is so situated as to be very little susceptible of morbid action.

Secondly, as concerns the *aortic* valves. Adhesion of two or more of these valves sometimes occurs in very young children. Thus Dr. Lloyd[1] exhibited to the Pathological Society the heart of an infant thirteen months old, in which there were only two aortic valves, and these were very red, rough, hard, cartilaginous on their surface and puckered: one of them was twice as large as the other, and had an indistinct ridge intersecting its centre. Dr. Workman,[2] again, showed to the same Society the heart of a little girl, only four years old at the time of death, in which the aortic orifice was much contracted, and its valves thickened and fused together. Again, adhesion of the aortic valves has sometimes been found in young persons, associated with the similar change in the pulmonary valves which is believed to be invariably of congenital origin. Thus Dr. Wilks[3] has recorded the case of a girl, æt. eighteen, in whom the pulmonary valves were adherent, and the aortic valves were two in number, the larger of them having in its interior a raised line indicating the point of union of two former valves. Dr. Ogle[4] saw another instance of the same kind, in a girl fourteen years old, in whom "two contiguous aortic valves had their adjoining angles torn away from their attachment to the aorta, and subsequently united to each other at a lower level." It is no doubt probable that in all these cases the union of the aortic valves occurred before birth: but such cases are extremely rare, and surely afford no ground for supposing that the adhesions which are so commonly found at an advanced age are also congenital.

I must next insist on the fact, that the cases last referred to have characters which distinguish them in a very important way from those in which the union of the valves is known to be congenital. In the first place, *partial* adhesions of the aortic valves are very commonly met with in older subjects, especially when the coats of the aorta are also diseased. The adjacent edges of the valves are found to have grown together for one or two lines, the rest remaining free. There can be no question of any congenital defect, since I am not aware that such partial adhesions are ever observed at an early period of life:[5] yet it is obvious that the continuation of the same process would lead to the complete fusion of the valves, after which the line of union might be expected to gradually waste and disappear. Again, when the aortic valves are adherent, the orifice is generally irregular, and the substance of the valves greatly puckered and often deformed by large masses of calcareous deposit. Of this several capital illustrations are given in Dr. Peacock's published Croonian Lectures. He supposes that in these cases the union of the valves took place before birth. But the appearance is very different from that which is seen in the affection of the pulmonary valves which (as has already been stated) is known to be congenital. In that case the united valves form a smooth, dome-shaped body, with a regular orifice in its centre, and three small ridges or frœna on its upper surface, placed at equal distances from one another.

An argument in favor of the view that adhesions of the aortic valves are congenital has been found in the fact that, when there are only two valves, they are sometimes of equal size. It is supposed that the union must have taken place while the valves were in course of development. In reality, however, this fact merely proves that adhesion occurred before they were fully grown. A case in point recently came under my notice. A man, æt. twenty-three, died in Guy's Hospital of febrile delirium in the course of acute rheumatism. There was no recent valvular disease; but two of the aortic valves were adherent, and the resulting valve was scarcely larger than the third valve, which was itself much thickened along its whole edge, and also contracted, so that it lay flat against the aortic wall. The pericardial sac was universally closed by old adhesions. Now this patient was said to have had chorea and rheumatic fever in childhood, and afterwards to have suffered from distinct symptoms of heart-disease, dyspnœa, palpitation, &c. His illness at that time was doubtless the cause of the morbid changes both outside his heart and in its interior.

Two points remain to be considered, which afford powerful, and to my mind convincing, arguments against the view that adhesions of the aortic valves are always, or even frequently, of congenital origin. The first is the extreme frequency with which such adhesions are found in the later periods of life. Thus, according to Dr. Peacock,[1] "of forty-three cases in which the aortic valves were diseased, either alone or in conjunction with the

[1] Path. Trans. i. p. 60.
[2] Ibid. xviii. p. 55.
[3] Ibid. x. p. 80.
[4] Ibid. v. p. 70.
[5] Since this was written I have met with an instance, in which a partial adhesion of two of the aortic valves was found in the body of a boy, æt. 16, who had been drowned.

[1] Croonian Lectures.

mitral valve, in eleven (or 25·5 per cent.) there was malformation of the valves, which probably laid the foundations of the subsequent disease." Dr. Peacock goes on to say that this proportion is much larger than would *à priori* have been expected. I think that it shows clearly how untenable is his position. It is scarcely conceivable that a congenital malformation, which we have seen to be extremely rare in infants, should be so commonly found in adults. Again, the duration of life in the cases under consideration is altogether adverse to the opinion that the valvular disease had existed from the time of birth. Congenital malformation may, of course, be found in the bodies of those who have lived long, provided these malformations were not such as to interfere with the functions of any vital organ: and in exceptional cases, even when they did so interfere. But, if we average a considerable number of cases, we may surely assert with confidence that a congenital adhesion of the aortic valves, greatly narrowing the aperture, must inevitably tend to shorten life. Yet we find Dr. Peacock deducing from his statistical inquiries that "in cases of aortic valvular disease assigned to malformation, the age of all the patients averaged 42·3 years, and the extremes of ages were eighteen and seventy-six: while the mean age of the patients in whom aortic valvular disease originated in other ways was only slightly greater, or 47·4 years, and the extremes of age were twenty-one and sixty-two."

The influence of congenital defect in the causation of diseases of the cardiac valves is perhaps not limited to the cases which we have hitherto been considering. According to Virchow,[1] who has recently devoted much attention to the defective development of the aorta that is found in patients affected with chlorosis, inflammation of the mitral or aortic valves is found with disproportionate frequency in these cases. He thinks that the congenital narrowness of the aorta impedes the outflow of blood from the left ventricle, and so increases the strain to which the valves are exposed. In connection with this subject, the late Dr. Barlow[2] must be mentioned. He believed that in certain young subjects the trachea failed to undergo due development. This, he thought, led to imperfect expansion of the chest, and consequently the supply of blood to the left side of the heart was impeded; and not only the aorta, but likewise even the orifices of the heart's chambers, were prevented from attaining their proper size. Dr. Wilks has put up in the museum of Guy's Hospital[1] a specimen illustrative of Dr. Barlow's theory. I find, however, that in this, as in both Dr. Barlow's cases, mitral stenosis existed in a degree quite disproportionate to that of the other changes; and I must confess that I am inclined to think that this was the primary lesion, that it arose in childhood (as seems often to be the case), and that the smallness of the trachea was but a part of a defective development of the body generally, consequent on the imperfect state of the circulation caused by the valvular disease.

We have now to ask, what *are* the causes by which diseases of the valves of the heart are generally produced? And the answer to this question is, that by far the most common cause is an attack of "rheumatic fever," or "articular rheumatism," as it is termed in Dr. Garrod's article in the first volume of this work. Dr. Garrod has there pointed out that as far back as 1788 Dr. Pitcairn had noticed that persons subject to rheumatism were attacked more frequently than others with symptoms of heart disease; and that other writers at the commencement of the present century mentioned the same fact. But they were exceptions. Few things in medical literature are more curious, than to read the works of Burns and Kreysig and Corvisart on diseases of the heart, and to find that they had not the slightest suspicion of the rheumatic origin of these affections. So late as the year 1835, indeed, Bouillaud[2] was able to claim for himself the discovery that rheumatic pericarditis (a disease at that time generally recognized), is frequently accompanied by inflammation of the lining membrane of the heart—for which disease he then proposed the name of endocarditis.

After this I suppose that the occurrence of valvular disease of the heart in the course of rheumatic fever soon became universally known; and several writers have published numerical statements with regard to its frequency. In these there is a fair general agreement. Dr. Peacock[3] quotes Dr. Fuller as stating that in his cases of acute rheumatism some cardiac complication was present in 49·3 per cent.; while Dr. Barclay found that in his cases the proportion was 39 per cent. Dr. Peacock gives 42·4 per cent. as the corresponding proportion in the cases which came under his care from 1846 to 1868.

It is to be observed that, in these figures, pericarditis is included as well as endocarditis; and also that in many cases

[1] "Ueber die Chlorose, und die damit zusammenhängenden Anomalien im Gefässapparatus," Berlin, 1872, p. 18.

[2] Guy's Hospital Reports, 1st series, vol. vi. p. 235.

[1] Prep. 1412[55]. Catalogue, vol. i. p. 31.

[2] "Traité Clinique des Maladies du Cœur," Paris, tom. i. p. 275.

[3] Clinical Society's Transactions, ii. p. 222.

there was old disease of the heart from former attacks of acute rheumatism. Both Dr. Fuller and Dr. Peacock have attempted to distinguish these cases, and to determine the exact frequency of recent endocardial mischief; but I doubt whether much reliance can be placed upon their results, which are based upon stethoscopical evidence only. Indeed, it is questionable whether we can trust to auscultation for determining the presence or absence of early endocarditis. I believe that at Guy's Hospital it has been found that in fatal cases of acute rheumatism (and still more of chorea) there has been by no means a close correspondence between the observation of a murmur during life and the detection of vegetations on the valves after death. Sometimes, when a systolic murmur has been present, the valves have been healthy; and, on the other hand, when no murmur could be detected they have been found to be diseased.

On the other hand, objections of at least equal force may be urged against the use of pathological observations to determine the question as to the numerical frequency of endocarditis in acute rheumatism. As far as I know, no series of unselected cases of fatal acute rheumatism has as yet ever been published. But I find that at Guy's Hospital, in a period of rather more than twenty years, there have been thirty-two such cases, in most of which the disease was a first attack. Now in twelve of these cases the valves were perfectly healthy; in twenty cases one or more of them was diseased. Six times the mitral valve was alone affected; three times the aortic valves alone: in ten cases both the mitral and the aortic valves were diseased; and in one other case, both these and the tricuspid also. This would give 62·5 per cent., as the proportion of cases of acute rheumatism in which acute endocarditis occurs.

Now I shall presently show that in all these cases the changes in the valves were slight, and that they were not at all concerned in causing death. The fatal termination was doubtless generally due to hyperpyrexia, which (as is well known) often comes on in cases that had previously appeared to be of a mild character. Still I think it cannot be denied that the thirty-two fatal cases were on an average cases of excessive severity; for in twenty-one of them there was recent pericarditis. Probably, therefore, we cannot accept these cases as showing that 62·5 per cent. is really the proportion of cases of acute rheumatism in which endocarditis occurs. Indeed, if the cases could be regarded as average ones, we should have to suppose the proportion to be really higher still, for in many of them death occurred at a very early stage.

After all, it may perhaps be said that the exact determination of the frequency of endocarditis in acute rheumatism is of less consequence than has been supposed: for Dr. Peacock's observations render it probable that this may vary considerably at different periods and among different classes of the population. For practical purposes we may perhaps take it at from 40 to 50 per cent.

Next to acute rheumatism, chorea is the disease which most frequently gives rise to disease of the cardiac valves. I believe that this fact was first pointed out by Dr. Hughes in a paper in the Guy's Hospital Reports.[1] He found that out of 14 fatal cases of chorea, in which the state of the valves of the heart is mentioned, there were only two in which these structures were reported to be healthy. In the last twenty years we have had at Guy's Hospital sixteen other fatal cases of chorea, in which post-mortem examinations have been made; and in only two of these were all the valves perfectly healthy. Nine times there were vegetations on the mitral valve alone; twice on the aortic valves alone; three times on both the mitral and the aortic valves. Probably, however, these figures must not be taken as indicating the liability to the occurrence of cardiac disease in non-fatal cases of chorea, since severe forms of the disease are at once more likely to destroy life than mild ones, and more likely also to be complicated with valvular inflammation.[2] Thus it would not be safe to infer (as might at first be supposed) that disease of the cardiac valves is absolutely of more constant occurrence in chorea than in acute rheumatism itself.

Even in protracted fatal cases of chorea, I believe that the cardiac affection is always slight in degree; not going beyond the presence of a row of minute granulations on the edge of one or more of the valves, which might easily escape notice, if not specially looked for. Pericarditis, again, scarcely ever occurs as a result of chorea apart from rheumatism; having, in fact, been present in only one of the thirteen cases that I have collected. It is, I think, generally supposed that acute rheumatism differs from chorea, not only in the liability to pericarditis, but also in the much greater severity and extent of the endocarditis which attends it. It was, therefore, with some surprise that I found that in each of the fourteen cases of

[1] Series ii. vol. iv. p. 360; and Series iii. vol. i. p. 217.

[2] I may mention, however, that in one of the fatal cases of chorea under consideration the girl's death was accidental, having been due to diphtheria, which she caught from another patient. In this instance vegetations were found in the aortic valves.

fatal acute rheumatism, which I have already mentioned as having presented valvular disease, the affection consisted merely in the presence of a row of minute granulations, precisely like those seen in chorea. In no instance were those larger vegetations present that are so commonly found under other conditions, nor was there ever any ulceration.

A third disease, which may also lead to changes in the valves, exactly like those which occur in acute rheumatism and in chorea, is pyæmia. In 1865 I exhibited to the Pathological Society two specimens in which there were well-marked vegetations on the mitral valve, in pyæmia after surgical operations. Similar cases have since been recorded by other observers. In six years (from 1866 to 1871 inclusive) I find that the records of post-mortem examinations at Guy's Hospital contain twelve cases of pyæmia in which one or more of the cardiac valves has been found diseased. In two or three of these cases, however, the affected valve has been found ulcerated. This has sometimes been observed, when the pyæmia was evidently of external origin. Thus in 1867 I find a case of pyæmia recorded, in which part of the flap of the mitral valve was found ulcerated away from its chordæ. The point is of some importance, because (as I have shown elsewhere)[1] it suggests a doubt as to the interpretation of some of the cases in which ulcerative endocarditis has been believed to have been the *cause* of blood-poisoning by Dr. Kirkes and others.

Another, but an indirect, cause of endocarditis is, I believe, the existence of chronic spinal deformity. I have recently[2] recorded several cases of this kind in which death took place from pulmonary obstruction and dropsy. In one of them the aortic valves were found to be retroverted and covered with vegetations. This at first seemed to be difficult of explanation: but I subsequently found reason to attribute it to the increased tension within the aorta that must have resulted from the sharp bend in its descending part where it was tied by its intercostal branches into the very acute angle formed by the diseased vertebræ. Since then I have seen acute endocarditis affecting the aortic and the mitral valves in a man who died of bronchitis and dilatation of the bronchial tubes, consequent on anchylosis of all the vertebræ together, and of the ribs to the vertebræ. But in this case I did not discover any evidence that the aorta had been compressed or interfered with. A somewhat analogous case to the first one mentioned in this paragraph has, however, occurred to me, in which the aorta was compressed by large masses of caseous glands, and in which the aortic valves were affected with acute endocarditis. And Dr. Goodhart lately met with a case of congenital stenosis of the descending part of the arch of the aorta in which there was a similar affection of the valves.

There are still some other diseases in which similar minute granulations on the cardiac valves have been occasionally found in the post-mortem theatre of Guy's. Thus in six years (1866–71) I find their presence recorded in three cases of cancer (of the uterus, the liver, and the gall-bladder respectively), in one case of phthisis, in one case of lobular pneumonia, in one case of Bright's disease, in one case of puerperal peritonitis, in one case of syphilitic disease of the liver, twice in cases of dilated heart, and once when there was old adhesion of the two surfaces of the pericardium. They were also found in one case of cholera; but as the disease proved fatal in 12 hours, it must be supposed that they existed before the attack commenced.

It has been stated that in all the fatal cases of rheumatic fever that have come under observation at Guy's Hospital within the last few years the changes in the valves have been slight, and in fact identical with those which are well known to occur in chorea. But I must not omit to mention that writers have recorded instances in which the valves have been much more severely attacked. Thus Sir Thomas Watson[1] relates two cases in which death is stated to have occurred in a first attack of rheumatic fever, complicated with acute pleurisy, three weeks and four weeks respectively after admission of the patients into hospital. In neither instance was any trace of pericarditis discovered after death. In each, one of the aortic valves was a mass of ragged ulceration; and the adjacent portions of the two other valves were in a slighter degree implicated. In one of the cases the ulcerating process had penetrated through the valve into the muscular substance beyond, and eaten a hole completely through the septum. In the other case an abscess as large as a hazel-nut was found in the muscular substance of the septum, immediately opposite the disorganized valve. Now the occurrence of such an abscess is so rare in acute rheumatism, that I almost think it is permissible to express a doubt whether the case was not rather one of pyæmia, or of primary ulcerative endocarditis, with articular pains: for such cases have often been mistaken for cases of rheumatic

[1] Path. Trans. xvii. p. 60.

[2] Guy's Hospital Reports, series iii. vol. xix. p. 199.

[1] Lectures on the Principles and Practice of Physic, 4th edition, 1857, p. 315.

fever. Sir Thomas Watson goes on to remark that these were the only instances of the kind which he had seen. In the other fatal cases of acute rheumatism related in his book only slight changes in the cardiac valves were found after death.

It may be convenient here to complete all that has to be said in reference to the general etiology of the acute destructive disease of the valves, which has recently attracted so much notice, under the name of Ulcerative Endocarditis—a name first given to it, I believe by Charcot and Vulpian in 1861. Besides its occasional origin in pyæmia and perhaps in acute rheumatism (as just mentioned), this affection has often been found to occur in the latter months of pregnancy, or a few weeks after delivery. Virchow[1] says that in the Charité at Berlin there is never a year in which several instances of this do not occur. It is true that in the majority of these cases inflammation of the uterus is present, so that the endocarditis might be supposed to be simply a manifestation of pyæmia, but occasionally the pelvic organs are quite healthy. Very often, however, ulcerative endocarditis can be traced to none of these conditions, and may be said to arise spontaneously, so far as our knowledge at this time extends. The patient may have previously been a healthy subject, and the disease may arise suddenly with shivering, so that it is often mistaken for enteric fever or some other acute disease. But such cases are exceptional; much more commonly ulcerative endocarditis attacks valves which were previously unsound, and its effects overlie and are blended with those of chronic valvular disease.

We may now pass to consider the causation of chronic affections of the cardiac valves; and of these a large proportion, probably the majority, arise out of the acute affections already described as occurring in the course of acute rheumatism and of chorea (for pyæmia, being itself almost always fatal, can hardly be credited with a share in the production of these more remote changes). With regard to the details of the processes by which these results are brought about, it may be said that at the present time we know scarcely anything. We do not even know whether an acute affection, once developed, ever subsides entirely, and leaves the valve perfectly healthy. I think that this must not infrequently occur, especially after chorea, for we have seen that the valves are very often affected in this common malady, and yet it has in my experience comparatively seldom happened that patients laboring under valvular disease have stated that they had previously had chorea. Another argument to the same effect may perhaps be found in the comparative rarity of chronic rheumatic disease of the aortic valves in women. We have seen that the aortic valves were found to be affected in thirteen out of twenty cases of acute rheumatic endocarditis. Now of these cases at least seven occurred in females. But in the years 1867–71, for 23 cases of chronic aortic disease with history of previous acute rheumatism in males, only 6 cases in females came under observation in the post-mortem theatre at Guy's. It would seem to follow that in women rheumatic inflammation of the aortic valves must often subside entirely without leading to chronic disease. If this be true, it is of very great importance, for it may teach us a most valuable lesson. We shall presently see that the aortic valves are in men liable to strain and pressure, from which in the other sex they are free; and that in consequence non-rheumatic disease of the aortic valves is in men very common, in women comparatively rare. It appears very probable that the same freedom from strain and pressure may also enable these valves in women to recover from rheumatic inflammation more perfectly than in men. And, if so, we may learn how to obviate the ill-effects of such inflammation in both sexes, and in the case of all the valves, by keeping the patients at rest, and making them abstain from work and exertion of every kind, for a long period after an attack of endocarditis.

It is at any rate certain that the granulations, which appear to be constantly present in acute affections of the valves, have generally but a transitory existence. Sometimes, indeed, they become calcified, and can thus be recognized long after all acute disease has passed away. But more often they disappear, and thus in chronic rheumatic disease the surface of the thickened and calcified valves is often found to be perfectly free from them. When uncalcified granulations or vegetations are found in cases of long standing, I believe that they are always of rather recent formation, and due to the supervention of an acute inflammation, to which (as we have seen) valves already diseased are particularly liable.

In a considerable proportion of cases, however, chronic valvular disease can be traced to none of the conditions that have as yet been mentioned. And its etiology appears then to be different in the case of different valves. Affections of the aortic valves often accompany similar morbid changes in the walls of the base of the aorta itself, changes often spoken of as "atheromatous," but really dependent

[1] "Ueber die Chlorose . . . Endocarditis Puerperalis," Berlin, 1872, p. 20; see also Trousseau, "Lectures on Clinical Medicine," New Sydenham Society's Trans. vol. iv. p. 459.

on a chronic inflammation of the arterial coats, or (as it is termed by Virchow and others) an arteritis deformans. This disease occurs especially in men (as, for example, sawyers, smiths, strikers and riveters, bricklayers' laborers, and hodmen) whose occupations involve great muscular efforts, by which the arterial pressure and the strain on the aortic coats are increased. Writers have generally stated that persons of rather advanced age are more liable to it, but Dr. Allbutt says that it is very common in Leeds among quite young men.[1] According to Peacock, a similar affection is not infrequently observed to occur in girls engaged as nursemaids, and in other servants, who are subjected to straining efforts before they have attained their full strength. It is further to be noted that, although the affection of the valves in all these cases appears to be identical in nature with that which occurs in the walls of the aorta, the two are by no means invariably affected in an equal degree. Dr. Allbutt has suggested the opinion that continuous labor, such as hammer-work, is more injurious to the aorta itself, and that sudden strains, like the lifting of weights, tell rather upon the valves. The relative frequency with which valvular disease is thus due respectively to mechanical strain or injury and to the effects of antecedent acute disease, probably differs greatly among different classes of the population, and in different localities, according to the occupations of the lower order in them. Dr. Allbutt tells us that in Leeds, in hospital practice, heart diseases due to acute rheumatism are among young men fewer than those which he has learnt to attribute to over-exertion of the body. In this statement, however, no account is taken of the affections of different valves separately. I believe that in hospital practice in London one fails to obtain a history of a past attack of rheumatic fever in at least half the cases of chronic regurgitant disease of the aortic valves that are met with in adults, and that in almost all these cases the changes in the valves are associated with similar changes in the walls of the aorta, and are the result of habitual or repeated straining efforts of one kind or another.

It is far otherwise in the case of the mitral valve. In this structure atheroma appears only in the form of slight cream-colored patches, placed near the base of the valve, and therefore incapable of impeding its closure. Nor can any morbid change in the mitral valve be traced in association with the disease of the aortic valves just described as due to arteritis deformans. Still, there are a large number of cases of mitral stenosis in which no previous attack of acute rheumatism or chorea seems to have occurred, and the subjects are many of them children, in whom no definite illness could have been overlooked or forgotten. Such cases have been by some writers attributed to congenital malformation, a view which I have already endeavored to disprove. Other observers have supposed them to be due to latent rheumatism: that is, to manifestations of the rheumatic state, which has for some reason failed to display itself in the characteristic articular malady. On closer inquiry it may sometimes be elicited that such patients have formerly suffered from "growing pains" or "rheumatic pains" of greater or less severity, and certain observers, among whom may be mentioned no less an authority than the late Dr. Addison, have pressed these into service as affording evidence of the existence of a constitutional state. It must be admitted that rheumatic pericarditis often precedes any affection of the joints, and that in young people already suffering from valvular disease of the heart without any history of previous rheumatism, the joints sometimes become swollen and painful, or chorea is developed. I have therefore no doubt that many of these cases of mitral stenosis are really the results of a rheumatic tendency. But it is still a question whether they are not too frequent for such an explanation to be applicable to all of them in which no history of previous rheumatism can be traced.

It would appear, therefore, that the mitral valve is very liable, even in children and young subjects, to undergo those changes which lead to stenosis, either as the result of a spontaneous chronic morbid process, or else as the consequence of some disease (other than rheumatism or chorea), the tendency of which to produce endocarditis is as yet unknown. Can this disease be scarlatina or diphtheria? I have read (but I do not know where) that M. Bouchut has recently brought forward diphtheria as often leading to the formation of granulations on the mitral valve, but in the few autopsies that I have made these have been absent. As is well known, scarlatina is often followed by acute rheumatism, or an articular disease allied to it: and this may be complicated with endocarditis, as has been shown by Trousseau and others. Nay, in cases of chronic valvular disease it is not very uncommon for the patient's illness to be referred back to an attack of scarlatina. But I am nevertheless very doubtful whether this disease can be called in to account for the cases that now need explanation, for I fail to find any evidence that scar-

[1] "The Effects of Overwork and Strain on the Heart and Great Bloodvessels," St. George's Hospital Reports, v. p. 23.

latina in itself is capable of setting up endocarditis. So far as I am aware, when a child dies of scarlatinal dropsy, or of any one of its other sequelæ, the valves are constantly found healthy.

Within the last few years it has been a matter of frequent discussion among pathologists whether syphilis is ever a cause of disease of the cardiac valves. The idea is indeed no new one; for Corvisart[1] long ago suggested that vegetations of the valves were of venereal nature. No less an authority than Virchow[2] has since stated his readiness to admit the possibility that this may sometimes be the case: but he has given no case in proof. When Mr. Myers[3] and other army surgeons recently showed the frequency of heart disease in soldiers, and attributed it to the faulty clothing and accoutrements which they are made to wear, or to the exercises they are called upon to perform, it was objected that soldiers are very subject to syphilis, and that this was really the cause of the cardiac affections to which they are liable. But to that argument a rejoinder was made that sailors are equally apt to have venereal disease, while they are not found to suffer in the same proportion from morbus cordis. For my own part, I confess that I have met with no facts, either by observation or by reading, that would lead me to believe that syphilis has anything to do with the diseases under consideration.

The effects of sudden violence in injuring the cardiac valves still remain to be considered. Corvisart appears to have been the first writer who reported a case in which the valves of the heart were clearly shown to have been injured during muscular exertion. Since that time several instances of the kind have been placed on record: and in 1865 Dr. Peacock[4] collected seventeen cases, four of which had come under his own observation.

It has already been stated that in advanced valvular disease softening and laceration are very apt to occur, whether as a result of slight muscular efforts, or independently of any such cause. But symptoms of heart disease have then generally existed for a long time. The peculiarity of the cases now under consideration is, that the subjects of them are apparently in perfect health when the injury arises, and have never had rheumatism, or been suspected of any cardiac disease. It is indeed true that such accidents have been observed chiefly in adult men, whose occupations have long been such as are known to carry with them the liability to induce chronic changes in the heart and great vessels: and some have therefore argued that the lacerated valve might not have been in a healthy state at the time of the injury, but might have previously undergone degeneration. And this supposition is very difficult to negative, since death seldom occurs in such cases until after the lapse of a considerable interval, when of course the state of the valves before their rupture cannot be determined. But in this, as in so many other instances, the maxim may be applied, "*De non apparentibus et de non existentibus, eadem est ratio.*" For practical purposes it is more important to remember that a valve may rupture in a man who has hitherto been active and robust, and free from the slightest symptom of cardiac disease, than to discuss whether the valve has or has not previously undergone slight degenerative changes, which no one could have discovered or suspected.

Perhaps the most striking example that could be quoted, in which mechanical injury led to the rupture of a previously healthy valve, is recorded by Dr. Wilks in the sixteenth volume of the Pathological Transactions (p. 77). The patient, a youth aged nineteen, fell from a height, and alighting on a stone struck his left side violently, so as to lacerate a portion of the intestine, as a consequence of which peritonitis arose, and proved fatal on the third day. It had been observed that he had considerable oppression at the chest, and much distress in breathing after the accident; but unfortunately no stethoscopical examination was made. At the post-mortem examination it was found that the most posterior of the aortic valves was torn through, from its free margin to its base, a little on one side of its attached edge. Only a ragged portion remained attached to the aorta, while the bulk of the valve was free to flap backwards and forwards. A small deposit of fibrin had already commenced to form on the ragged edges.

In this case there was no mark of bruising on the chest, nor any sign of injury external to the heart. But I think it can hardly be doubted, from the history of the accident, that the cause of the laceration was the blow on the side, rather than any muscular effort made by the youth at the moment. The case would then be strictly parallel to those which are not unfrequently met with, in which an accident gives rise to severe laceration of some one of the abdominal viscera, or of the interior of the brain, without there being any bruise on the surface, or visible track by which the vibrations had passed to the deeper structures.

In this respect, however, Dr. Wilks's case would appear to be exceptional, if the conclusions of Dr. Peacock are to be

[1] Op. cit. p. 194.
[2] Arch. f. Path. Anat. xv. 1858, p. 288.
[3] Path. Trans. xx. p. 141.
[4] Croonian Lectures.

relied on in reference to the question at issue. The last-named observer collected seventeen cases of rupture of a valve from injury. In three or four of them the patient had sustained direct injuries at the same time; but Dr. Peacock was nevertheless of opinion that in all of them the immediate cause of the rupture was the violent effort made at the same moment. "In one case the patient had made a long and rapid journey on horseback: two men were pulling or loading heavy casks, two were running violently, one was rowing, another was striking with a heavy sledge, a third was endeavoring to force open a door, and others were climbing rapidly, endeavoring to leap over a fence, and carrying heavy deals. In others, violent coughing appears to have been the cause of the rupture."

The comparatively small number of cases which Dr. Peacock could collect is in itself a sufficient proof that rupture of a valve in a previously healthy subject is after all a decidedly rare occurrence; and this conclusion is confirmed by the fact that few cases of the kind are recorded in the Pathological Transactions, which are generally particularly rich in examples of the more striking forms of disease. Among the different valves, those of the aorta are the most liable to injury, having probably been ruptured in ten out of Dr. Peacock's seventeen cases. Laceration of the columns of the mitral valve seem to have occurred in four instances, and of the tricuspid in the remaining three. In the aortic valves the part torn appears to be usually the attached margin or angle.

Effects.—Diseases of the cardiac valves produce serious effects of various kinds, by which the patient's health is disturbed and his life often endangered. In these are to be found the "*symptoms*" of the diseases in question. But before entering upon their consideration it will be convenient to discuss first another class of effects also resulting from such diseases, and in the eyes of the physician no less important, although to the patient himself they are of but little direct concern. I refer to the altered sounds, accompanying the heart's action, that are heard by the ear or stethoscope applied to the patient's chest—the "*auscultatory signs*" of valvular lesions.

In England these altered sounds are termed indifferently "*murmurs*" or "*bruits.*" The latter term is of course a relic of the French influence that predominated in this country for many years after the discovery of auscultation. But it may be worth while to note that French writers themselves apply the word "*bruit*" indifferently to the natural heart sounds, and to the murmurs heard in disease, adding the epithet "*anormal*" when a murmur is to be referred to, or else designating it a "*bruit de souffle*," from the blowing character which generally belongs to such morbid sounds.

Numerous theories have been formed to explain the production of cardiac murmurs; but they have attracted more attention abroad than in this country, English writers having generally passed them by, as of theoretical rather than of practical importance. One of the earliest of such theories was, however, originally propounded by Sir Dominic (then Dr.) Corrigan, in the year 1829; and, quite recently, the labors of certain French observers have gone far towards establishing the correctness of this view, to the exclusion of all others.

It must be remarked that murmurs are by no means confined to the heart, but may arise in almost any part of the circulatory system; and this fact has to be taken into account by any theory that would explain their production. Laennec had ascribed the *bruit de souffle* to "a special vital state—a sort of spasm or tension of an artery."[1] Corrigan[2] easily showed that this opinion was untenable. "Apply," he says, "the stethoscope under the outermost third of the clavicle, not allowing it to pass (? press) on the subclavian. In a strong healthy man, not agitated, the mere impulse of the diastole of the vessel is felt. Now compress the artery *above* the clavicle, so as to diminish the current of blood through it: a loud *bruit de souffle* is heard. Make strong pressure, so as to stop the flow of blood: no sound is heard. If the sound in this experiment arose from the arterial tube being excited into muscular action by the stimulus of the pressure, why does it cease when the stimulus is increased?" And he goes on to give the following explanation of the *bruit de souffle*:—"When an artery is pressed upon, as in the experiment above related, the motion of the blood in the artery immediately beyond the constricted part (looking from the heart) is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube, and continuing its way through surrounding fluid. The rushing of the fluid is combined with a trembling of the artery, and the sensation to the sense of hearing is the *bruit de souffle.*" Further on he applies the same theory to the murmurs heard in aneurisms

[1] "Traité de l'auscultation médiate," seconde édition, 1826. In his first edition Laennec had described the *bruit de souffle* as occurring when the heart was too full of blood, and as caused by contraction of one of the heart's orifices. But afterwards, finding that there was no organic lesion which coincided constantly with the *bruit*, he expressed the opinion cited in the text.

[2] Lancet, 1829, vol. ii. p. 1.

and in narrowing of the auriculo-ventricular orifices of the heart, &c.; and he proves that the condition supposed to produce murmur may be imitated by passing a forcible current of water through a portion of small intestine. In this experiment, as soon as constriction was made on any part, a very loud *bruit de souffle* immediately became evident just below the narrowed part, where no sound had been previously heard.

The writers who followed Corrigan dealt with the causation of cardiac murmurs from an entirely different point of view. By Gendrin (1841–2) they were placed in the same category with the morbid sounds heard in pericardial inflammations; and since the latter are due to friction between the two serous surfaces, he naturally attributed the former, which he termed "*bruits de frottement endocardiaques*," to friction between the blood and the surface over which it passes. This *friction theory* has since been generally adopted.[1]

But in the year 1858, Chauveau, of Lyons, published an important memoir, in which he endeavored to show that the friction theory was untenable, while he revived Corrigan's views, and placed them on a firmer physical basis.[2] In the first place, he proved that roughening the interior of an artery does not cause a bruit. Thus he exposed the carotid artery of a horse, and tore through the internal and middle coats, at four or five points near one another. The tube was, of course, greatly roughened, but no bruit was produced. On the other hand, whenever a dilatation was placed in the course of an artery, the blood entering the dilated part gave rise to a *bruit de souffle*. This Chauveau ascribed to the fact that under such circumstances a sonorous jet is formed, such as Savart studied experimentally under the name of the "*veine fluide*." He even laid bare the pulmonary artery of a horse (in which artificial respiration was kept up after pithing), and introduced his finger into the artery through a slit in its wall. When the vessel was narrowed by tightening a thread round its base, he could feel the vibrations of the *veine fluide* which was generated, whereas the flow of the blood had previously been scarcely perceptible.

Chauveau therefore sums up the results of his experiments in the statement that "the *bruit de souffle* is produced by the vibrations of the *veine fluide*, which is always formed when the blood passes into a part of the circulating apparatus actually or relatively dilated."

Very soon after the discovery of auscultation, it was found that a *bruit de souffle* could sometimes be heard even in persons in whom the heart was perfectly healthy, especially in those who were chlorotic or anæmic. Such a bruit has been generally attributed to the thin and watery state of the blood, rendering it liable to be thrown into vibrations while flowing through the vessels. This explanation, however, is far from satisfactory, and has indeed been rendered untenable by the experiments of Chauveau and others, who have shown (in opposition to some earlier experiments of De la Harpe) that the production of murmurs in general is altogether independent of the nature of the fluid in which they are formed.

It would seem, however, that the theory of Chauveau, just stated, is applicable to such anæmic murmurs. As is well known, these are of two kinds—the arterial, and the venous, or "*bruit de diable*." The former is audible chiefly at the base of the heart, along the aorta, or the pulmonary artery. Now, Chauveau has shown that in anæmic horses the arteries generally are one-third smaller than in healthy animals; the mass of blood is greatly reduced; the heart and its orifices become diminished in size, so as to adjust themselves to the altered volume of the blood; but the great arteries, being comparatively inelastic, retract less perfectly. The conditions for the production of a murmur are thus satisfied. Moreover, the arterial pressure during the cardiac diastole is found to be very much lower than usual; hence, when the artery becomes distended by the heart's contraction, the force with which the blood enters is far greater than in health. In other words, the *range* of pressure within the arterial system is greatly increased.

The venous anæmic murmur, or *bruit de diable*, receives a very similar explanation. As Hamernyk long ago showed, it is met with only at the root of the neck; and the cause of this lies in the anatomical fact (first pointed out by Bérard) that in this region the lower ends of the jugular and subclavian veins on each side are adherent to the deep cervical fascia, and therefore cannot collapse. This venous ampulla, as it has been termed, evidently affords the conditions necessary for the generation of a *veine fluide*, whenever the blood-stream in the jugular vein above is narrowed, whether by simple adjustment of its calibre to the diminished volume of the blood in anæmia, or by the pressure of the stethoscope, or by both combined. Thus, as might be expected, in some healthy subjects a *bruit de diable* can be generated by nice compression of the jugular vein with the stethoscope; and, on the other hand, even in those who are anæmic a certain amount of pressure is required to develop the murmur, unless

[1] See Walshe, "A Practical Treatise on Diseases of the Heart and Great Vessels," 1862, p. 86.

[2] Gazette Médicale de Paris, 1858, p. 247.

the morbid state is present in an extreme degree.

Since the publication of Chauveau's essay, this subject has been studied by several French writers, especially by Marey, Luton,[1] and Bergeon,[2] who have expressed their general adhesion to his views. And for my own part I think that they have proved that a *bruit de souffle* occurring in an artery or vein at a distance from the heart is invariably caused by the generation of a sonorous *veine fluide*, and due to the passage of a narrow jet of blood into a wider cavity or part of the vessel.

But this explanation is certainly not applicable to all *cardiac* murmurs. The bruit caused by a sonorous *veine fluide* is heard only in the dilated part of the channel, and not at all (or very faintly) in the narrowed part behind it. In other words, it is propagated in the direction of the stream of fluid. Now, as we shall see presently, some cardiac murmurs obey this law; among which are those of mitral and aortic stenosis. But in mitral and in aortic imperfection this is not the case: the murmur is audible, not only in the direction of the regurgitant blood-stream, but also on the other side of the orifice (over the left ventricle in the case of the mitral valve; along the aorta in the case of the aortic valves). Now Bergeon has given a complete explanation of this, and has shown that it may be easily imitated in experiments (such as have several times been referred to), in which water is made to traverse tubes narrowed at a certain point. One has only to provide the tube at the seat of constriction with a lip or rim projecting backwards into the stream, and a second murmur is at once generated, which is heard behind the obstruction. A *cul de sac* is formed, and the fluid which occupies this receives the shock of the onward current, and is thrown into sonorous vibrations. It is evident that this experiment exactly meets the case. The incompetent valves, whether mitral or aortic, project backwards into the blood-stream, exactly like the lip or rim employed by Bergeon.

But I think that the very success of this attempt to enlarge the range of conditions to which Chauveau's narrow theory would limit the production of a cardiac *bruit de souffle*, shows how cautious we ought to be in assuming that we are now perfectly acquainted with all these conditions. In expressing my belief that *vascular* murmurs have always such an origin as Chauveau supposes, I am mainly influenced by the consideration that the circulation of a stream of fluid through a tube is a very simple physical matter, the phenomena of which have been thoroughly studied experimentally. But it is far otherwise in the case of the heart. In the left ventricle we have a contracting chamber, with projections of various kinds from its inner surface. During its systole, in particular, the mitral valve with its tendons and columns must tend to project into its cavity, with a space between it and the posterior wall of the ventricle. Under normal conditions the chamber empties itself completely during its systole, and this space can hardly be said to exist. No murmur is then generated. But let the ventricle be dilated, and let its contraction be imperfect and incomplete—as we must necessarily suppose it to be, if the quantity of blood poured into the aorta be not greater than in health, and if there be no mitral regurgitation (of which there is certainly in many cases no evidence). Is it not very probable that under such circumstances the blood in the space behind the mitral valve may be thrown into vibrations, and so a *bruit de souffle* be generated, exactly as in the *cul de sac* employed in Bergeon's experiments? Such a bruit would be heard at the heart's apex, and nowhere else. We shall hereafter see that precisely such a bruit is very frequently heard, in various diseases, and that its interpretation is still open to very great doubt.

Now, cardiac murmurs, instead of being soft and blowing, are sometimes very rough and harsh. The older French auscultators laid stress on such varieties, and gave them special names, as "*bruit de râpe*," "*bruit de scie*," "*bruit de étrille*," devoting great pains to the determination of their precise physical causes. Little success, however, appears to have attended their efforts: as might indeed be expected from the erroneous views that they entertained concerning the origin of murmurs in general. The rough and harsh murmurs in question are very generally accompanied with a thrill that can be felt if the hand be placed on the surface of the body at the spot where the murmur is audible: and to this Laennec gave the name of *frémissement cataire*. Now, according to Bergeon, murmurs are rough and attended with frémissement, when they are intense, and when the tube (he is speaking of simple physical experiments) is thin and elastic. It might, therefore, be thought that such murmurs owe their peculiar quality to the fact that the walls of the orifice take part in their production, and that they are not produced by the vibrations of the fluid alone. Such a view, however, is entirely incon-

[1] "Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques," art. Auscultation.

[2] "Des Causes et du Méchanisme du Bruit de Souffle," Paris, 1868, p. 103. In this essay will be found a detailed investigation into the physical cause of cardiac and vascular bruits.

sistent with Savart's experiments already referred to. And clinical facts are equally adverse to it. As we shall presently see, no murmur is so generally harsh, and so commonly attended with thrill, as the so-called presystolic murmur of mitral stenosis. But in this affection, the margin of the orifice, far from being thin and elastic, is almost always thick and hard, and often contains much calcareous matter. The peculiar quality of the murmur in this case is evidently not due to the fact that the orifice itself, as well as the fluid, vibrates. What, then, is its cause? There can, I think, be hardly any doubt that it depends upon the circumstance that the jet of blood in which the murmur is generated, entering the flaccid empty ventricle, impinges on its inner surface at a point which must be very close indeed to the part of the ventricle which strikes the chest-wall and produces the heart's impulse. The physician may thus almost be said to receive with his finger the full shock of the sonorous jet propelled into the left ventricle through the narrowed mitral orifice. It would be interesting to determine whether similar conditions are traceable in other cases in which similar murmurs occur: for instance, in cases of aneurism. For the present it must, I think, be concluded that the harsh rasping quality of a bruit, and the accompanying thrill, are not due to any peculiar state of the orifice at which the bruit is produced, but rather to the intensity of the murmur itself, and to the fact that the jet of blood which generates it is directed towards the surface of the patient's body.

Another modification of murmurs is that in which they are high-pitched and resemble the note of a musical instrument, or a whistle, the cooing of a dove, the puling of a chicken, or the mewing of a cat. These are generally spoken of as "*musical*" murmurs; and according to Bergeon, they may arise in either of two ways. Sometimes they are due to the fact that the channel into which the *veine fluide* passes is not straight but bent, so that the *veine* impinges on its wall on one side. This is the case, for instance, in the jugular fossa at the base of the skull; where (according to this writer) a musical bruit is often generated, which gives rise to an intolerable singing in the ears. More frequently such a bruit is due to the presence of a thin membranous flap or valve, vibrating in the stream of blood which flows over its surface; the musical character of some cardiac murmurs appears generally to be due to something of this kind. But the subject is one still admitting of further elucidation. In vol. vi. of the Pathological Transactions, Dr. Peacock has recorded a case in which a musical murmur, exactly resembling the sound of a cuckoo-clock, was audible at the distance of some feet from the patient: after death no special morbid appearance was discoverable in explanation of it.

But the differences in the *quality* of cardiac murmurs, which we have hitherto been considering, are of trifling consequence (so far as the interpretation of their cause is concerned) in comparison with two other points, to which we must now turn our attention. The first of these is their *rhythm*, or relation to the movements and natural sounds of the heart; the second their *seat*, or capability of being heard at different parts of the surface of the chest.

The passage of the blood through the heart and arteries is effected by three successive movements, each of which may, under certain circumstances, cause a bruit. (1) The most important of these is the ventricular systole: and since the contraction of one or other ventricle is invariably the cause of any murmur that coincides with it in time, such murmurs are very fitly termed *systolic* (or, sometimes, *ventricular-systolic*). They, of course, take the place of, or follow, the first sound: they coincide with the closure of the auriculo-ventricular valves, or at least occur when these ought to close. (2) After the ventricular systole comes the elastic recoil of the aorta and pulmonary artery. This, again, may generate a bruit, which coincides with (or replaces, or follows) the second sound, and occurs at the moment when the sigmoid valves should fall together. It would have been better that the name given to such a murmur should have indicated its origin: but no convenient title suggests itself, and since the ventricle is dilating at the time, the bruit in question has always been termed *diastolic*. This is unfortunate, for the ventricular diastole is only very indirectly concerned in its production, and may indeed have nothing at all to do with it. (3) Moreover there is a third movement, which likewise occurs during the ventricular diastole, and generates a third kind of bruit. This is the auricular systole. In health, it produces no sound; but in disease it may give rise to a very loud murmur: the best name for this would undoubtedly be that of *auricular-systolic* (proposed for it by Dr. Gairdner); but in practice it is generally called *presystolic*, because it more or less closely precedes the ventricular systole.

Thus it is usual to designate the rhythm of a bruit by indicating its relation to the contraction of the ventricles; a murmur that is synchronous with this contraction is called systolic: one that follows it is called diastolic; one that precedes it is called presystolic. Now, when the heart is beating slowly, it is generally easy to distinguish which of the cardiac sounds

or murmurs is systolic, from the fact that the pause before the first sound is very much longer than that which follows it. But when the pulsations are more rapid, this criterion is lost, for the increased pace is gained at the expense of the period of rest, and the one pause may then be as short as the other. The well-known difference in quality between the first sound and the second may then enable the rhythm to be detected; but this again often fails; and one is driven to determine the ventricular systole by noting at what period the heart's apex strikes the chest or (which is to me more easy) by feeling the carotid pulse with the finger while one is listening to the heart.

A systolic sound or murmur having been thus identified, it remains to consider whether any other bruit that may be audible is diastolic or presystolic. And here, again, all depends on the rate of the heart's beats. When these are infrequent, and the diastolic pause is prolonged, the so-called *diastolic* murmur, occurring at the commencement of this pause, is easily differentiated from the *presystolic* murmur that occupies its termination, and runs up to the following ventricular systole. But it is quite another case when the heart's action is rapid, and the pause proportionately shortened. The distinction between a presystolic and a diastolic murmur may then, as I believe, become quite artificial, so far as their mere rhythm is concerned. But there still remain differences of quality and seat, which usually enable the nature of the murmur to be determined without much difficulty.

We will now consider the three kinds of bruits in the order of their occurrence: I. the Presystolic; II. the Systolic; III. the Diastolic. And since each of these may be developed on either the right or the left side of the heart, it will be necessary to mention two varieties of each. But, as has already been stated, disease of the left valves is greatly more common than of the right.

I. A presystolic murmur, due to the auricular systole, is never produced unless the auriculo-ventricular orifice is narrowed. And practically it is almost always indicative of that chronic change in the corresponding valve that has been described under the name of stenosis.[1]

a. When developed at the mitral orifice, this murmur is much louder at the heart's apex than anywhere else. It is also remarkably local, being sometimes audible only at a single spot, and not being traceable round the side of the chest towards

[Fig. 99.

(Gairdner.)]

the left scapula, as is the case with the systolic murmur of mitral regurgitation.

The quality of a presystolic, or (as it is sometimes called) "*direct*," mitral murmur is in most cases peculiarly harsh, and it is often accompanied by a thrill perceptible to the touch. It is generally spoken of as having a "churning" or "grinding" character; and this may enable a practised ear to distinguish it at once from other bruits. I think I have never yet heard a direct mitral murmur which has been soft or musical. There is, however, an important modification of the presystolic murmur, which, I believe, I first described in a paper on this subject in the Guy's Hospital Reports for 1870–71. Such a murmur is often very short; and it may be so short as to resemble a tone, and thus to be hardly distinguishable from the natural first sound of the heart. Now, it happens that in cases of this kind the real first sound is commonly peculiarly sharp and clear, and so resembles the second sound; while the second sound is itself inaudible at the heart's apex. Thus the sounds heard at this spot may at first appear to be normal; while on closer examination it may be discovered that their rhythm is entirely different from that of the healthy sounds; and that one of them is in fact an abbreviated presystolic bruit.

[1] It is indeed possible that a mass of vegetations, formed upon the surface of the valve during acute disease, might so obstruct the channel as to lead to the development of such a murmur; but (so far as I am aware) no case of the kind has as yet been placed on record. I have always believed hypertrophy of the auricle to play an important part in the development of a presystolic murmur; and this implies the existence of chronic disease.

In the paper above referred to, I have described a case in which this observation led to the confident assertion that mitral stenosis existed in the case of a woman who had no other sign or symptom of cardiac disease, having been admitted into a surgical ward for gangrene of the leg. She died six weeks later; and the mitral orifice would admit only one finger-point.

It is only within the last few years that presystolic murmurs have been rightly interpreted. The name was invented by Gendrin.[1] He did not, however, attach any special importance or diagnostic value to such murmurs. But in 1843, Fauvel communicated to the *Archives Générales* a paper in which he showed by the narration of four cases (three of them fatal) that a presystolic murmur was indicative of mitral stenosis. Subsequent French writers, however, have thrown very little light on this subject. For many years the Paris School of Medicine was divided into two camps with regard to the rhythm of the heart's impulse, which Beau would have to be synchronous with the ventricular diastole. Agreement on minor points was therefore out of the question; and Hérard,[2] Bouillaud,[3] and Durosiez,[4] may be mentioned as having written on the subject of mitral stenosis, and expressed views opposed to those of Fauvel. Durosiez, in 1862, thought it sufficient to make a passing reference to "ce fameux bruit présystolique, dont tout le monde a parlé, sur lequel personne ne s'entend, que Hope lui-même avoue n'avoir jamais entendu, que M. Bouillaud enfin neglige et même nie." Racle, again, in his "Traité de Diagnostic médical," published in 1859, speaks of it as "une distinction plus subtile que réelle."

In Great Britain the first writer who alluded to this subject was, I believe, Dr. Gairdner of Glasgow, who expressed views precisely similar to those of Fauvel, except that he preferred to term the murmur auricular-systolic, rather than presystolic. Subsequently papers on the same subject were published by Dr. Wilks, Dr. Gull, Dr. Hayden (of Dublin), Dr. Peacock, Dr. Sutton, Dr. Simpson (of Manchester), and Dr. Hyde Salter.[5]

[1] Leçons sur les Maladies du Cœur, &c., 1841–42.

[2] Arch. génér. de Méd., sér. v. tom. ii. p. 543. 1853.

[3] Traité clinique des Maladies du Cœur, 1836.

[4] Arch. génér. de Méd., sér. v. tom. xx. p. 385.

[5] Edinburgh Medical Journal, vol. vii. part 1, p. 438. 1861.

[5] In my paper in the Guy's Hospital Reports, I have gone into the literature of this question in much greater detail than is possible here.

Thus in my communication to the Guy's Hospital Reports I was able to refer to twenty-eight cases (seven contributed by myself), in each of which a post-mortem examination proved the existence of mitral stenosis, and in which this condition had been diagnosed from a presystolic murmur heard during life. Since then the subject has been taken up by Dr. Douglas Powell and Dr. Silver. Even now, however, there are observers who deny that the rough grinding murmur heard in cases of mitral obstruction is really presystolic in rhythm. In the year 1872 Dr. Barclay contributed to the *Lancet*[1] a series of papers, in which he endeavored to prove that the peculiarity in the rhythm of this murmur really depends on the circumstance that the closure of the mitral valve is delayed. Instead of this closure occurring at the commencement of the ventricular systole, he believes it to take place only when the systole is nearly completed; the first sound being of course postponed likewise. Dr. Barclay thus regards the murmur as really regurgitant and not obstructive, although he does not deny its constant association with mitral stenosis. But it appears to me that no one who has studied the relation between the murmur and the heart's beat or the carotid pulse can admit that Dr. Barclay's hypothesis is tenable. Neither beat nor pulse can be felt while the bruit is audible; they both follow it.

It is important here to mention that the presystolic bruit by no means always merges gradually into the heart's first sound, as would appear from the accounts given of it by some writers. Much more often it is separated from the first sound by a distinct interval which seems to me sometimes as long as that which separates the natural first from the second sound. The murmur, too, is often prolonged through a period much exceeding that of the natural auricular systole. This has been explained in two different ways. The late Dr. Salter supposed that the first part of the murmur is generated while blood is flowing passively from the auricle into the ventricle. I have argued that the auricle begins to contract earlier, and goes on contracting longer, than in the healthy heart, and that the whole of the bruit is thus due to the auricular systole. This view has since been established by the cardiographic observations of Mr. Mahomed.[2] I append copies of two of his tracings, taken from the heart's apex in the same patient at an interval of seven months. It will be observed that the slight elevation which Marey proved to be due to the auricular contraction takes

[1] Vol. i. pp. 283 et seq.

[2] Med. Times and Gaz., 1872, vol. i p. 569.

place very soon after the preceding ventricular systole, and is succeeded by a gradually ascending line, throughout the whole duration of which the auricular systole is sustained. The figures seem to speak for themselves: and unless it can be shown that their peculiarities are capable of some different interpretation, it appears to me that they not only establish the point now under consideration, but also give the *coup de grâce* to Dr. Barclay's hypothesis.

Fig. 100.

Fig. 101.

Cardiographic tracings.

It is the more necessary to insist on the fact that the presystolic murmur is often separated from the following first sound of the heart by a distinct interval, because I believe that this fact has had much to do with the impression that so long prevailed as to the real rhythm of such murmurs. The old view was that the murmur caused by mitral obstruction should be diastolic in rhythm; and with the single exception of Dr. Markham all writers were agreed that diastolic apex murmurs were very rare. Evidently, therefore, those observers mistook for systolic the murmurs which they heard: and collateral evidence of this is further afforded by the fact that they described as systolic the *frémissement* which we know to go with the murmur. Nor did the mistake end here. I have shown in my paper that the real first sound of the heart at the apex was mistaken for the second sound, which it resembles so closely in character. It might appear needless to discuss the errors of a bygone period. But a little experience in clinical teaching shows that these very errors are still committed by every student, who has not had his attention specially drawn to them. And it appears to me that some of the most recent German writers have not yet extricated themselves from the same pitfall. Dr. P. Niemeyer, of Magdeburg, in an elaborate work on "Percussion and Auscultation," published in 1870, gives as diagnostic of mitral stenosis "a loud long systolic apex murmur and strong *frémissement cataire;* in rare cases, also, a short diastolic murmur." But Traube, Felix von Niemeyer (of Tübingen), and Friederich, describe the direct mitral murmur as presystolic.

I have already remarked that presystolic murmurs are often of long duration, and thus commence very soon after the second sound has completed the previous cardiac movement. It must be added that when the heart-sounds are traced downwards from the base, these murmurs have sometimes an apparent relation to the second sound, which is very apt to mislead the student, and which I cannot altogether explain. At the base, the second sound is clear and single; lower down, it appears to be reduplicated; still lower, the presystolic murmur seems to grow out of it. In my paper in the Guy's Hospital Reports I have discussed this subject at some length, and quoted the statements of Hamernyk, Drasche, and Guttmann, in regard to it. Here I must limit myself to a simple statement of the fact.

An objection frequently made to the view that these long murmurs are due to a prolonged auricular systole—and indeed to the view that they are due in any way to mitral obstruction—is, that since the pulmonary veins are unprovided with valves, blood would be forced back into them during the whole duration of the auricular systole, and the circulation through the lungs would be brought to a standstill. But it is forgotten that, in cases of mitral stenosis, the tension in the pulmonary vessels is very high—much higher than under normal conditions; whereas the left ventricle is in the condition of an empty flaccid sac, and thus readily receives the blood expelled by the contraction of the auricle. This objection, therefore, appears to have but little weight.

b. When developed at the tricuspid orifice, and due to stenosis of the corresponding valve, a presystolic murmur is heard, according to Dr. Hayden,[1] principally over the fifth left costal cartilage, and the fourth intercostal space, close to the sternum. Dr. Hayden has lately recorded a case of this kind, in which, between the area over which the tricuspid presystolic murmur was audible, and that over which a coexistent mitral presystolic murmur was audible, there was a space in which neither could be distinctly heard. Both lesions, therefore, were diagnosed; and

[1] Dublin Journ. of Med. Science, May, 1874.

after death the right auriculo-ventricular orifice would admit only the point of the middle finger; and the left one was smaller still. The tricuspid murmur was even harsher in quality than the mitral one, and began earlier in the ventricular diastole. As far back as 1864, Dr. Haldane[1] related a similar case, in which the tricuspid orifice was found after death to admit only the point of the forefinger. But it must be added that a mitral presystolic murmur was at the same time audible; and the mitral was in fact much the narrower of the two valves. Indeed, although tricuspid stenosis in moderate degree is common enough when mitral stenosis is considerable or extreme, I am not aware that it is ever clinically met with apart from such an association.[2]

II. A systolic (ventricular-systolic) murmur may have various origins. As we shall presently see, it has not always anything to do with the valves. And when it is due to valvular disease or imperfection, it may be formed at any one of the orifices into either ventricle; namely, either the mitral, the aortic, the tricuspid, or the pulmonary. Evidently a mitral or tricuspid systolic murmur must be due to regurgitation: an aortic or pulmonary systolic murmur must be obstructive or direct. These four varieties of systolic murmurs may be in part distinguished by their seat.

a. A mitral systolic murmur is loudest at or near the heart's apex; that is, if the left ventricle be of normal size, about the fifth costal cartilage, and a little internal to the nipple; if the heart be enlarged, further downwards and outwards. It is not heard over the base of the heart, nor near the ensiform cartilage; or, if it can be heard there, it is much less loud than at the heart's apex. It can very generally be traced along the left ribs (or, to use a common expression, *into the axilla*), and is audible at the angle of the left scapula. The question will hereafter be discussed whether it is not invariably heard in these positions when of sufficient intensity.

b. An aortic systolic murmur is most plainly heard in the *second* right interspace, and is traceable over the ascending

[Fig. 102.

(Gairdner.)]

arch, that is, towards the inner end of the right clavicle; and often also along the arteries of the neck, or even of other parts of the body.

c. A tricuspid systolic murmur is heard over the ensiform cartilage, and sometimes to the right of it. It is also (according to Gairdner and Sutton[1]) heard over the surface of the right ventricle; that is to say, a little to the left of the sternum; but it "is little audible above the level of the third rib." I should myself have fixed its upper limit at a much lower point. In some rare cases it is very loud, and may then be heard over a wide area; but most commonly it is a faintly audible bruit; and I think it is then generally discoverable at one spot only. Indeed this appears a principal reason for its presence being often overlooked.

d. A pulmonary systolic murmur is loudest about the third left costal cartilage, and is transmitted upwards and to the left, towards the middle or inner end of the left clavicle.

The *clinical significance* of these four murmurs varies widely in different cases. They must, therefore, be discussed sepa-

[1] Ed. Med. Journ., vol. x. 1864, p. 271.

[2] An exception must be made for a very remarkable case which occurred to Dr. Gairdner, and in which a rounded tumor projected into the interior of the right auricle, in such a way that it formed a kind of ball-valve to the tricuspid orifice. In that case a tricuspid presystolic murmur was heard several years (I think, ten years) before death by Dr. Gairdner, who published his diagnosis in his work on Clinical Medicine. I am not aware that he has yet placed the result of the post-mortem examination formally on record. I saw the preparation of the heart, with the tumor, at the meeting of the British Medical Association in 1873. One remarkable feature about the specimen was that there was no marked hypertrophy of the right auricle. This certainly throws some doubt on the opinion which I have expressed in a note to p. 381.

[1] London Hosp. Reports, iv. 1867–68, p. 288.

rately; and it will be convenient to take the two *basic* murmurs first.

As we have seen, the pulmonary valves are scarcely liable to any disease beyond congenital malformation. In practice, therefore, a pulmonary systolic murmur, if due to change in the valves, almost always indicates a congenital defect, and needs no further discussion here. An aortic systolic murmur, on the other hand, is frequently caused by acquired stenosis of the orifice in question. But, as has already been stated, such stenosis is (far more constantly than is generally supposed) accompanied by regurgitation; and the systolic murmur, therefore, is followed by one which is diastolic.

A systolic murmur, however, audible at the base, and traceable along the aorta, is by no means limited to cases in which there is actual stenosis. Formerly it was held that any roughening of the orifice, or of its valves, or even of the lining membrane of the vessel, would suffice to generate it. But even then it was recognized that such a murmur was frequently heard under various conditions, when after death no morbid change in any of these parts was discoverable. This led to the theory that the murmur was due to an altered state of blood; at first, that an anæmic state only could produce it; but afterwards, that various changes in the composition of the blood might generate it. I have already, in discussing the physical theory of murmurs, mentioned the ingenious explanation given by Chauveau of some of the more striking of these anæmic murmurs, as they have been called. This explanation, indeed, hardly covers the whole range of the bruits that have been regarded as hæmic, in the wider sense of the term. And it must be admitted that the precise significance of many basic murmurs has still to be determined. It is important to note that many undoubtedly anæmic murmurs appear to be seated rather in the pulmonary artery than in the aorta; and that they are sometimes of a harsh quality, such as might *à priori* have been supposed to belong rather to murmurs due to some very definite organic cause.

It must be added that the so-called hæmic murmurs are believed to arise in many acute diseases, including not only fevers, but also those affections in which endocarditis is apt to occur, as, for instance, acute rheumatism. In this disease there is a further ground for uncertainty as to the cause of a basic murmur, in the fact that a similar sound may probably be caused by the presence of lymph in small quantity outside the heart, round the bases of the great vessels.

In this connection I must not omit to mention the fact that in children (even when in good health) a murmur over the pulmonary valves may be generated by the pressure of the stethoscope, as is shown by the fact that it disappears when the instrument is lightly applied. It is said that a similar murmur has sometimes been observed even in adults, when the chest-walls are thin and yielding. And consolidation of the anterior edge of the left lung appears sometimes to cause pressure on the trunk of the pulmonary artery, and consequently a systolic murmur. Yet another suggestion with regard to these basic pulmonary murmurs has recently been made by Quincke; and Dr. Balfour[1] has adopted it. It is that they sometimes depend upon the edge of the left lung being retracted, in consequence of which the heart, during its systole, compresses the pulmonary artery against the parietes of the thorax, instead of merely pushing aside this edge of the lung. In support of this it is asserted that the murmur disappears when the diminution of the cardiac dulness shows that the lung has recovered its normal dimensions. Dr. Balfour even relates a case in which the murmur ceased whenever the patient inspired deeply and held his breath. But I must confess that I see little probability in this explanation.

This is perhaps the most convenient place for noticing the suggestion of another German writer (Naunyn), which is also quoted with approval by Dr. Balfour; namely, that the systolic murmur of mitral regurgitation is sometimes heard an inch or two to the left of the sternum, between the second and third ribs. The seat of such a murmur is supposed to be in the appendix of the left auricle. I must confess that when I read Naunyn's paper on the subject I thought there must be some mistake: and this suspicion is not removed by Dr. Balfour's remarks on the subject, for I find him saying that this remarkable modification of the mitral regurgitant murmur is almost invariably present when the insufficiency is dependent upon anæmia and chlorosis!

Passing on to consider the clinical significance of *apical* systolic murmurs, we may take first that which is audible near the ensiform cartilage, and which is referred to the tricuspid valve; and of this murmur the interpretation is seldom difficult. According to universal belief, it it always due to regurgitation through the tricuspid orifice. In some cases this is the result of primary disease of the valve itself, which (as we shall see further on) is occasionally affected with an acute ulcerative change. In such cases a bruit would doubtless be heard by any physician sufficiently acute to search for it. Most commonly, however, there is no actual change in the valve itself; its seg-

[1] Med. Times and Gaz., 1874, ii. p. 556.

ments are kept apart by the dilatation and distension of the right ventricle, while at the same time its orifice is greatly widened. The distension of the ventricle may result either from disease of the valves on the left side of the heart, or from some chronic affection of the lungs, such as emphysema or fibroid disease. The cases in which I have heard the loudest tricuspid regurgitant murmurs have been those in which there was cirrhosis of one lung. Two good examples of this are recorded in Dr. Bastian's table in the second volume of this work (cases vi. and xiii.). I well remember the second of these cases, which occurred in the practice of the late Dr. Addison, when I was his clinical clerk. The murmur was so loud that it was heard over the heart's apex, as well as over the ensiform cartilage; and Dr. Addison, although repeatedly pressed upon the point, would not admit that the case was other than one of primary mitral regurgitation. Indeed, as Dr. Wilks has pointed out, cases of cirrhosis of the lung are often so like those of primary heart disease in their general aspect and symptoms as to be mistaken for examples of such disease.

There might, indeed, well be the same uncertainty about the theoretical significance of tricuspid, that we shall see to prevail in regard to the corresponding mitral, murmurs. But in practice such doubts have not arisen, since tricuspid systolic murmurs are not very often heard, and they are perhaps never heard unless those conditions of obstructed pulmonary circulation are present which most physiologists regard as readily capable of inducing regurgitation through the orifice in question. Physicians, therefore, have been more disposed to admit the occurrence of regurgitation when murmur is absent than to doubt its existence when murmur is present.

It is very different with those systolic murmurs which are audible at the apex of the heart, and which (if of valvular origin at all) must be referred to the *mitral* orifice. They are perhaps the commonest of all murmurs, and their significance is the most uncertain.

There are, in the first place, certain sounds which an inexperienced auscultator may easily mistake for endocardial murmurs, but which really arise not in the heart, but in that little flap or tongue-like process of the lung which commonly projects forwards over the apex of the heart, just below the seat of its visible impulse. The contraction of the ventricle, altering the form of the heart, causes a movement of air into or out[1] of this portion of the lung, and thus produces a murmur which, though of respiratory origin, is distinctly systolic in rhythm. The sound in question is generally soft and blowing; but I have several times known it to be of distinctly musical quality. Its most important peculiarity is that it is not constant, but accompanies only those beats of the heart which occur at a particular period of the respiratory act, this period being generally that of inspiration. Thus, when the patient breathes out, the first sound may be quite natural; but when he draws in his breath, a systolic murmur may be audible, which acquires its maximum intensity when the cardiac beat happens to coincide with the acme of the inspiratory effort. When the patient is made to hold his breath, the murmur in question is often, but not always, suppressed for the time.

A little care, however, excludes this source of fallacy. If the murmur be heard uniformly with every ventricular systole without exception, we may conclude that it arises within the heart itself.[1] And the same conclusion may also be arrived at, even when the murmur fails to accompany certain beats, provided that its absence depends not upon any relation to the respiratory rhythm, but upon the circumstance that the corresponding heart-beats are feeble and imperfect. How, then, is such a systolic apex murmur produced?

Now, if after death some of the tendinous cords of the mitral valve be found softened and ulcerated through by disease —or if the edge of the valve have become turned inwards towards the auricle—or even if the orifice be so thick and hard that it obviously must have remained patulous: if any one of these conditions should be present, we may be sure that regurgitation occurred during life, and we have good grounds for inferring that any systolic apex murmur that may have been audible was due to regurgitation.

The conditions just mentioned are, however, comparatively seldom met with. But it is to be observed, that, if we exclude these conditions, we can never with certainty determine, when we are examining the heart after death, whether the mitral valve was or was not competent. We have no means of testing satisfactorily the action of the valve. We may, indeed,

[1] According to certain modern views on the theory of the respiratory murmur (of which a full account is to be found in the Med.-Chir. Review for July, 1873), a sound within the lung can be generated only by the entrance of air into that portion of lung, and not by its exit. We must therefore suppose that, when the heart assumes a globular form during its systole, air is sucked into the flap of lung in question.

[1] Evidence is, I think, wanting to show that a white patch on the serous surface of the apex, or any like condition, can generate the murmur in question.

tie the base of the aorta; and having cut open the apex of the left ventricle, may hold the heart upside down, and pour water into the cavity to see whether it runs out. But in such an experiment the conditions are very different from those which obtained during life. Then, the base of the muscular columns was moved towards the orifice by the ventricular contraction: while those columns at the same time underwent shortening, so as to keep the tendinous cords stretched to the proper degree. Now, ventricular wall and fleshy columns are alike relaxed. Errors may thus arise in either direction. When the muscular columns are converted into non-contractile fibrous tissue, the valve may have been very imperfect in the living body, and yet may close well enough when tested after death. Conversely, when the ventricle is dilated, without the tendinous chordæ being increased in length, it may happen that the valve allows reflux to occur after death, although it had before been efficient.

This deficiency in the proof of mitral regurgitation, when a case has reached the dead-house, would be of but little consequence, if the orifice or its valve were constantly found to be obviously diseased in those cases in which a systolic apex murmur had been heard during life. But all who have worked at the subject know that this is not so. To quote the words of Dr. Bristowe,[1] "In a large proportion" of such cases "the mitral valve and the orifice it protects are found to present a perfectly healthy appearance." Now Dr. Bristowe has proposed a very ingenious solution of the difficulty. He believes that valvular incompetence exists whenever a systolic apex murmur is heard; and in the case now under consideration he attributes this incompetence to "disproportion between the size of the ventricular cavities and the length of the chordæ tendineæ and musculi papillares." He has shown in fact that while the former are found after death to be dilated, the latter are often small and seem to be on the stretch. But these observations are exposed to the full force of the objections already made to the post-mortem evidence of mitral regurgitation. The appearance of the mitral cords and columns in a dilated ventricle relaxed by death can surely afford no proof that these parts were too short to allow the valve to close, when the ventricle itself was shortened by its own systole.

Dr. Bristowe regards it "as an axiom, that the existence of a systolic murmur at the apex of the heart is a sure indication of incompetence of one or other of the auriculo-ventricular valves." And he deems it unnecessary to offer any evidence in support of this position, beyond the fact that in all the cases recorded in his paper the general symptoms and the condition of internal organs (lungs, liver, spleen, &c.) were such as are found in this form of disease. Subsequent writers, however, have dealt with this question in a different way. Both Dr. Austin Flint[1] and Dr. Andrew[2] have expressed the opinion that the murmur by no means necessarily indicates such regurgitation: according to the latter observer, indeed, regurgitation is absent in 34 per cent. of the cases in which the murmur is audible.

These authorities believe that there are two criteria which may be applied to the determination of the fact, that in a particular case a systolic apex murmur is really due to mitral regurgitation. The criteria are: 1. That the murmur should be audible in the left side of the back, about the inferior angle of the scapula; 2. That the pulmonary second sound should be intensified.

1. A good illustration of the fact that the murmur caused by mitral regurgitation is heard in the left side of the back is afforded by cases in which the tendinous cords are ruptured or ulcerated through. It has been so in the cases which I have seen, and I have not met with any recorded instance to the contrary. But in such cases the murmur is generally loud, and the amount of regurgitation probably large. I am not sure that when the murmur is feeble one can fairly expect that it should always be carried backwards: for one must remember that, though the direction of the blood-stream is towards the vertebral column, the auricle is not itself in any close relation with the part of the chest-wall at which one looks for the murmur. Consequently, although I am prepared to admit that whenever a systolic murmur is heard in the back, it is caused by mitral regurgitation, I cannot regard the fact that a feeble murmur is not heard in that position as conclusive against its being so caused.

2. Intensification of the pulmonary second sound (that is, of the second sound heard at the second left, as compared with the second right costal cartilage[3]) is undoubtedly present in many of the most marked cases of mitral regurgitant dis-

[1] Med.-Chir. Review, 1861, July, p. 215.

[1] Am. Med. Times, 1862. Quoted in Braithwaite's Retrospect, xlvii. 1863, p. 69.

[2] St. Bartholomew's Hospital Reports, 1865, i. p. 13.

[3] Dr. Andrew has shown that it is necessary, in instituting this comparison, to remember that the same difference may be due to enfeeblement of the aortic second sound, while the pulmonary second sound is natural; and also that an emphysematous lung overlapping the heart on one side may modify the intensity of the sound.

ease. Its cause is evidently the increased tension of the blood within the pulmonary system of vessels. But this (as I shall endeavor to show further on) may arise from any cause which prevents the left side of the heart from emptying itself. I cannot see, therefore, how intensification of the second sound can be indicative of regurgitation through the mitral orifice, rather than of other conditions which will then be mentioned. Moreover, I believe that intensification of the pulmonary second sound requires, as a condition of its occurrence, that the right ventricle should be powerful, and that the tricuspid valve should be efficient. I think I have observed that this sign is present chiefly in the early stages of mitral regurgitant disease, before it has begun to tell upon more distant parts.

My own views with regard to the interpretation of systolic apex murmurs may therefore be summed up as follows :—

1. If such a murmur be audible in the back, it indicates mitral regurgitation.

2. If such a murmur be heard only at the heart's apex, we are unable at the present time to pronounce any positive opinion as to its cause. Should the murmur be loud, we may probably conclude that it is not due to mitral regurgitation: since really regurgitant murmurs, when loud, are, perhaps, always audible in the back, though for slight murmurs the same statement may not be tenable.

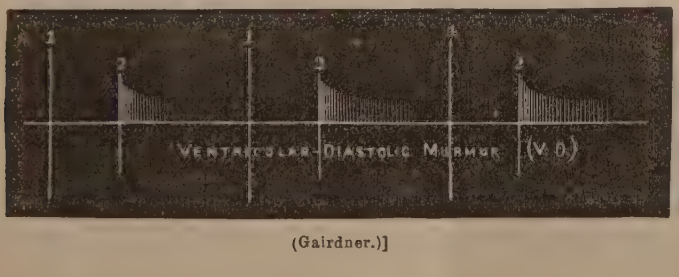
The question still remains, How is a systolic apex-murmur produced when it is not caused by mitral regurgitation? I have already (vide p. 724) suggested that it may be due simply to dilatation of the left ventricle, as was long ago supposed by many of the earlier writers on auscultation.

III. A "diastolic" murmur, as has been stated, accompanies the elastic recoil of the aorta and pulmonary artery. It almost invariably indicates regurgitation, through the space that should be closed by one or other set of sigmoid valves into the ventricle; and in the immense majority of cases the valves affected are those of the aorta.

The quality of the diastolic murmur of aortic regurgitation varies greatly in different cases; it may be soft and blowing, rough, and attended with thrill, or even musical. It may be so loud as to be audible at some little distance from the patient; or so slight as to require the utmost vigilance for its defection.

The seat of this murmur is somewhat variable. As a rule, it is very plainly audible over the base of the heart; its point of maximum intensity is generally stated to be at the sternal end of the second right costal cartilage, or in the second right interspace; and it is carried downwards along the length of the sternum (apparently in consequence of the fact that osseous substance is a good conductor of sound), so that it may often be loudly heard near the ensiform cartilage. This fact has been especially insisted on by Dr. Gairdner.[1] Again, this murmur is frequently plainly audible at the heart's apex, and sometimes it is louder there

[Fig. 103.



(Gairdner.)]

than at the base. Lastly, it may be conducted along the arteries, sometimes, to a surprising distance: according to Dr. Gee, as far as the radial arteries.

In discussing the theory of murmurs in general, I have pointed out the conditions upon which some of the varieties in the seat of this bruit appear to depend (see p. 724). If the views there stated are correct, the fact that in a particular case an aortic diastolic murmur is transmitted upwards along the aorta may be interpreted as indicating that the valves are so free from serious damage that, although they do not meet, they nevertheless project inwards into the aorta to a greater or less extent; while in those cases in which the murmur is solely carried downwards it may be concluded that the valves are more completely destroyed.

A further refinement in regard to diastolic murmurs has lately been suggested by Dr. Balthazar Foster[2] of Birmingham. He believes that when such a murmur is heard at the apex of the heart it is due to incompetency of the left aortic segment,

[1] Clinical Medicine, p. 587.

[2] Med. Times and Gazette, 1873, ii. pp. 658, 686.

so that the regurgitant blood-stream falls upon the mitral curtain and is carried downwards; and, on the other hand, that a similar murmur propagated towards the ensiform cartilage indicates defect in the right and posterior segments, by which the blood is thrown upon the septum. He alludes to cases corroborative of his views, to which he further attaches considerable importance as regards prognosis. He thinks that incompetency of an aortic segment must specially interfere with the flow of blood into the coronary artery contained within the corresponding sinus (which flow he, in common with many other authorities, believes to occur during the recoil of the aorta), and so must tend to impair the nutrition of the heart. Now the left aortic segment has no coronary artery in relation with it. Dr. Foster therefore infers that, cæteris paribus, life is more likely to be prolonged when this segment is affected, or (in other words) when the murmur is audible at the apex. But my belief has hitherto been that the murmur is propagated in this direction especially when the regurgitant stream is large: and if so, one would suppose that the prognosis must be particularly unfavorable. I think that the point is one which needs further observations.

It has been stated that in the immense majority of cases a regurgitant murmur has its seat at the aortic orifice. In fact a pulmonary regurgitant bruit is so rare as scarcely to need consideration. In 1865 Dr. Wilks exhibited to the Pathological Society[1] a specimen of disease of the valves in question, in which a double bruit had been heard during life: and one or two other cases are recorded in medical literature. In Dr. Wilks's case the question of disease of the pulmonary artery was considered during the patient's life, for the pulse gave no indication of aortic regurgitation, and the bruit became less marked towards the right, and in the course of the aorta, but was equally distinct, or even somewhat more intense, towards the left, clavicle.

[Fig. 104.

Auricular systolic and ventricular systolic murmurs combined. (Gairdner.)]

[1] Path. Trans. xvi. p. 74.

But the great rarity of such disease led to its rejection as a diagnosis. Indeed, one can hardly expect in future to attain to greater accuracy: for (as we shall presently see) the pulse may fail to be characteristic of aortic regurgitation even when this disease exists; and the tendency of aortic diastolic murmurs to be transmitted downwards along the sternum must always prevent a pulmonary regurgitant murmur from being identified by its being heard over the right ventricle. Still, acquired disease of the pulmonary valves is so exceedingly rare (and in congenital disease I do not know that marked regurgitation ever occurs), that one hardly needs to make a reservation on account of it in attributing diastolic murmur to aortic regurgitation. The real necessity for reservation lies in the fact that aortic aneurism sometimes causes such a murmur, probably because it receives blood during the elastic recoil of the aorta, as well as during the ventricular systole. It is only when an aneurism arises from the commencement of the arch that its murmur could be mistaken for one of regurgitation through the valves: and even then the former would perhaps never be transmitted to the heart's apex, as is so generally the case with the latter. Very frequently, indeed, the two conditions are combined.

Another infinitely rare condition, in which a diastolic murmur, not due to regurgitation through the aortic valves may be heard at the base of the heart, is that in which the aorta communicates with the pulmonary artery, either by a patent ductus arteriosus, or through an aneurismal sac. Of the former affection I have recorded a remarkable instance.[1] The murmur (which was in part musical) was audible at the second left costal cartilage, and was transmitted to the left along this cartilage, but not downwards along the sternum. It was not everywhere continuous with the second sound. It had a wavy character, quite unlike anything that I had ever heard before. It was clearly distinguished (during the patient's lifetime) from an aortic regurgitant murmur; and it was thought not unlikely to be due to an opening from the aorta into the pulmonary artery. A case in which an aortic aneurism was correctly diagnosed to open into the pulmonary artery has been related by Dr. Wade,[2] of Birmingham. The diastolic murmur was prolonged, and of a hissing character with distinct purring tremor. It was audible over the cartilage of the fourth left rib, and in the neck, back, and upper part of the chest.

With these exceptions, a diastolic mur-

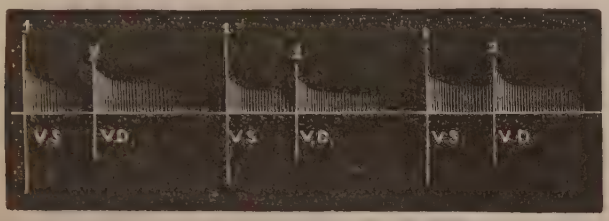
[1] Guy's Hospital Reports, 1872–73, series iii. vol. xviii. p. 23.

[2] Med.-Chir. Trans. vol. xliv.

mur (as I believe) invariably indicates regurgitation through the aortic orifice into the left ventricle.

It may be expected that something should be said as to the not infrequent coexistence of two or more of these murmurs in the same case. I have already remarked on the rarity of systolic murmurs indicating actual obstruction of the aortic orifice, unless a diastolic murmur be also present, and discoverable on careful examination. It may be added that in disease of the aortic valves the tendency is for regurgitation to follow obstruction. In the case of the mitral valves the opposite is observed. Commencing disease appears to produce a regurgitant murmur: and it is only as the orifice becomes more and more contracted that an obstructive murmur is heard. There is this further peculiarity, that when mitral stenosis causes a marked presystolic murmur, it rarely happens that any systolic murmur is at the same time audible. I have scarcely ever heard a systolic murmur in association with the rough grating bruit, attended with thrill, that is so characteristic of the more extreme degree of constriction of the mitral valve. A more or less distinctly double murmur at the apex is not, indeed, very uncommon: but in this case both portions of the mur-

[Fig. 105.



Ventricular-Systolic and Ventricular-Diastolic murmurs combined. (Gairdner.)]

mur are rather of a soft and blowing quality: and the inference probably is that the stenosis is moderate in degree.

With regard to the coexistence of murmurs developed at different orifices I have nothing particular to say. Their determination must be based on the principles which regulate the diagnosis of each murmur separately: guided, of course, by the known liability of particular valves to undergo simultaneous or consecutive changes.

The other effects of disease of the cardiac valves—those which affect the patient's health, and are consequently commonly called the *symptoms* of such disease—are divisible into three distinct classes.

I. We may take first a class of effects, which are of great importance, but which have only recently attracted notice, and probably do not yet receive a due share of attention. The valves of the heart are bathed on all sides by the circulating fluid. When they are inflamed or ulcerated, the blood flows directly over the diseased surface. When any portion of their substance, or of the products of inflammation, becomes disintegrated, the detached fragments necessarily pass into its stream. This is so obvious, that we may well be surprised to find that no one had recognized it until Dr. Kirkes pointed it out in the year 1852.[1] And as he showed, the phenomena attendant on this process are divisible into two distinct groups:—

(*a.*) *Embolism.*—In the first place, a mass of some size may be detached, which, passing into the arterial system, sooner or later reaches a vessel which it cannot traverse, and which it consequently plugs. The result is that the circulation is entirely arrested in the region supplied by the artery, unless indeed blood from collateral arteries enters the obstructed vessel beyond the seat of the obstruction. It might have been expected that the region in question would become anæmic. Recent observations, however, have shown that such is not the case. Prévost and Cotard,[1] and afterwards Lefeuvre,[2] have studied this question experimentally. They injected foreign bodies (especially the seeds of tobacco) upwards into the abdominal aorta of dogs, and exposed the kidneys and spleen by opening the abdomen, so as to make apparent the earliest effects of obstruction of the arteries of those viscera. They found that the regions supplied by the blocked arteries instantly become of a dark purple color, and in the spleen were distinctly raised above the level of the rest of the region. This

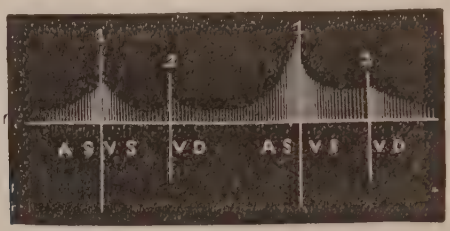
[1] Med.-Chir. Trans. xxxv. p. 281.

[1] Gaz. Med. 1866, p. 202.

[2] Étude physiologique et pathologique sur les Infarctus Viscéraux, Thèse de Paris, 1867. A review of these observations will be found in the Med.-Chir. Review for October, 1871, p. 368.

state of engorgement is believed to be due to a paralysis of the muscular coat of the vessels. They become unable to resist the pressure of the blood in the veins, which consequently flows back into the capillaries and arteries, and distends them up to the point of obstruction. Hemorrhage then takes place. After a time the effused blood and the elements of the tissue undergo fatty degeneration: and the affected part acquires a characteristic yellow color. This always extends to the surface of the organ, and penetrates more or less deeply towards its interior in the

[Fig. 106.



Auricular-Systolic, Ventricular-Systolic and Ventricular-Diastolic murmurs combined. (Gairdner.)]

form of a wedge or cone, which is generally surrounded by a red halo of congestion. Still later, absorption takes place: and in the end nothing is left beyond a deep fissure or puckering. It must be added that sometimes, instead of the whole mass undergoing fatty degeneration and conversion into the peculiar yellow matter, a part of it sloughs: in other cases it breaks down into pus.

The changes just described do not occur in all organs alike. They are especially well marked in the spleen and kidneys.[1] The reason appears to be that the branches of the splenic and renal arteries anastomose but little or (in the case of the splenic artery) not at all. In the liver, on the other hand, a true infarctus is, perhaps, never met with, apparently because its lobules do not derive their supply of blood entirely from a single source. The mesenteric arteries occasionally become the seat of embola. This occurred in one of Lefeuvre's experiments with tobacco seeds. The affected part became first pale and afterwards of a livid purple color. Embolism of a mesenteric artery has also sometimes been observed as a result of disease in the human subject. The cerebral arteries are very liable to embolism; this is believed to occur more frequently in the left middle cerebral than in any other artery, apparently because its course in some way favors the entrance of a detached mass. In the brain, the result of arterial plugging is generally white softening of the corresponding part of the brain; but sometimes a firm yellow infarctus is produced. When embolism occurs in one of the arteries of the extremities, the tendency is for the limb beyond the seat of obstruction to mortify. The gangrene is not then always of the dry variety, as was formerly taught. It may be moist, and attended with the formation of bullæ. This is doubtless preceded by an hyperæmia, like that which we have seen to follow plugging of an artery in the spleen or kidneys, except that as the veins of the limbs are provided with valves, the blood probably comes from the collateral arteries of the limb. In the arteries of the extremities, and indeed in all arteries, embola are especially apt to be arrested at those points where the vessel is dividing, or where a large branch is given off, so that the calibre of the channel is suddenly diminished. Thus in the upper limb, they are most commonly found in the axillary artery, and at the bifurcation of the brachial artery: in the lower limb, at the points of division of the common femoral and the popliteal arteries respectively. The left lower limb is decidedly more subject to embolism than the right: and by Virchow[1] this is atttibuted to the fact that the left common iliac artery comes from the abdominal aorta in a more direct line than the right. The peculiar wedge-shaped masses in the abdominal viscera appear to have been described independently by Hodgkin,[2] Cruveilhier, and Rokitansky. Their association with heart-disease was first noticed by the last-named observer, and has been admitted by all modern writers on morbid anatomy. It is, however, only within the last few years that they have been regarded as possessing any clinical interest, or that their formation has been supposed to be attended with any symptoms affecting the health of the patient. Following Kirkes,

[1] According to Sperling (Inaug. Diss., Berlin, 1872; London Med. Record, Jan. 1873), the kidney is more frequently the seat of embolism than the spleen, in the proportion of 75 to 51.

[1] Gesammelte Abhandlungen, p. 444.

[2] Med.-Chir. Trans. xxvi.

Virchow[1] is the writer to whom credit is especially due for having drawn attention to this subject: and recently several French memoirs and papers have been written on it, in which the affection is described as a special disease, under the title of "Ulcerative Endocarditis."

The clinical features observed in these cases are of two kinds. In the first place, there are the direct efforts of intercepted blood-supply to the part served by the obstructed vessel. Thus, as we have seen, a limb may mortify as the result of embolism of its main artery. Many of the cases of spontaneous gangrene in young subjects that come under the care of the surgeon are of this kind; and, with the stethoscope, the existence of disease of the valves of the heart may often be recognized without difficulty. It may be worth while to note that the embolism in these cases is not always derived from the diseased valve itself; sometimes it comes from the auricle or ventricle, having been one of the little rounded ante-mortem clots which are so apt to form in the heart's chambers behind any obstruction.[2]

Embolism of the cerebral arteries, again, may give rise to a great variety of symptoms, according as one or another part of the brain is deprived of its due supply of blood. The most frequent effect is the production of right hemiplegia, with or without aphasia. This corresponds with the fact that the left middle cerebral artery is especially apt to become plugged. Embolism of the retinal arteries leads to changes which can be studied with the ophthalmoscope.

It has already been stated that in the viscera, instead of the usual yellow wedge-shaped masses or infarctus being formed, suppuration, or even sloughing, sometimes occurs in the regions supplied by an artery that has become the seat of embolism. It is perhaps doubtful whether these changes ever in themselves produce any appreciable influence on the patient's health, or on the symptoms from which he suffers. But they may set up a peritonitis, and this will usually be attended with a great aggravation of his complaint, and even with danger to his life; and embolism of a mesenteric artery may cause severe enteritis, which may be quite capable of clinical recognition.

(*b.*) *Infection.*—But in almost all these cases the effects of the occurrence of embolism in particular arteries are complicated with, and probably overpowered by, those which depend upon a general contamination of the blood, as it passes over the surface of the diseased valve. This was clearly pointed out by Dr. Kirkes, in his classical paper already more than once referred to; and of late years many observers have worked at the subject, in the hope of explaining it more fully. So severe and rapidly fatal are some of these cases, that Virchow has given them the designation of *Endocarditis Maligna.*

A principal symptom in these cases is the presence of *fever.* The temperature is raised two or three degrees, or more, above the normal standard. Dr. Goodhart[1] mentions one case in which it was several times noted at 104°; and in a case which I recently examined it reached 105·8°. Not rarely there are repeated attacks of shivering: indeed, the illness is often ushered in by a sudden rigor. The pulse is quickened; the tongue is often dry. Extreme prostration, delirium, and somnolence, are occasionally present. According to Dr. Wilks, articular pains are often complained of. Vomiting and diarrhœa are common. The spleen is greatly enlarged, and is sometimes tender on pressure. The skin has an icteroid tinge, and there may even be jaundice, of which Lancereaux[2] has recorded several examples. Petechiæ may be present, or even distinct purpuric blotches.[3] Ecchymotic spots may also be found on the surface of the pleura and pericardium, and on the mucous membranes lining the larynx, stomach, intestines, and urinary bladder. The liver after death is found to be pale, supple, and flabby. The tissue of the spleen (which is many times larger than natural) is soft and pulpy.

When a patient is known to be suffering from disease of the cardiac valves, there is but little difficulty in assigning to their true cause the symptoms just enumerated. By carefully examining the heart several times at short intervals, one may be able to detect such variations in the physical signs as may demonstrate the fact that acute changes in the valves are going on. Charcot and Vulpian[4] mention one case in which the most marked signs of aortic insufficiency became prominently developed within a week.

But in many instances there is nothing to draw the physician's attention to the state of the valves; and the real nature of the case may then be easily overlooked. The valves may previously have been quite healthy. And since palpitation, præcordial pain, and oppression of the breathing may all be absent, there may

[1] Gesammelte Abhandlungen, pp. 636–729.

[2] Such an ante-mortem clot may, when a valve is stenosed, be the direct cause of sudden death: getting washed into the blood-current, it may completely occlude the narrowed orifice. See a case recorded by Dr. Van der Byl, Path. Trans. ix. p. 91.

[1] Guy's Hosp. Reports, xv. p. 415

[2] Gaz. Méd., 1862, p. 662.

[3] Path. Trans. xxi. p. 109.

[4] Gaz. Méd. 1862, p. 388.

be nothing to suggest the necessity of examining the heart. The case is thus very likely to be mistaken for one of enteric fever, or, if there be much shivering, of idiopathic pyæmia, or even ague; or again, if there be marked jaundice, for one of pylephlebitis. The relation to purulent infection has been especially insisted on by Dr. Wilks, and he has proposed to designate the affection an "arterial pyæmia."

In the previous paragraphs it has been taken for granted that the diseased valves are those on the left side of the heart, and that the phenomena of embolism or of infection therefore show themselves in the course of the distribution of the systemic arteries. However, when the tricuspid valve is diseased, or the pulmonary valves, precisely similar effects show themselves; but, of course, within the lungs. A striking case of this kind has been recorded by Charcot and Vulpian,[1] which was diagnosed during life. One flap of the tricuspid valve was softened and perforated, and presented numerous vegetations. The lungs contained scattered abscesses. Other instances have been related by Dr. Kirkes and Dr. Moxon.[2] Dr. Moxon's case occurred in a woman, within a month after her delivery.

The precise nature of the process of *Infection* in the cases under consideration has been much discussed of late years, and even now it has not been fully ascertained. In almost his earliest paper on the subject, Virchow related some experiments that he had made of injecting different substances into the jugular veins of dogs. And he proved that while portions of caoutchouc simply produced obstruction of branches of the pulmonary artery into which they were carried, animal substances (pieces of muscle, fibrin, &c.) set up severe inflammation of the corresponding tracts of lung tissue, leading to suppuration or even to sloughing. Hence he concluded that the phenomena of infection are not merely of *mechanical* origin, but must result from some *chemical* action. The same fact has since been insisted on by Feltz[3] of Strasburg, who maintains that solid elements by themselves never carry infection: this is always propagated by septic fluids. Another writer, Panum of Kie[4] endeavored to show that the immediate cause of irritant effects is the decomposition, *within the bloodvessels*, of the masses by which they are plugged. By Lancereaux, again, stress was laid on the opinion that the poisoned state of the blood in these cases is due to the alteration and transformation of the connective tissue of the valves themselves, and never to the mere disintegration of fibrinous concretions.

These speculations have, however, been almost superseded by observations of a different order. As far back as 1855, Virchow[1] found that a small coagulum upon the mitral valve (in a case of erysipelatous perimetritis with a diphtheritic inflammation of the large intestine) contained a number of small white miliary bodies, which consisted almost entirely of fine closely aggregated granules, embedded in a gelatinous substance. These granules were insoluble in potash, acetic acid and hydrochloric acid, but were dissolved by chloroform, so that he regarded them as probably of a fatty nature. Charcot and Vulpian[2] afterwards insisted on the peculiar micro-chemical relations of the detritus of diseased valves, shown in their power of resisting strong acids and alkalies. But still more recent observations have tended to show that the properties of these minute granules are not due merely to their chemical constitution, and that they are in fact living organisms. Prof. Winge, and Prof. Heiberg,[3] of Christiania, appear to have been the first writers to express this view in a decided form: it has since been adopted by no less an authority than Virchow himself. It is proposed by these writers to give to the affection in question the name of *Mycosis Endocardii*. Winge's case, which occurred in 1869, was that of a man, æt. 44, who died with symptoms of blood-poisoning apparently dependent on a suppurating corn. On the aortic valves there were certain grayish masses, the size of peas or beans, which could be easily picked off, leaving the surface slightly uneven and ulcerated. The tricuspid valve presented similar masses. With a microscope of moderate power these appeared to consist of a fine network of fibrin threads. But under a higher objective these threads were seen to be made up of rod-like or spherical bodies, arranged in chains, and thus resembling leptothrix. There were also a number of fine rounded or rod-shaped bodies, some of which were probably bacteria, others fat granules. Similar bodies were found in the cylindrical plugs in the smaller arteries of the kidney, corresponding to infarctus. Heiberg's case was that of a girl, æt. 22, who died six or seven weeks after delivery, with symptoms of blood-poisoning. The mitral valve was perforated by a recent ulcer,

[1] Gaz. Méd. 1862, p. 428.
[2] Path. Trans. xxi. p. 107.
[3] Traité clinique et expérimentale des embolies capillaires. 2ème éd. Strasbourg, 1870.
[4] Experimentelle Untersuchungen zur Physiologie und Pathologie der Embolie, &c., Berlin, 1864.

[1] Op. cit. p. 709.
[2] Gaz. Méd. 1862, p. 385.
[3] Virchow's Arch. lvi. 1872, p. 409.

the margins of which and the chordæ were coated with vegetations. These contained numerous minute granules, apparently simple detritus: and in addition, many rod-shaped bodies resembling bacteria, and a considerable number of rows of granules, of uniform size, arranged in chains of greater or less length, which Heiberg therefore regarded as leptothrix. These, and many of the isolated bodies, resisted the action of even boiling caustic potass. Specimens from both these cases were forwarded to Virchow, who confirms the accuracy of the accounts given by the Swedish writers, and states that he has no doubt as to the parasitic nature of the bodies in question. He is not yet prepared, however, to admit the propriety of using the name leptothrix for them. Eberth,[1] of Zürich, has since recorded another case of the same kind, which differs from those previously referred to, in the fact that there was no evident external source of blood-poisoning. He entitles it "Diphtheritic Endocarditis."[2] It occurred in a young man, previously healthy, who died after little more than two days' illness. Two of the aortic valves were ulcerated through, and the disease extended into the muscular substance of the heart, penetrating almost to the endocardium lining the right auricle. The margins of the affected valves were covered with soft vegetations. These consisted mainly of a finely granular substance: and the individual granules were shining spherical bodies of uniform size, some of which exhibited slight movements, the majority being motionless and embedded in a gelatinous material. Neither boiling alcohol nor boiling alkalies affected these granules, beyond making them slightly paler. Tincture of iodine and sulphuric acid gave them a yellow color. It is therefore almost certain, says Eberth, that they were really spherical bacteria.

So far as I am aware, no similar observations have as yet been published in this country. But my colleague, Dr. Goodhart, informs me that he has in three instances detected minute organisms in the fungating masses attached to ulcerated valves. In each case he found, besides innumerable spheroids, rod- and dumb-bell-shaped bacteria, as well as some which formed beaded strings. Most of these had feeble oscillatory movements. Vertical sections of the deepest part of the diseased valves showed a cell growth, to a small extent, such as is described at page 708. On this was deposited a hyaline clot in small rounded masses: and upon these, and in the crevices between them, the bacteria clustered. Dr. Goodhart, however, considered that the appearances which he observed were strongly suggestive of the view that the bacteria were derived from the elements of disintegrating blood-clot.

The precise scope and bearing of these observations are, as yet, imperfectly understood; but I think there can be little doubt that they will hereafter be found to play an important part in the explanation of blood-poisoning now under consideration. Heiberg, indeed, expressly states that he does not attribute all cases of ulcerative endocarditis to a Mycosis, since he has failed to find any parasitic organisms in specimens of this disease preserved in the Museum of Christiania. And when bacteria are present in the tissues of diseased valves, it is as yet quite impossible to say what relation they bear to the processes of embolism and infection to which the disease gives rise. This question is in fact only a part of the much wider one which concerns the relations of these minute organisms to pyæmia, septicæmia, and allied processes. The theory advocated by Eberth[1] is that the bacteria originally enter the blood from without, and then become aggregated together into a sticky mass, which adheres to the surface of the cardiac valves, when it is brought to them in the stream of the circulation. In confirmation of this opinion, he appeals to observations showing that the ante-mortem coagula in the appendices of the auricles are likewise often coated with a complete layer of bacteria. The valves and chambers of the heart thus form a kind of halting-place for the microphytes, which multiply, and subsequently distribute to all the arteries of the body masses of bacteria in the form of embola, which set up suppuration wherever they are deposited. In the arteries of the kidneys especially, agglomerations of this nature have been demonstrated: and also within the glomeruli and the uriniferous tubules of the affected parts of these organs.

II. Another series of effects produced by diseases of the cardiac valves consist in the modifications that they tend to induce in the circulation of the blood, and

[1] Virchow's Arch. lvii. 1873, p. 228.

[2] This designation has also been frequently used by Virchow. It is important for English readers to remember that German writers use the term diphtheritic in a sense very different from that to which we are accustomed in this country, applying it to inflamed structures of which the most superficial layers, infiltrated with inflammatory materials, are gangrenous.

[1] In a large number of recent cases of pyæmia Eberth has constantly found microphytes, not only on the surface of the wound, but also in the subjacent tissues, sometimes to a considerable depth.

in the consequent morbid changes which arise in the several cavities of the heart, in the bloodvessels, and in distant organs. To these effects we must now turn our attention, and as they are both numerous and varied, it is needful that we should arrange them in as orderly a manner as possible.

Each of the cardiac valves may be viewed as separating from one another two of the chambers of the circulatory system, and when any one of the valves is diseased, we may consider that (1) the *primary* effect of the disease is exerted upon that chamber which lies immediately behind the valve in the order of the circulation, and which was protected by the valve when in its normal state. From the chamber in question, again, disturbance of the circulation is, or may be, propagated in two directions:—(2) *forwards*, or with the blood-stream; and (3) *backwards*, or against the blood-stream. The effects of disease of the several valves have, therefore, to be considered under these three heads.

A. It will be found convenient that we should begin with diseases of the *aortic valves*. These, as we have seen, may be of two kinds, obstructive and regurgitant; but in the immense majority of cases obstruction and regurgitation coexist.

(1) The primary effect of diseases of the aortic valves may be said to occur in the left ventricle, which is of course the chamber that lies behind the valves in the order of the circulation. Now in aortic stenosis or obstruction the blood cannot be forced into the aorta so easily nor so quickly as in health. The ventricle, therefore, tends to be overloaded with blood, and its walls become stretched or dilated; at the same time it has to exert increased force to propel its contents onwards; and it consequently becomes hypertrophied. In aortic regurgitation the ventricle may empty itself readily enough during its systole, but in its diastole it not only has to receive the blood flowing onwards from the auricle, but also that which is poured back into it from the aorta; it therefore becomes both dilated and hypertrophied. The changes which occur in the left ventricle are thus the same in the two conditions of stenosis and regurgitation respectively. They constitute the *compensation* by which these several morbid changes are more or less completely prevented from further disturbing the circulation. But there is a distinction of some importance, which has not, I think, been noticed by writers on this subject. In aortic stenosis, hypertrophy of the ventricle is all that is needed to restore the balance; dilatation is directly injurious, tending to impair the power of the chamber, and to render still more hypertrophy necessary. But, in aortic regurgitation, dilatation is the main requirement, since the ventricle has to accommodate the blood that enters it from both sides during its diastole; hypertrophy is needed only secondarily, and because a dilated ventricle has to exert more force than one of normal size, in order to propel its contents onwards.

The dilatation and hypertrophy of the left ventricle in cases of aortic disease may be extreme in degree. The heart then acquires a peculiar pointed form, the right ventricle often looking like a mere appendage. The organ often weighs between 20 and 30 ozs., and many instances have been observed in which it has been even heavier. In one case which I have myself examined—that of a young man, æt. 26—the heart weighed 48 oz. I am not sure whether this is not the largest heart on record; the next largest being one weighing 46½ oz., which Dr. Bristowe exhibited at a meeting of the Pathological Society.

These changes, of course, require time for their development; but Dr. Peacock has adduced evidence to show that they may take place more quickly than might have been expected. Valvular affections themselves often arise gradually; and the compensatory processes are induced *pari passu* with the disease. On the other hand, when the valves give way or are lacerated suddenly, time may not be allowed for the ventricle to become dilated and hypertrophied; and this is probably one of the main reasons why in such cases the fatal termination is often rapid. Again, either obstruction or regurgitation may of course be so extreme as to render compensation impossible. Lastly, when perfect compensation has existed for a considerable time, it may begin to fail; and then further effects arise which will be considered hereafter. It is generally supposed that this is due, either to the progressive increase in the valvular changes (with which the compensatory processes are unable to keep pace), or to the occurrence of fatty degeneration in the hypertrophied ventricular wall.[1]

[1] Dr. Allbutt has recently given another explanation of loss of compensation, which is certainly of great interest. It was first suggested to him by Mr. Busk, who compared the change in question to that which occurs in the arms of file-cutters. These men constantly practise rapid flexions of the elbow-joint, and the biceps enlarges greatly. But after a few years the muscle again wastes, and falls far below the normal value. This is so certain a consequence, that the file-cutters receive high wages, calculated upon the average duration of an hypertrophied biceps. ("On the Effects of Overwork and Strain upon the Heart and Great Vessels," p. 43, Macmillan and Co., 1872.)

(2) The onward effects of disease of the aortic valves consist in changes in the blood current in the aorta and its branches; in other words, in changes in the arterial pulse. These are not the same in aortic obstruction, as in regurgitant disease; and the two affections must therefore be considered separately.

In aortic stenosis the character of the pulse appears to be but little altered, unless the obstruction to the blood current is extreme, in which case Walshe says that "the pulse, though regular in force and rhythm, is small, hard, rigid, and concentrated." Dr. Wilks has mentioned to me that in certain cases he has observed the number of pulsations of the heart, per minute, to be greatly reduced. In illustration of this fact, I find in the notes of post-mortem examinations at Guy's Hospital two cases recorded by Dr. Wilks himself. One[1] is that of a man, æt. 68, in whom "two of the aortic valves were adherent and bony; the aperture was reduced to a very narrow chink; the edge of one valve slightly overlapped the bony margin of the other, and thus no doubt prevented regurgitation. The pulse during life had been 40 per minute, very small, and sometimes hardly perceptible." The other[2] is that of a youth, æt. 19, in whom the pulse was said to have been "small and slow. The aortic orifice would only admit a catheter; all the valves were adherent together, leaving only a small rounded hole in the middle." Such cases are doubtless exceptional; but, as has already been stated, aortic stenosis, without regurgitation, is decidedly a rare affection.

In regurgitant aortic disease the pulse presents characters so remarkable that they have led to its receiving several special designations, and that they often enable the physician to diagnose the nature of the case without aid from any other source. A passage has already been quoted from Vieussens (1715)[3] in which the peculiar character of pulse that is now known to belong to this affection is clearly indicated. So far as I am aware, the next writer to mention it was Dr. Hodgkin, who, in his paper on "Retroversion of the Aortic Valves,"[4] published in 1829, says that in one case there was "inordinately violent arterial action, which was very rapid and frequent, although regular, there was a remarkable thrill in the pulse, and the carotids were seen violently beating on both sides." But it was Sir Dominic Corrigan,[5] who in 1832 first laid stress on the peculiarity of the pulse in this disease, a fact commemorated in the designation of "Corrigan's pulse," which is commonly applied to it both on the continent and in this country.

The feature on which Corrigan especially insists, as indicating "inadequacy of the aortic valves," is the existence of visible pulsation in the arteries of the head and superior extremities. He describes the subclavian, carotid, temporal, brachial, and even palmar arteries as being "suddenly thrown from their bed, and bounding up under the skin." In the arteries of the lower extremities, even of larger size than those which present it about the head and neck, pulsation is not (he goes on to say) seen to any comparative degree, and generally not at all, while the patient is sitting or standing. The pulsation of the brachial and palmar arteries is increased in a most striking degree by merely elevating the arm above the head: and the same effect is produced in the lower limbs by lying down and elevating them on an inclined plane.

In addition to these points, it may be added that, in aortic regurgitation, the arteries are elongated during their pulsations much more than in health, and can be seen in many positions to become distinctly flexuous with each beat of the heart. Consequently, one name for the pulse in question is that of the "locomotive" pulse.

But these visible characters of the pulse of aortic regurgitant disease are after all of little consequence in comparison with those which can be felt. To the touch, the pulse in question gives a sensation of peculiar largeness or fulness, immediately followed by an equally peculiar collapse. Instead of the artery slowly receding beneath the finger, it falls as rapidly as it rose. The pulse is, therefore, often spoken of as "jerking," "splashing," or "collapsing;" or as the "water-hammer" pulse, from the well-known scientific toy of that name.

Lastly, the pulse of aortic regurgitation differs from that of health in travelling along the arteries much more slowly. Normally, even the radial pulse follows very quickly upon the ventricular systole; in the disease under consideration, it may almost be synchronous with the second sound of the heart.

There is little difficulty in explaining the peculiarities that have been enumerated. We have seen that when the aortic valves allow of regurgitation, the ventricle is greatly, often enormously, dilated and hypertrophied. The quantity of blood injected into the aorta is, therefore, much increased. No wonder that the pulse feels full and large, that the arteries lengthen, and seem to bound from their seats, beating much more plainly than in

[1] Inspection 109, in the year 1859.
[2] Inspection 72, in the year 1862.
[3] Œuvres Françoises.
[4] London Med. Gaz. vol. iii. p. 438.
[5] Ed. Med. and Surg. Journal, April 1, 1832, p. 225.

health. Then comes the elastic recoil of the larger arteries. Under normal conditions, this is gradual. The aortic valves are closed, and the blood moves slowly onwards into the small arteries and capillaries, meeting considerable resistance. But when the valves in question are diseased, and allow reflux to take place through them, there is nothing to support the column of blood in the aorta and its branches during their recoil; the blood is rapidly driven out of them, part one way and part another; and the pulse as suddenly collapses.

Since the invention of the sphygmograph, no description of the peculiarities of the pulse in any morbid state can be regarded as complete unless full reference is made to the results obtained with that instrument. And probably diseases of the aortic valves were among the first in which the sphygmograph was applied. It cannot, indeed, be said that those who have specially devoted themselves to this subject have as yet come to a complete agreement in reference to the indications which it affords. But I believe that the existing state of our knowledge is fairly expressed in the following account of the matter:—

In aortic stenosis, one might expect that, in proportion as the aortic orifice is obstructed, the exit of blood from the ventricle would be impeded. The upstroke of the sphygmographic tracing should, therefore, be oblique, or sloping. According to Mahomed, this is the case. I append (Figs. 107 and 108) copies of two tracings given by this observer in the *Medical Times and Gazette* for 1872,[1] which show well the sloping upstroke and the rounded summit, indicative of the fact that "the influence of percussion is lost; the tidal wave alone remains."

Fig. 107. Fig. 108. Fig. 109.

Very similar to this is another diagram (Fig. 109), which is a copy of one given by Jaccoud.[1] According to Mahomed, however, another very different form of pulse may accompany aortic obstruction. It is illustrated in the following diagrams (Figs. 110, 111, 112), which are copied from those given by him.[2] It will be observed that there is a marked separation between the percussion and tidal waves. It ought perhaps to be mentioned that, in the case from which the tracing No. 112 was taken, there was a double murmur over the aorta, but the existence of considerable aortic obstruction was made out, not only from the characters of the pulse, but also from the fact that a tracing obtained from the heart showed the contractions to be very slow and gradual. It is to be borne in mind that only extreme degrees of aortic stenosis can be expected to affect the pulse in the ways described by Mr. Mahomed. He himself gives a tracing from a case in which "considerable obstruction was produced by the adherence of two of the aortic valves;" in this tracing no sign of the obstruction is apparent.

In aortic regurgitation, the sphygmographic tracings of the pulse present peculiarities which correspond in a very striking way with what might theoretically have been expected. The percussion-wave is strongly marked, and the upstroke is therefore high. On the other hand, the dicrotic wave (or "diastolic expansion") is wanting, in consequence of the aortic valves failing to support the column of blood in the aorta during its recoil. Lastly, a high pressure is required to bring out the characters of the pulse fully; this being the result of the hypertrophy of the left ventricle, which is constantly present in cases of aortic regurgitation.

The three following figures, which are copies of tracings given by Mr. Mahomed,[2] illustrate these points. It ought perhaps to be added that Marey originally laid great stress on a little peak or point at the summit of the long upstroke, as indicative of aortic regurgitation; but this was soon shown to be a mistake. At the present time, there seems to be a fair agreement among different observers as to the characters in a sphygmographic tracing which point to the disease in question.

In some cases of aortic regurgitation the pulse does not present its peculiar characters in any marked degree, whether to the touch or to the sphygmograph; and

[1] Plate V., Figs. 12 and 13, p. 142.

[2] Traité de Pathologie Interne, quatrium Ed. tome i. p. 676.

[1] Loc. cit., Pl. V., Figs. 17, 18, 19.

[2] Loc. cit., Plate V., Figs. 7, 4, 6 respectively. In Fig. 118 (Fig. 4 in Mr. Mahomed's plate) there are also indications that the arteries are atheromatous.

this, although the diastolic murmur may be loud and prolonged. This may be due either to the circumstance that the reflux of blood is really small in amount, or to the fact that mitral regurgitation is also present. Mr. Mahomed gives in his papers in the *Medical Times and Gazette* some very valuable illustrations of the

Fig. 110.

Fig. 111.

Fig. 112.

way in which the sphygmograph may be used in cases of this kind, both to determine the degree of valvular incompetency, and to gauge the amount of compensatory hypertrophy of the left ventricle; and also to decide which of two coexistent affections—mitral and aortic—is of preponderating importance. It is in the solution of such questions as these that the great value of the instrument appears to lie, so far as diseases of the cardiac valves are concerned. The mere detection of valvular incompetency can be effected more easily, and perhaps as surely, by the

Fig. 113.

Fig. 114.

Fig. 115.

stethoscope; but in prognosis the sphygmograph seems to lend great assistance.

The onward effects of diseased conditions of the aortic valves are not necessarily confined to the arterial system. The capillaries may be imperfectly supplied with blood, and both the nutrition and the functions of the different organs may in consequence be greatly impaired. This is perhaps especially marked in the case of the brain. Attacks of giddiness are far from uncommon in aortic regurgitation, and are ascribed to failure in the due supply of arterial blood to the nervous centres. Anæmia and wasting of the whole body are also frequent symptoms: the former being in fact so constantly present as to be a marked feature in the physiognomy of the disease.

(3) *Backward* effects of aortic disease are absent, so long as the changes in the left ventricle above described enable the heart to do its work efficiently, even though this result should be attained at the expense of increased labor and friction, and under augmented frequency of beats. And since patients with aortic regurgitation very often die suddenly while these conditions are fulfilled, backward effects are not rarely wanting to the last. But whenever the compensatory processes fail, so that the arteries no longer receive for transmission onwards their full supply of blood per minute, the necessary result is that the quantity discharged into the ventricle by the left auricle must also be deficient. The inevitable consequence of this, again, is the development of a fresh series of changes, which we are about to study in detail, as the effects of primary disease of the mitral orifice. It is often stated that in affections of the aortic valves these changes occur only when the mitral valve has been stretched, so as to allow of regurgitation through it—this being probably a common result of the dilatation of the left ventricle. But I conceive that the statement in question is an error, and that backkward effects must necessarily arise in the way I have indicated, even though the closure of the mitral valve may still remain perfect.[1]

B. Diseases of the *mitral* valve, again, are of two kinds—obstructive and regur-

[1] So far as post-mortem evidence can be brought to bear upon this question, I believe that such evidence is favorable to the view expressed in the text. Thus I find in my notes one case (in which I made an autopsy in July, 1873) of aortic disease with retroversion of one of the valves. Dropsy occurred before death, and the lungs contained apoplectic patches. The mitral valve appeared to be quite healthy; and, after death, it did not allow regurgitation to occur. The left auricle was dilated and hypertrophied, and the right auricle was still more so.

gitant: which will to some extent require to be considered separately from one another.

(1) The *primary* effect of diseases of the mitral valve may be said to be exerted upon the left auricle. In mitral stenosis the effect in question is well marked. The cavity becomes dilated, often enormously so.[1] The appendix is elongated—in one instance I find it noted as 2¾ inches long by Dr. Moxon—and acquires a peculiar curved form; and its aperture of communication with the auricle is much wider than natural. The walls of the auricle also become much hypertrophied; they no longer collapse when the cavity is cut open, but support themselves stiffly: the muscular substance may in places be from ⅛ to ¼ of an inch thick. The endocardial lining is said to be more opaque than usual.

These changes are almost constantly met with in cases of mitral stenosis. And were the current doctrines in regard to mitral regurgitation true, they would doubtless be found no less uniformly in cases of the latter affection;—just as dilatation and hypertrophy of the left ventricle occur equally in aortic obstruction and in aortic incompetency. However, this is not so. Definitely marked hypertrophy of the muscular wall of the left auricle is seldom present in cases of the so-called mitral regurgitant disease. It is true that the cavity in question is often found to be dilated; but then all the other cardiac cavities are generally enlarged at the same time. I shall endeavor to explain these facts further on.

(2) The *onward* effects of diseases of the mitral valve are of course seen first in the left ventricle. In mitral stenosis this chamber is very generally found to be small, and its muscular substance is no thicker, and may perhaps even be thinner, than under normal conditions. The aorta too is often small and thin-walled. But in some cases of mitral stenosis and in almost all cases of "mitral regurgitation" the left ventricle is large and fleshy; and not infrequently it is as much dilated and hypertrophied as in aortic regurgitation. Various explanations of this have been given. By Friedreich[2] it is supposed that the augmented tension in the systemic venous system (which we shall presently show to be one of the consequences of mitral diseases) causes an increased resistance in the systemic arteries likewise. But, apart from the difficulty of admitting that the effects of obstruction thus traverse the complete circuit of the circulation, a fatal objection to this theory is that it would require dilatation of the left ventricle to be the rule in fatal cases of mitral stenosis, instead of its being quite exceptional. Another view is that when the ventricle is enlarged in mitral disease, this is not really due to the valvular affection, but depends upon some other cause. Thus, in rheumatic cases many other conditions generally exist (such, for example, as diseases of other orifices, or thick pericardial adhesions) to which the change in the ventricle may be ascribed. Indeed, according to some observers, primary dilatation of the left ventricle commonly occurs in the course of acute rheumatism, and may persist after the subsidence of that disease. But, again, in very many cases of so-called "mitral regurgitant disease" the valve is itself healthy: and the imperfection in its working (if we are to assume that it does close imperfectly) is itself the result of ventricular dilatation. There is, however, one class of cases in which it certainly appears that mitral imperfection leads to enlargement of the left ventricle—I refer to those cases in which rupture of the tendinous cords of the valve occurs in persons who had not previously exhibited any symptoms of cardiac disease.[1] It may indeed be objected that both the ventricle and the valve were possibly affected with latent disease before the sudden rupture took place: but of such disease there is no evidence, and to suppose its existence is to abandon in favor of an arbitrary hypothesis the direct interpretation of the facts observed. The explanation, indeed, seems to be sufficiently easy. In such cases, the ventricle has greatly increased labor; a good deal of the blood which enters it having to be expelled twice over from its cavity. On the other hand, in cases of uncomplicated mitral stenosis, the work thrown upon the left ventricle is in no way augmented, if it be not even less than under normal conditions: and, as I have already stated,

[1] This condition was long ago described as "true aneurism of the left auricle" by Dr. Thurnam (Med.-Chir. Trans. ser. ii. vol. iii. 1838, p. 244), who expressly insists on its association with contraction of the mitral orifice, and mentions that the lining membrane is opaque and rough, and in some cases even ossified, and that it is lined with fibrinous layers very similar to those met with in arterial aneurisms.

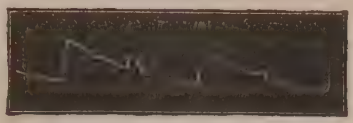
[2] Op. cit. pp. 161 and 227.

[1] Thus in Dr. Dickinson's case (Path. Trans. xx. p. 150) the heart weighed 20 oz.; all the cavities were dilated to at least three times their natural capacity; the auricles and right ventricle were thinned. The left ventricle was hypertrophied to such an extent as to retain, notwithstanding its dilatation, about its normal thickness. And in the report of the post-mortem examination of a similar case that occurred in Guy's Hospital under Dr. Habershon's care, Dr. Moxon states that "all the cavities were dilated."

I believe that in such cases the left ventricle is always small, and its muscular substance no thicker than natural.

The arterial pulse in mitral diseases may present very varied characters, the variations depending not merely upon the nature of the valvular lesion, but also upon the changes secondarily induced by it in the heart's chambers. Formerly, it was supposed that in mitral stenosis the pulse is always small; but since the presystolic murmur has enabled this condition to be diagnosed before severe symptoms set in, it has been found that the pulse is often perfectly natural. Indeed, there is no reason why it should be otherwise, so long as the hypertrophied auricle keeps the ventricle duly supplied with blood. In a very large proportion of cases in which a presystolic murmur is audible, the pulse is perfectly regular, and has ample volume and force. Accordingly, Mr. Mahomed says[1] that "in this disease the sphygmographic tracing does not necessarily present any diagnostic characteristics." I have already quoted this writer as having demonstrated that cardiographic tracings, taken at the heart's apex, often afford proof of the existence of mitral stenosis (or, at least, of hypertrophy of the left auricle), by showing that the auricular systole commences at an earlier period in the ventricular diastole than is normal. He further maintains that in some cases this premature contraction of the auricle stimulates the ventricle to contract likewise; and that in this way the tracing of the pulse at the wrist may indicate a second ventricular systole, alternating with the main beat, but very much less forcible. The accompanying diagram is copied from one of Mr. Mahomed's tracings, taken from a patient of mine who was suffering from mitral stenosis, and in whom the double

Fig. 116.



ventricular systole was made very marked by the administration of digitalis. Both contractions were felt in the pulse at the wrist, the beats of which were alternately strong and feeble. I have observed a similar double rhythm in several other instances of valvular disease; but I am unable to say whether they were or were not all of them cases of mitral stenosis.

In the later stages of the disease—when the peculiar murmur can often be no longer detected—the pulse assumes very different characters. It is now rapid, soft, small, and very irregular, both in volume and force.

The accompanying tracings (Figs. 117, 118, 119) copied from Jaccoud,[1] show the sphygmographic character of a pulse of this kind; they are very much what might have been expected from the impression which it gives to the touch. It has long been known as the *mitral* pulse; and, in fact, it is met with, not only in the

Fig. 117.

Fig. 118.

Fig. 119.

advanced stages of mitral stenosis, but also in those cases which are commonly grouped under the heading of "regurgitant mitral disease." Whether it is of any diagnostic value, as indicating that the valve in question is impaired in structure or function, is a very difficult question to answer. I have already stated more than once that "regurgitant mitral disease" has no constant pathological appearances, but that it includes a variety of conditions, in some of which the valve certainly admits of regurgitation, while in others there is doubt whether this occurs. I must now add my belief that for the production of the so-called "mitral pulse" the mitral valve need not be either narrowed or incompetent. The same kind of pulse probably arises whenever the ventricle does not empty itself completely during its systole, so that the stream of blood projected into the aorta is greatly diminished. Now it would appear that such a perversion of the heart's action is far from being uncommon, being liable to occur in the course of various cardiac and pulmonary diseases without presenting any characters peculiar to one rather than to another of these diseases. The condition in question was first described by Beau, who gave it the name of

[1] Op. cit. No. 6, p. 569.

[1] Op. cit. No. 21, p. 678; No. 9, p. 616; No. 7, p. 615.

asystolie; and most recent French writers have adopted this designation. Dilatation of the heart appears to be the morbid change which is most constantly present in cases of this kind; but very frequently valvular disease also exists. The sphygmographic tracings (Figs. 117 and 118), which I have copied from Jaccoud as illustrative of the "mitral pulse," are given by that writer as indicating the existence of a condition of "*asystolie.*"

(3) *Backward Effects.*—So long as the left auricle can duly empty itself, and receive its full supply of blood from the pulmonary veins, the parts of the circulatory apparatus behind the auricle are in no way affected by the existence of mitral disease, whether obstructive or regurgitant. But, except in the earlier stages or slighter degrees of such disease, the compensatory action of the auricle is very seldom thus complete; and whenever it fails, the necessary consequence is an augmented tension in the pulmonary system of vessels and in the chambers of the right side of the heart. It has already been stated that the same result occurs also in diseases of the aortic valves, as soon as compensatory changes fail to enable the left ventricle to carry on the circulation properly.

This increase of tension in the pulmonary vessels soon leads to changes in their walls, which become thickened, or hypertrophied. In the main trunk of the pulmonary artery this is particularly noticeable. The records of post-mortem examinations at Guy's Hospital contain notes by Dr. Moxon of the case of a boy, æt. ten years, in whom the coats of the pulmonary artery were nearly twice as thick as those of the aorta at its thickest part; and less striking examples of the same kind are very commonly met with. The artery also becomes greatly dilated.

Another result of the increased tension of blood within the pulmonary artery is the fact that in these cases the branches of the vessel are very apt to become atheromatous, although under normal changes they are but little liable to such a change. Perhaps the most striking instance of this that could be quoted is one which Dr. Conway Evans[1] has recorded, and which occurred in a boy, who died of dropsy, consequent on mitral stenosis, at the age of fourteen years. It would appear that Dittrich[2] was the first to point out the frequency with which atheroma of the pulmonary artery is found in cases of this kind, and that he described it as occurring especially in the smaller branches, and as being the immediate cause of the patches of "pulmonary apoplexy" which are so commonly met with under such conditions. The explanation of pulmonary apoplexy, however, is still open to doubt. The branch of artery leading to an apoplectic patch is generally, perhaps always, plugged with fibrin; and this has led many modern observers to regard the affection as of embolic origin. In the first volume of the "System of Medicine," Dr. Bristowe has discussed this question at considerable length.

The pulmonary tissue is also liable to assume a peculiar appearance, which is generally known to German pathologists under the name of "brown induration." In this volume of the present work, at p. 274, Dr. Wilson Fox has given a detailed account of this affection; but he seems to have laid hardly enough stress on the dilated and varicose state of the pulmonary capillaries, which Buhl has shown to be present, and which is so striking a proof of the increased pressure upon these vessels. I have found that this dilated state of the capillaries is recognizable without difficulty, even in uninjected specimens.

Before leaving the subject now under consideration, I must not omit to mention another way in which the left lung suffers from cardiac disease—namely, from the dilated left auricle pressing directly upon the bronchus. Mr. Wilkinson King first pointed this out, in the year 1838, and his preparations, which are now in the museum at Guy's Hospital, show that the anterior surface of the tube may in this way be rendered quite flat, and its calibre diminished by one-half. But the most remarkable instance is one recorded by Friedreich[2] in which narrowing of the left bronchus was diagnosed four years before the patient's death, from the presence of a loud humming sound accompanying both the inspiration and the expiration, heard most plainly over the root of the left lung, near the spine, but also audible over the whole left side of the chest. There was extreme stenosis of the mitral orifice with enormous dilatation of the left auricle. Virchow made the autopsy; and the left main bronchus was found to be compressed, so that only a small narrow channel was left.

The cavities of the right side of the heart also become greatly dilated and hypertrophied under the conditions now being considered. The muscular tissue of the right ventricle grows much harder than natural—indeed, it is peculiarly hard, in comparison even with the substance of an hypertrophied left ventricle. The tricuspid orifice is stretched.

C. & D.—It is at this point that we

[1] Trans. of the Path. Soc. xvii. p. 90.

[2] Ueber den Laennec'schen Lungen-Infarktus. Erlangen, 1850.

[1] Guy's Hospital Reports, series i. vol. iii. p. 178.

[2] Op. cit. p. 30.

ought to consider the effects of primary disease of the pulmonary and the tricuspid valves respectively. But such diseases are so rare (excepting malformations, which are treated of separately) that they need scarcely interrupt us in tracing out the backward effects of diseases of the valves of the left side of the heart. It will suffice to state that (1) the *primary* effect of disease of the pulmonary valves is to cause dilatation and hypertrophy of the right ventricle; and that that of disease of the tricuspid valve (if primary chronic disease of this valve ever occurs) would probably be to cause dilatation and hypertrophy of the right auricle; (2) Concerning *forward* effects of the disease in question, no definite statements could perhaps be made; (3) Their *backward* effects must be the same as those which more remotely arise from uncompensated diseases of the mitral and aortic orifices, and to these our attention may now be directed.

Taking first the vena cava superior and the veins from which it arises, we find that they are enlarged and gorged with blood. Hence the livid countenance, the turgid cheeks, the purple ears, cheeks, and lips, that are so commonly seen in patients suffering from affections of the cardiac valves. The veins of the upper limbs are also distended; the hands and nails acquire a livid purple color, and the hands, and often even the arms, become œdematous. The lividity may approach, if it may not even equal, that which is seen in cases of malformation of the heart, in the condition known as *cyanosis*. A further consequence of the congestion of the upper limbs which exists in these cases, is that the finger-ends often become enlarged, or (as it is usually termed) "clubbed." Dr. Dobell[1] has recently stated that the clubbing of the fingers from heart disease differs from that which is due to phthisis, in the circumstance that the sides and tips of the nails are not at the same time incurved; the reason for this difference being, that in heart disease wasting of the adipose tissue is absent, which wasting he believes to be the cause of incurvation.

At the root of the neck the jugular veins, besides being enlarged and unnaturally full, present another phenomenon which requires further consideration—they can often be seen to pulsate with each beat of the heart. This seems to have been first noticed by Lancisi.[2] Jugular pulsation is commonly taken as a certain indication of regurgitation through the tricuspid orifice; and the frequency of its occurrence, when the circulation through the right side of the heart is impeded, is supposed to bear out Mr. Wilkinson King's views of the existence of a physiological safety-valve action, by which reflux is allowed whenever the right ventricle becomes unduly charged with blood. It has, however, been shown by Friedreich that the matter is by no means so simple. In the first place, when the jugular veins are distended they often exhibit rhythmical movements synchronous with the respiratory acts. Each expiration causes an increased pressure upon the large venous trunks within the thorax; and even though the valves at the root of the neck may close perfectly, the blood that is pouring in from the veins of the head and upper limbs is stopped, and accumulates behind the obstruction. An apparent pulsation may thus occur without any blood really regurgitating into the jugular veins from below. So, again, it is possible that when these veins are very full, variations in their size may occur, synchronously with the heart's movements, from the temporary arrest of the onward flow of blood during the closure of the tricuspid valve, quite independently of reflux. In this case, however, compression of the veins in the middle of the neck will at once stop the apparent jugular pulsation.

When jugular pulsation is really due to regurgitation of blood, it is of course necessary that the valves at the junction of the subclavian and jugular veins should be incompetent. Dr. Parkes[1] is said to have taught that this is due to rupture of these valves: but as Dr. Walshe points out, it is doubtless sufficient that the veins should be greatly distended, so as to prevent the edges of the valves from touching one another. According to Friedreich it is possible for a true jugular pulsation to be produced by the pressure of the ascending aorta, when dilating during the ventricular systole, upon a distended vena cava superior. But this explanation appears far-fetched, and unnecessary. Friedreich will not allow that tricuspid regurgitation is present, unless a systolic murmur is audible. I shall presently show, however, that almost any kind of valvular defect may exist, without the corresponding murmur: and my belief at present is that regurgitation through the tricuspid orifice exists in all cases in which the jugular veins really pulsate. Indeed, I cannot even agree with Friedreich that if pulsation disappears when the vein is compressed higher up, the existence of regurgitation is absolutely disproved: for this procedure may simply prevent the wave being transmitted upwards in the empty vessel. The most that can be said

[1] On Affections of the Heart and in its neighborhood, 1872, p. 17.

[2] De motu Cordis et aneurysmatibus. Rom. 1728, Lib. ii. Propos. 57.

[1] Walshe, op. cit. p. 138.

is that it renders the occurrence of reflux doubtful.

Friedreich gives sphygmographic tracings of the jugular pulse, which appears to be dicrotic, the beat due to the ventricular systole being preceded by a smaller elevation accompanying the contraction of the auricle.

It must be added that pulsation is generally more distinct in the right than the left jugular vein. In exceptional cases the veins of the face, arms, and hands have been seen to pulsate: and also the thyroid and mammary veins.

Turning now to the vena cava inferior and its tributaries, we find that these veins become greatly dilated as a consequence of distension of the right auricle. Senac[1] mentioned a case in which the cava was as thick as an arm. The hepatic veins also become much enlarged, running as wide open channels through the substance of the liver, and opening into the cava by orifices much larger than natural. These facts are of some importance, as throwing light on the epigastric pulsation, which is often observed in cases of chronic disease of the heart. It was long ago suggested by Allan Burns[2] that this is due to regurgitation of blood along the inferior cava, and into the vessels of the liver. And Friedreich at the present time maintains the same view.[3] English writers in general, however, describe the dilated right ventricle as giving a shock to the neighboring parts which can be felt in the substernal notch: and some have even spoken of the heart as "beating in the epigastrium," the impossibility of which it did not need the labors of Hamernyk to point out.

The probability that epigastric pulsation is often due to reflux into the hepatic veins is increased by the fact that the liver itself is greatly enlarged under these conditions. It is also much congested and fatty, presenting a peculiar mottled appearance, which has gained for it the name of the nutmeg liver. At the same time it is very liable to a chronic inflammatory process, attended with an increase in its connective tissue, approaching that which occurs in cirrhosis. The congestion is transmitted through the liver to the portal vein and its radicles. The spleen becomes enlarged and its tissue very hard, in this respect contrasting with the still larger but soft spleen which is found in association with ulcerative diseases of the cardiac valves. The veins of the omentum and mesentery are gorged with blood. The stomach has its lining intensely reddened and coated with mucus: hemorrhage takes place into its submucous tissue, and the ecchymosed spots often become exposed by solution of the mucous membrane over them, forming the so-called "hæmorrhagic erosions." The intestines are also greatly congested and lined with mucus: and hæmorrhoids are often developed. These changes in the digestive organs are attended with more or less marked symptoms: partial jaundice; dyspepsia, nausea, sickness, even hæmatemesis; constipation. The engorgement of the veins lying beneath the peritoneum leads to ascites, often of considerable amount.

Nor do the other veins that open into the inferior vena cava escape. Thus the renal veins become distended; and the kidneys are deeply congested, a condition which easily passes into one of chronic inflammation, and often leads to the presence of albumen in the urine. The return of blood from the lower limbs is impeded: the veins are gorged, and very often thrombosis of the femoral veins arises, which, as has already been stated, is perhaps the remote cause of the development of pulmonary apoplexy.

This engorgement of the veins of the lower limbs, although we mention it last in tracing backwards the consequences of disease of the cardiac valves, is in fact often one of the first effects of such disease to be observed; manifesting itself by the transudation of serum through the walls of the most distant venous radicles, and the production of œdema of the ankles and feet. The anasarca, slight at first, may increase until the whole of the lower extremities, the abdominal parietes, and even the genital organs, have become dropsical in the highest degree. As a rule, however, the genital organs remain comparatively free: and in this respect cardiac dropsy differs from that which occurs in renal disease, and the distribution of which is not in the same way dependent upon simple mechanical conditions. On the other hand, the icteroid tinge of the skin, which is generally present in cases of heart disease, is wanting in other forms of dropsy.

III. A third series of effects, produced by diseases of the cardiac valves, consist in sensations of various kinds experienced by the patient. These are the *subjective symptoms* of the diseases in question. They may present all degrees of intensity; they may even be entirely absent.

Pain may be felt either over the heart itself, or in the left shoulder; or it may extend down the inner side of the left arm

[1] Friedreich, p. 41.

[2] Op. cit. p. 265.

[3] My colleague, Dr. Frederick Taylor, has observed distinct pulsation of the liver in four cases of chronic cardiac disease. When one hand was placed in the epigastrium and the other in the right loin, the organ could be felt to expand with each beat of the heart. Guy's Hosp. Rep. (vol. xx. 1875).

to the elbow, or even to the fingers. It may either be a constant aching, or have a "shooting" or "stabbing" character. It is often distinctly paroxysmal, especially in cases of aortic regurgitation, in which it frequently assumes all the features of true angina pectoris. Pain in the arm and hand is sometimes accompanied with numbness: and sometimes (according to Dr. Dobell) these parts are deadly white while the numbness lasts. In some cases the pain is limited to the little and ring fingers, following the distribution of the ulnar nerve to these fingers: but in other cases it affects all the fingers, and even the thumb. Sometimes the pain also passes from mid-sternum to the right shoulder and down the right arm: but when pain occurs in these parts earlier than in the cardiac region, Dr. Dobell thinks that the presumption is in favor of disease of the aorta rather than of the heart.

A very important character of the reflected pains due to cardiac disease is that they are generally aggravated by anything which disturbs the heart's action, and especially by muscular exertion. Not unfrequently, pain is absent so long as the patient is at rest, but comes on at once as soon as he attempts to walk.

Another point, on which Dr. Dobell has particularly insisted, is that the pain of heart disease is often greatly increased by distension of the stomach with food or gas. Hence, when dyspepsia is present, it may easily be regarded as the cause of pain really due to heart disease; and relieving the indigestion may prevent the return of the pain.

Not infrequently, instead of pain, the patient speaks rather of a fluttering sensation in the præcordial region: or simply of palpitation. But it is to be observed that a spontaneous complaint of palpitation is heard far more often when the patient is sufferiug from one of its indirect causes, than when any of the cardiac valves are diseased. Indeed, as a rule, the subjective symptoms of valvular affections are subordinate to the other symptoms. And it may be said that when a patient comes to the physician complaining of pain in the heart, and fearing that he has heart disease, the great probability is that that organ is perfectly healthy.

Another morbid sensation, belonging to the diseases under consideration, is dyspnœa. Very often, indeed, the first thing that suggests a suspicion that there is anything wrong with the patient is that he is conscious of shortness of breath after mounting stairs, or making some moderate muscular effort. When he is at rest, he may be able to breathe comfortably enough; but this freedom from distress often continues only so long as he sits up. As soon as he lies down on an ordinary bed or couch, he becomes aware of unpleasant feelings, which compel him to change his posture. Thus, even in the slighter forms of cardiac valvular disease, it will generally be found that the patient lies at night with his head raised, employing two or three pillows, whereas a man in health would only require one. And in the more severe degrees of such disease, the patient is often utterly unable to lie down, or even to recline backwards. This condition has received a special name, that of Orthopnœa. It doubtless depends upon the circumstance that in the recumbent posture the diaphragm is pressed upwards by the contents of the abdomen (themselves greatly augmented in size), so that the enlarged heart is embarrassed in its movements.[1] Orthopnœa is in many respects a serious symptom. By preventing sleep, it greatly taxes the patient's strength, and diminishes his power of resisting the disease. Moreover, as Dr. Dobell has pointed out, it fatigues the lumbar muscles, and makes the back ache. It keeps the lower limbs at right angles with the trunk, and so, leading to compression of the veins and lymphatics in the groins, increases the œdema of the legs. Scarcely any condition is, in fact, more pitiable than that of a patient in this plight; and any mechanical appliance by which it can be remedied must certainly be an unspeakable boon. For this purpose Dr. Dobell has contrived a "Heart Bed," of which he has given a description and a figure in his book; and from his account it seems to be well worthy of trial in these distressing cases.

There are other subjective symptoms, belonging to the various secondary effects of diseases of the cardiac valves; but space fails me to describe them in detail; most of them have been incidentally referred to in other parts of this article.

DIAGNOSIS.—Under this heading I do not propose simply to recapitulate facts that have already been stated in previous paragraphs; nor shall I attempt to construct any tables which might aid the student in distinguishing diseases of the cardiac valves from other affections with which they may be confounded. In my opinion such tables are scarcely ever made use of in practice; indeed, I do not think that they are applicable to really doubtful cases, in which the difficulty of diagnosis most commonly depends upon either a deficiency of symptoms, or their ambiguity: their being, in fact, such as might belong indifferently to any one of several maladies; or else their being in part such as commonly occur in one dis-

[1] Even when the heart is healthy, the position of its impulse may be higher or lower, according as the patient sits up or lies down, if there be an enlargement of the liver.

ease, in part such as belong rather to another disease. In cases of this kind, diagnostic skill is a matter of judgment and experience; and all that could be said under the present heading could do but little to further it.

There are, however, some important questions in reference to the detection of affections of the valves of the heart which have not yet been touched upon. In discussing each kind of murmur, I have endeavored to indicate all the causes to which it may be due, and to point out how these may be distinguished from one another. But of the absence of murmur I have as yet said nothing. I now propose to consider this question, and to discuss whether abnormal sounds or bruits are constantly present in the various diseases of the different cardiac valves.

And first, with regard to the aortic valves. It may almost be said that in practice the diagnosis of aortic regurgitation depends wholly upon the discovery of a diastolic murmur, audible at certain parts of the thoracic parietes. If such a murmur is heard, the stethoscopist regards it as certain that regurgitation exists. If no such murmur can be detected, there is perhaps no combination of symptoms (unless it be by the aid of the sphygmograph) that would justify the physician in asserting that the aortic valves fail to close. It is therefore a most important question whether a diastolic bruit can always be detected in those persons in whom after death the valves are found to have been incompetent. Now, on looking through the records of post-mortem examinations at Guy's Hospital, I have found that this condition was discovered in 40 cases during the years 1870–71. And on referring to the clinical reports attached to these cases, it appears that in 26 of them regurgitation was positively diagnosed during life; and that in 11 out of the remaining 14 cases the patients came from the surgical division of the hospital, or were less than seven days in the wards (some having been dying at the time of admission, or brought in dead), or had no notes taken of the auscultatory signs which they presented. Thus the proportion of cases of this disease that may be said to have resisted diagnosis was very small.

It has been stated that several of the cases in which the aortic valves were found incompetent after death during the period named were cases of surgical disease or injury, in which one may presume that there were no obvious symptoms of cardiac disease. This accords well with the fact that aortic regurgitation is more frequently than any other valvular affection discovered by the auscultator when the patient's history and symptoms had not previously suggested any suspicion of its existence. Dr. Walshe relates the case of a man about 35 years old, the very picture of robust health, who had never had a symptom of disease connected with any organ of his body, and who presented himself for life insurance. Almost as a matter of form, Dr. Walshe put his stethoscope to the chest; his attention was at once arrested by a loud diastolic murmur. The man dropped dead in the street within a fortnight. I lately saw a bank clerk, aged 32, whose sole complaint was a pain in the chest about the ensiform cartilage, with occasional pain in the back, such as might have been due to any trifling cause. On listening to his chest I heard a well-marked diastolic bruit.[1]

It might be supposed that there would often be a difficulty in distinguishing between the to-and-fro sounds of pericarditis and of those of disease at the aortic orifice. And for my own part I believe that this difficulty would arise oftener than it does, were not for the very different clinical history and course and other symptoms belonging severally to these two diseases. The comparatively superficial seat of pericardial friction-sounds, their want of definite localization at the spots where valvular murmurs are most

[1] A very striking instance, in which the patient discovered the murmur, has just come under my notice in a young medical man, a friend of my own. On January 23d, 1875, he had gone to his brother's for a day's shooting; and while at lunch, he noticed a strange noise, which he thought came from his stomach. He forgot all about it, and went out shooting for two hours. After dinner he heard the noise again. On the next day, while standing in his dining-room, he became conscious of a loud sound in his chest; and his wife, who was three or four feet off, heard it also. During four days it remained audible at a distance. He consulted a medical friend, who discovered valvular disease. Dr. Wilks saw him two weeks afterwards, and kindly sent him to me. His health remained perfectly good. He would not have known that anything was the matter with him, except that when he made any exertion he could feel a vibration in his chest. A loud diastolic murmur was audible over an extensive area. There was no excessive impulse; but the apex beat was situated below the sixth rib; and the heart's dulness extended downwards and outwards for six inches. In this case I think it is clear that, whatever may have been the original cause of the sudden development of the transitory murmur heard at a distance from the patient's body, the valve had previously been diseased. He had, however, been apparently in perfect health: able to ride, shoot, and run as well as ever. The only sudden effort that he remembered making on the day when he first noticed the murmur was that he had lifted his wife out of a high dog-cart; but this he had done many times before.

marked, their intensification by pressure with the stethoscope, and their failing to correspond accurately with the cardiac rhythm, are all valuable points of distinction; but as a matter of pure auscultation, I think that doubt would sometimes be admissible; and as a matter of fact I have occasionally experienced this difficulty, especially when (as in cases of Bright's disease at an advanced age) the presence of either chronic pericarditis or disease of the aortic coats would accord with the other features of the case.

The diagnosis between a presystolic and a diastolic murmur is not generally difficult to those who are well acquainted with the seat and quality of these murmurs respectively. But I have sometimes found it to be far from easy; and a distinguished physician, who has himself written much on the subject of heart disease, has informed me of one case in which he confidently asserted the existence of a presystolic murmur, but in which the aortic valves proved to be unsound, while the mitral valve was healthy. The mistake most likely to happen to the unpractised or careless auscultator is that of supposing the murmur of aortic regurgitation, when it happens to be loud at the apex of the heart, to be a mitral regurgitant bruit. To commit this error is completely to misunderstand the rhythm of the heart in the patient under examination. But I have nevertheless seen it committed more than once. Either no pains at all were taken to determine the period of the ventricular systole; or the radial pulse was employed as a guide to it. Now it has been already stated that in aortic incompetency the radial pulse is often delayed, so as to be almost synchronous with the recoil of the aorta; or, in other words, with the regurgitant bruit. Hence by feeling the wrist in cases of this kind one may easily mistake a diastolic for a systolic murmur.

It still remains to be mentioned that an aortic regurgitant murmur is sometimes hard to detect. I well remember that, when I was a student, I had very great difficulty in hearing the murmur in more than one case in which my teachers spoke confidently of its presence. And I now find that I in my turn discover murmurs which my pupils cannot hear, even when I tell them what to listen for. When such a murmur is once heard, it often seems so distinct that one wonders that one could have overlooked it. In other instances the sound is really very slight, and it is thus drowned by any little noise, although plainly audible at night, or when a ward is very quiet. Lately I had a patient under my care, in whom the existence of an aortic regurgitant murmur was matter of the most lively discussion. I was sure that I had heard it two or three times, but on every other occasion I failed to detect it. After death the valves were found to be obviously incompetent. There is of course no relation between the amount of reflux and the loudness of the murmur.

The diagnosis of mitral disease is far from resting on so satisfactory a footing as that of aortic obstruction and regurgitation. We may first take the comparatively simple case of mitral stenosis. I have already said that a presystolic murmur, when heard at the heart's apex, is pathognomonic of this affection. But we have now to approach the subject from the opposite point of view, and to inquire in what proportion of cases such a murmur is audible. Some years ago I collected for the Guy's Hospital Reports all the instances in which mitral stenosis was found after death during a period of some years. They amounted to forty-seven; and in only seven (or perhaps six) of them had a presystolic murmur been detected during life. It is true that from them a considerable number (fifteen or twenty) had to be subtracted, as having proved fatal soon after admission, or as having been cases of surgical disease or injury, or as having in some other way failed to afford an opportunity for diagnosis. But there still remained at least three cases of mitral stenosis without presystolic murmur, to one in which such a murmur was recognized.

At that time the whole question of presystolic bruits was comparatively a new one; and I thought that, with further experience, the number of undiagnosed cases of mitral stenosis would diminish. I am bound to say that this appears not to be the case. I have not indeed submitted to numerical analysis the observations that have been made since my paper was written; but my impression is that, in the very large majority of the cases in which mitral stenosis is found after death, there is no record of the presence of a presystolic murmur during life. Some observers, I know, hope to reduce this proportion of failures in diagnosis, by the more frequent detection of a short presystolic murmur preceding the systolic murmur of mitral regurgitation. I must confess that my own experience in this direction has not hitherto been very encouraging. In more than one instance in which I thought I had detected such a second murmur, the mitral orifice has been found after death of its natural size.

It remains to add that, even when a presystolic murmur has once been detected, it may often cease for a time to be audible, or even altogether disappear. In the later stages of the disease, when the heart is beating quickly and irregularly, it is almost always absent. Thus, at first there was some difficulty in verifying the correctness of the modern view with re-

gard to the rhythm of presystolic murmurs by post-mortem evidence; and in the majority of cases that have terminated fatally soon after the diagnosis of mitral stenosis, some accidental complication has been the cause of death. Again, when the patient is prostrated by any depressing intercurrent disease, the murmur may become temporarily inaudible, returning with convalescence. Of this Dr. Sutton has related a capital instance.[1] In other cases, no murmur can be heard as long as the patient remains perfectly quiet; but muscular exertion or effort soon makes it audible. Sometimes even making the patient sit up in bed will bring out a presystolic murmur that had a moment before been absent; sometimes it is necessary that he should walk two or three times the length of a ward, or even go quickly upstairs. One can never safely assert the absence of a presystolic murmur when one has examined the patient only in a recumbent posture. It may be added, parenthetically, that in aortic stenosis (the chief other form of obstructive disease at a cardiac orifice), a loud murmur may sometimes be brought out by making the patient run upstairs, although none had previously been audible. I state this on the authority of Dr. Wilks.[2]

From the remarks that have already been made with regard to the so-called mitral regurgitant disease, it will be evident that there can be no question here as to the frequency with which its diagnosis is effected during life. I believe that a systolic murmur, louder at the apex than elsewhere, and audible at the angle of the left scapula, proves the existence of mitral regurgitation; but it is certainly present in comparatively few of the cases that are commonly placed in this category.

There is, in fact, a large residue of cases of valvular disease in which either no murmur is audible at the time of observation, or only a systolic murmur, confined to the apex. These cases constitute the sandy desert of cardiac pathology—not, indeed, unexplored, but with a surface so precarious and shifting as to have hitherto prevented the laying down of roads across it, much less the division of it into territories by fixed boundary lines. As we have seen, the cases in question do not differ at all, so far as stethoscopical evidence goes, from others in which the presence of valvular disease is altogether doubtful. It may be true that, since advanced organic changes in the mitral valve almost always lead to stenosis, the diagnosis of stenosis becomes exceedingly probable in any case which can be shown to be primarily one of organic disease of this valve. But it is precisely here that the difficulty arises; and for such cases I think that the diagnosis of "morbus cordis" is often the most exact that can be given.

I may refer, for example, to a series of cases of fibroid disease of the heart that I have recorded in the Pathological Transactions for 1874, vol. xxv. p. 64. In several of these cases there was a systolic apex murmur; and it is probable that, at least in some of them, the mitral valve was really inefficient, since the fibroid change often invaded one of its fleshy columns. Now, during life, there was nothing to distinguish these cases from those of ordinary "mitral regurgitant disease," and even in the other cases, in which no murmur existed, valvular disease might really have existed, and been latent. Since my cases were published, it has occurred to me that perhaps one positive indication of the presence of fibroid disease of the heart, rather than of any affection of the valves, may be found in its resisting treatment with greater obstinacy. When a considerable part of the wall of the left ventricle has had its muscular substance replaced by fibrous tissue, it appears reasonable to suppose that the remedies which would be useful in a case of valvular disease should prove to be altogether powerless.

I have still to lay stress on the importance of watching, with great care, for the occurrence of those changes in valves already diseased which have already been described, and the recognition of which is so important for purposes of prognosis. The development of incompetency in aortic valves that had hitherto simply obstructed the onward current, the production of stenosis in a mitral valve previously the seat of regurgitation alone, the rupture of the chordæ of a diseased mitral valve, the tearing down of a softened aortic segment, the supervention of acute inflammation in valves long thickened, atheromatous, or calcified,—all these might probably be discovered much oftener than is now the case, were the physician to pay more regard to the probability of their occurrence. Nor should the liability to intercurrent pericarditis, and to the development of changes in the heart's muscular tissue, ever be forgotten by those who would have their diagnosis complete for the post-mortem examination.

Prognosis.—To determine the probable duration of life in a patient affected with valvular disease of the heart, and the chance that existing symptoms may be relieved or removed, is generally very

[1] Lond. Hosp. Rep. vol. iv. 1867–68. The patient was very much weakened by frequent vomiting during the time when the murmur disappeared.

[2] Dr. Walshe taught this clinically twenty-five years ago. See his "Diseases of the Lungs and Heart," 1851, p. 217.—Editor.

difficult; and it can hardly be discussed systematically in an article of this kind, since it requires that all the circumstances of the case should, one after another, be taken into consideration. But some leading points may be briefly stated.

And in the first place, can a diseased valve ever recover its normal structure and functions? In reference to the acute affections of the valves, arising in rheumatic fever or in chorea, some facts have already been adduced which indicate that this is possible. And a further argument in favor of the same view may perhaps be found in the circumstance that in each of the diseases in question a systolic murmur is heard, which in many cases disappears after recovery. If such a murmur, when audible at the heart's apex, be regarded as proof that the mitral valve is affected, it would seem to follow that endocarditis is curable. Such an opinion has, in fact, been recently maintained by Dr. Peacock, who, in an analysis[1] of 146 cases of acute rheumatism that had been under his care, found that "the proportion of cases of recent cardiac complication (which he states to have consisted in endocarditis more frequently than in pericarditis) entirely cured was 41·5 per cent." But the conclusion, of course, depends for its validity upon the question whether the determination of the cause of the murmur is accurate. And this I am not prepared to admit unreservedly.

A valve once affected with chronic disease is no doubt almost always damaged beyond possibility of repair. Thickened and calcified aortic valves can never again become thin and supple.[2] Nor is it probable that a stenosed mitral orifice can become widened. Friedreich has indeed suggested that in young subjects this may not be impossible: but in proof of it he can only refer, in general terms, to cases in which there were at one time symptoms of extreme stenosis, but in which these gradually diminished, and after death the mitral orifice was found capable of admitting two fingers.

There is, however, no doubt that thirty years ago the most practised auscultators of the day condemned, as the victims of organic valvular disease that would soon destroy them, children who have since grown up to be men and women, and who to all appearance enjoy excellent health. It is probable that they attached too absolute an importance to the existence of a murmur, and that they also committed the error of supposing that the louder the murmur, the worse the disease. One cannot insist too strongly on the fact that between these two things there is no relation whatever. The prognosis in the cases under consideration must be based not upon the physical qualities of the murmur, but upon a determination of the degree to which the disease disturbs the circulation; or, if compensation be complete, upon the degree of increased strain thrown upon the heart.

I have already pointed out how compensation is in many cases effected by dilatation and hypertrophy of certain of the heart's chambers. According to Jaksch there is another kind of compensation, consisting in conservative changes in the valves themselves, which absolutely prevent diseases of the valves from producing their natural consequences. When one cusp of the mitral valve is diseased, he imagines that the other may grow broader, and its chordæ may lengthen until it meets its fellow. When one aortic valve is puckered up, the others may gradually become deeper and wider, so as to fill up the gap. The change last mentioned is one which I have myself seen; but it doubtless occurs only in very young patients.

It has already been stated more than once that in valvular diseases of the heart the development of serious symptoms is often very long delayed. Dropsy may first show itself in a person advanced in years, and destroy life in a few months: but the mitral disease which is rightly regarded as the cause of the dropsy may be traceable to an attack of rheumatic fever twenty or thirty years back: and in the interval the patient may either have had excellent health, or may always have suffered more or less from dyspnœa on exertion, which has shown that the heart was defective.

It is a question discussed by almost all writers on Heart Diseases, whether a prolonged existence, and delay in the development of serious symptoms occur in all forms of valvular disease alike, or belong especially to any one group of cases. Considerable interest would indeed attach to the determination of the relative prognosis of the various affections of different valves: and although statistical accuracy is not to be looked for, a general concurrence of opinion on the subject might fairly be expected. The case is not so, however. According to one of the most recent French writers, Jaccoud,[1] stenoses in general are more serious than regurgitations: and mitral stenosis is more so than aortic stenosis. Again, Friedreich, the author of perhaps the latest German monograph,[2] says that "as

[1] Clinical Society's Transactions, ii. p. 221.

[2] The analogy of scleroderma, however, perhaps suggests that even this is not absolutely out of the question.

[1] Traité de Pathologie Interne, tome i. p. 657.

[2] Krankheiten des Herzens, Handbuch der spec. Path. und Ther. 2te Aufl. 1867, p. 282.

a rule the prognosis in obstructive forms of valvular disease is less favorable, and the duration of life shorter, in obstructive than in regurgitant affections." Now, according to all English writers this is absolutely incorrect. Walshe places "the chief valvular derangements in the following descending series on the basis of their relative gravity,—that is, estimating this gravity not only by their ultimate lethal tendency, but by the amount of complicated miseries they inflict:—Tricuspid regurgitation: mitral constriction and regurgitation: aortic regurtitation; pulmonary constriction; aortic constriction." Thus Dr. Walshe regards aortic stenosis as admitting of a far better prognosis than aortic regurgitation: and Dr. Peacock agrees with him, stating that in the former disease life may be prolonged for many years, and a large amount of health and vigor be enjoyed; whereas in aortic incompetency it is very rare to find life sustained for a considerable period. Dr. Peacock, indeed, differs from Dr. Walshe and from most other English writers in believing the prospects of longevity to be actually less in persons who labor under aortic regurgitation than in those who have mitral disease. I confess that I am unable to reconcile these conflicting statements. It is evident that the discrepancy is in great part due to the uncertainty which still attaches to the interpretation both of auscultatory phenomena and of morbid appearances. I have shown that, according to experience at Guy's Hospital, aortic stenosis, without regurgitation, is far more rare than has generally been supposed: and certainly it would not within the last few years have been possible to make any observations that would have allowed of a numerical comparison between its mortality and that of regurgitant disease of the same orifice. The latter disease, however, is undoubtedly a very fatal one. I find from the clinical records at Guy's, that from 45 to 50 per cent. of the patients who have aortic regurgitation die within the comparatively short period during which (under ordinary circumstances) they are allowed to remain as in-patients. But then it is to be observed that the fact of their admittance implies the existence of severe symptoms at the time: and the observations in question are not incompatible with the fact that the disease often exists for a lengthened period before such symptoms show themselves. I have already remarked that changes in the aortic valves, allowing regurgitation, have often been found in persons who have presented themselves for life assurance, or in the dead bodies of those who have been killed by accident. Instances of this kind appear to be fairly comparable with the case, on which Dr. Peacock lays so much stress, of a woman, æt. 76, who died of strangulated hernia, and in whom two of the aortic curtains were completely blended into one, and the orifice reduced to a mere slit, although she was not known to have had any symptoms of disease of the heart. Unless we agree with Dr. Peacock in supposing that disease of this kind always originates in congenital malformation, there is no proof whatever that in the case in question the disease had existed longer than in the examples of unsuspected regurgitant aortic disease which are so common. But while thus criticizing some of the evidence brought forward in proof that aortic stenosis is a less serious disease than aortic regurgitation, I nevertheless believe that this is really the case.

Again, it is very difficult to institute a comparison between the duration of life in mitral stenosis and mitral regurgitation respectively. For, as we have seen, the cases included under the latter designation present no one pathological lesion, but rather a variety of more or less allied conditions. Many cases of mitral stenosis, with marked presystolic murmur, remain under observation for some years, and are admitted into the wards again and again, without the symptoms undergoing any great increase of severity, and without there being at any time reason to apprehend an immediately fatal issue. And on the other hand, it is well known that the systolic murmur of mitral regurgitation may be detected by auscultation for years before any serious symptoms show themselves.

Lastly, I doubt whether any data exist from which one could accurately determine the relative gravity of regurgitant aortic, and of regurgitant mitral disease. For, in addition to other points that have already been noticed, there is between these two affections an important distinction in the fact that one of them is far more constantly traceable to a past attack of rheumatic fever than the other. Hence, while one can often with confidence say, in the case of mitral regurgitation, that the cardiac affection began years before, when the patient had acute rheumatism, one is commonly obliged to refer the commencement of aortic disease to the date when the patient first began to suffer from definite symptoms of heart-disease. Now it is certain that aortic disease sometimes exists for a long time without any symptoms at all; but whether this is the rule or the exception we have no means of knowing.

There is, however, one particular mode of death which appears beyond doubt to occur in regurgitant aortic disease far more frequently than in any other affection of the cardiac valves; and it is one which for many persons has especial terrors,—namely, that in which the fatal termination is sudden. It is a curious

circumstance that the contrary is stated by Corrigan, in the interesting paper which is almost the first that was written on this subject. In permanent patency of the mouth of the aorta, he says, "*the fatal result is never sudden.*" "Under proper restrictions the patient is not only able to lead an active life for years, but is actually benefited by doing so." All recent writers, however, recognize the tendency to the occurrence of sudden death in the disease in question. Thus Dr. Walshe says:[1] "Taken as a group, valvular impediments cannot fairly be cited as frequent causes of sudden death: but there is one among the number, of which the tendency to kill instantaneously is so strong that the fact must always be borne in mind in estimating its prognosis, and that is aortic regurgitation. . . . The manner of death is clearly syncopal: but the immediate mechanism, whether mechanical or dynamic, is difficult enough of comprehension. I have known death take place during the act of walking, of eating, of speaking,—while the patient was emotionally excited, and, *per contra*, at a moment when he was perfectly calm." Further on, Dr. Walshe appears to imply that the liability to sudden death is greater when the heart itself is perfectly healthy than when it presents dilatation and hypertrophy of the left ventricle or other morbid changes. But in this he differs from Dr. Peacock, who says[2] that "in cases in which the heart is most remarkably enlarged, sudden death is yet of common occurrence," and who cites two instances of the kind, in which the hearts weighed 40 oz. and 46 oz. respectively.

With regard to the prognosis of the diseases of the valves believed to originate in injury, all that can be said is that in recorded cases the duration of life has been very variable. Dr. Peacock states that the period of death in the different cases of injury to the aortic valves collected by him was "twenty-one days, three months and a half, thirteen months, two years, twenty-seven months, and three years and a half: and two persons were still surviving after five months and five years had elapsed" in their respective cases. "In the cases of rupture of the mitral valve, the patients lived nine days, and twenty months: and two still survived eighteen months, and two years, after the occurrence of the accident."

TREATMENT. — The prophylaxis of acute affections of the cardiac valves belongs to the treatment of those diseases in which such affections are most apt to arise; and if endocarditis can really be prevented by medicine, this is, in fact, the most important part of the treatment of the diseases in question. But at present I do not know that one can really say any more about it than that rest should be strictly enforced, and that the chest should perhaps be protected from cold by a layer of cotton-wool.

Scarcely less important is the prevention of the development of chronic disease in valves that have once been damaged by acute inflammation. I have already adduced facts which tend to prove that endocarditis not rarely subsides without leaving any injurious effects behind it; in particular, that a large proportion of the cases of rheumatic inflammation of the aortic valves in women must terminate in the restoration of the normal structure of the valves. The comparative immunity of the female sex from the more remote changes which so frequently arise in the male sex can only be ascribed to the fact that women lead less active lives than men, and are not compelled to endure such continuous exertion, or to make such violent muscular efforts. The plain inference is, that in either sex the way to prevent chronic disease of the valves, after endocarditis in rheumatism or chorea, is to keep the patient for many months—or even some years—as perfectly as possible at rest; to insist on abstention from violent exercise, athletic sports and games, of all kinds; to direct the choice of a light, sedentary employment, and to urge the avoidance of all emotional excitement. General hygienic conditions should at the same time be carefully attended to. I think, too, that it may hereafter be shown that medicines are useful. I have pointed out how the anatomical characters of chronic disease of the valves differ from those of acute endocarditis; that the vegetations disappear, but that the edges of the valves become thickened and fused together. Surely it is possible that iodide of potassium, mercury, or arsenic, may be able to arrest or prevent these changes, as much as those which belong to certain skin diseases, or the chronic inflammations of parts accessible to the sight or touch of the surgeon.

Similar principles must be applied in the endeavor to prevent those forms of valvular disease which are from the first of gradual origin. A very large proportion of the cases of aortic regurgitant disease that occur so commonly in laboring men past middle life, are due to the fact that these men have gone on with work involving straining efforts, which can with safety be made only by younger individuals, whose tissues are still elastic and supple. Dr. Peacock and Dr. Allbutt have indeed shown that such diseases of the cardiac valves frequently occur at an earlier period of life than has generally been supposed; but even then they are

[1] Op. cit. p. 390.
[2] Croonian Lectures, p. 108.

perhaps favored by some particular diathetic condition, or by habitual excessive indulgence in alcoholic drinks, which promotes degenerative changes in the tissues. It may hereafter be possible for the physician to select certain individuals as especially liable to suffer from the harder kinds of labor, and to recommend for them less arduous employments. Among the higher classes, again, chronic disease of the cardiac valves appears very frequently to be due to men forgetting that they are advancing in years, and to their continuing to take violent exercise long after they have ceased to be fit for it. This is especially apt to occur in professional men, whose habits are generally sedentary, and who, during an occasional holiday, often run great risks. The physician should always be on the lookout for the earliest signs of tissue-degeneration in such persons, and should be ready to warn them of the necessity that they should avoid too great exertions or straining efforts. It is no longer believed that the signs in question are an early arcus senilis, and the fact that the hair has turned prematurely gray; and I am myself inclined to doubt whether tortuosity of the temporal arteries, or an apparent rigidity of the radial arteries to the touch, is to be much relied on, as indicative of degeneration of those vessels; but, taken with other points, they are probably of value; and it seems that the sphygmograph may here lend very valuable assistance.

Even when valvular disease is fairly established, the prophylactic measures already referred to by no means cease to be applicable. Probably such disease is almost always *progressive;* and it is, moreover, liable to become complicated at any period of its course by the supervention of acute endocarditis.

But the treatment of diseases of the cardiac valves, properly so called, reduces itself to the treatment of their effects. To these we must therefore refer in brief detail.

1. Very little, and perhaps nothing, is known of any effectual treatment for the contamination of the blood with morbid materials, which is so apt to occur in the more acute forms of valvular disease, or for the occurrence of embolism in the larger vessels. Quinine would seem to be indicated in the former condition, and may perhaps be of some service; but Lancereaux observes that its failure has often been demonstrated in cases that had been mistaken for ague, and had therefore been treated with this drug. The mineral acids are recommended by Friedreich. I am not aware that any evidence is to be obtained as to the use of the sulphites or hyposulphites, as recommended by Polli in septic conditions, but I should conceive that there is, at any rate, more chance that they might be useful in the cases under consideration than in the specific fevers against which they have chiefly been employed. Cases in which "typhoid" symptoms occur, with hemorrhages into the skin and mucous membranes, &c., are probably of necessity fatal; and it is almost useless to administer the ammonia, ether, and musk, which are generally recommended, and which at once suggest themselves to the mind as the drugs that can be most appropriately given.

When there is evidence of the occurrence of embolism in any particular artery, it is possible that the administration of ammonia, as suggested by Dr. Richardson,[1] may favor the solution of the coagulum—if indeed he is right in attributing success to this treatment in cases of fibrinous deposition within the heart. The plan which he recommends is the administration of ten-minim doses of the liquor ammoniæ in iced water, every hour, with three to five-grain doses of the iodide of potassium every alternate hour.

2. The changes which diseases of the cardiac valves induce in the circulation of the blood, and in the several chambers of the heart, are capable of being modified in a very remarkable degree by various medicines and modes of treatment; and to these we must now turn our attention, following as far as possible the same order which was adopted in the account of these changes given in pages 394 to 404.

In cases of aortic regurgitation, so long as the state of the ventricle is such as perfectly to compensate for the valvular defect, medicinal treatment is scarcely applicable. Patients admitted into an hospital sometimes lose all their symptoms as a consequence of the rest which they obtain, and which is so essential to them. The avoidance of all violent or straining efforts should in fact be insisted on in this, even more than in other forms of cardiac disease, on account of the marked tendency to sudden death, which must always be borne in mind.

For the less severe effects of aortic regurgitant disease, the slighter degree of malaise and discomfort caused by it, senega is the common remedy. It is difficult to say how this drug acts; and as ammonia is generally given with it, this has been supposed to be the really efficient remedy. I have, however, repeatedly prescribed it alone, and patients have sometimes declared that it has given them distinct relief. I am therefore disposed to believe that it is of value, and the more so, as the late Dr. Barlow (a physician of much experience in such matters) used to teach that in many cases only moderate

[1] Med. Press and Circular, Nov. 20, 1872.

doses could be borne. The dose usually given is half an ounce to an ounce of the infusion, with or without half a drachm or a drachm of the tincture, and perhaps the same quantity of the aromatic spirits of ammonia, or five grains of carbonate of ammonia.

When compensation fails in aortic regurgitant disease, we have seen that effects are developed which are identical with those that occur in mitral disease. They require the same treatment, which I shall describe in the next paragraph.

In the treatment of a case of "mitral disease"—using that term for the moment in its widest sense—the primary point is the due regulation of the contractions of the left ventricle, for which we have in *digitalis* a remedy of wonderful power. Within the last few years a great change has taken place in our views as to the action of this herb, and our knowledge is very much more accurate than it formerly was. The older opinion was that it enfeebled the power of the heart,[1] and therefore that dangerous effects might in certain cases follow its administration, from its tendency to cause fatal syncope. It is true that Dr. Withering in the last century stated it to be most useful in those cases of dropsy in which the pulse was feeble or intermitting, declaring also that it seldom succeeded in men with a tight and cordy pulse. But its good effects in such cases were attributed to its diuretic action, not to its having any power of strengthening a feeble heart.

Within the last few years, however, it has been demonstrated that the action of digitalis on the heart is in fact that of a tonic. The proofs of this are varied. In cold-blooded animals, in which the cardiac pulsations can be watched after exposure of the organ, digitalis causes spasm of the left ventricle, beginning at isolated points in its wall, and finally affecting its whole substance, so that its beats cease, and it remains rigidly contracted and white. In conjunction with Dr. Stevenson, I some years ago performed a number of experiments on frogs, in which this result was uniformly observed.[2] In the higher animals it is less easy to study directly the action of digitalis on the heart, but according to Fothergill,[3] Handfield Jones and Fuller have noticed similar effects as regards the state of the heart after death in mammals.

The present doctrine with regard to digitalis, then, is that it strengthens the heart's contractions. It is true that when very large doses are given, the pulse may become weak, frequent, and intermittent; but this is supposed to be due to the fact that the ventricle is in a state of spasm, and therefore that its beats are imperfect, and throw but a small quantity of blood into the arteries.

Thus the cases of heart disease in which digitalis is most useful are those in which the organ beats feebly and irregularly, in which a condition of "asystolie" exists, and in which the pulse presents the sphygmographic characters indicated at p. 745. In such cases the action of the remedy is to diminish the frequency of the cardiac pulsations, to make them regular, and to increase their force.

Among affections of the cardiac valves, "mitral regurgitant disease" is that one which most commonly presents the indications for the administration of digitalis; and in a large proportion of cases of this kind, great relief is afforded by the remedy; the symptoms may for a time be entirely removed, and the patient restored to a state of apparent health. On the other hand, it is often useless and perhaps injurious in cases of mitral stenosis; for the left ventricle in the earlier stages of this affection generally contracts regularly and with due force, as is apparent from the normal character of the pulse. At a later period in the course of mitral stenosis, digitalis is often very useful; but the physical characters of the diseases are then less distinctive; it is often difficult or even impossible to determine its exact nature. Again, in aortic regurgitation, when the hypertrophied ventricle is carrying on the circulation vigorously, digitalis often aggravates all the symptoms; and if the patient should die suddenly, it is liable to the charge of having caused the fatal result, a charge which cannot be refuted, and is probably often justly made against the drug. But Dr. Ringer has shown that the existence of aortic disease does not contraindicate the use of digitalis, if the symptoms suggest its administration. When there is dilatation of the heart (rather than hypertrophy), and the pulse is feeble, frequent, fluttering, and (above all) irregular, it may be given with a fair expectation that it will afford relief.

The dose of digitalis is a matter of some importance; a drachm of the infusion is enough to begin with, or five or ten minims of the tincture. According to Dr. Fothergill, the injurious effects of digitalis in aortic disease, with hypertrophy of the left ventricle, may be avoided by employing very minute doses, which will in such cases do as much good as is produced under ordinary circumstances by larger quantities of the remedy.

It is doubtful whether any other remedies are capable of exerting the same ac-

[1] Pereira's Mat. Med., 4th ed., 1855, vol. ii. p. 536.

[2] Proc. of the Roy. Soc. 1865; Guy's Hosp. Rep. 1866.

[3] "Digitalis: its mode of action and its use," 1871.

tion as digitalis on the diseased human heart. Dr. Stevenson and I found that squill and two species of helleborus (*H. viridis* and *niger*) produced the same peculiar effects in the healthy frog. *Veratrum viride* is often supposed to resemble digitalis in this respect; and in America it has been largely used to diminish the frequency of the heart's beats. But in frogs its action is the very opposite of that of digitalis; it rather resembles aconite, paralyzing the heart, which, when it stops, is dilated and of a deep purple color.

The treatment for the *backward* effects of diseases of the valves of the heart must of course aim at reducing the increased tension in the pulmonary and venous systemic vessels, upon which these effects depend. And there are two principal ways in which this can be done. The first is the removal of a portion of the venous blood by venesection, leeches, or cupping. Now, if we take into consideration the fact that blood is forced into the veins from the capillaries in a continuous stream, we shall not at first suppose that much benefit is likely to accrue from the abstraction of a few ounces of blood from one part of the venous system. It seems like taking a cupful of water from a pail that is running over with the supply from a spring. We cannot help imagining that the veins will almost instantly become again distended. But there is abundant evidence to show that such a supposition is erroneous. Thus the hæmoptysis which accompanies pulmonary apoplexy often relieves the patient's breathing for several days or even weeks; and nausea and vomiting, due to congestion of the stomach, are frequently removed for a considerable time by an attack of hæmatemesis. It is clear that the relations, as regards tension, of the different parts of the circulating system can be much more steadily maintained than one would at first sight have imagined. Equally decided are the therapeutical proofs of the same fact. The withdrawal of a small quantity of venous blood is often attended with the most beneficial results in cases of heart disease. Perhaps the most striking example that I can cite is one, recorded by Dr. Dickinson,[1] of a man who had ruptured almost all the chordæ of the posterior flap of the mitral valve. "This patient was frequently relieved temporarily by the abstraction of blood. He was frequently cupped, always with apparent relief of the dyspnœa and distress. Towards the close of his sufferings, when, though there was much cardiac action, the pulse was nearly imperceptible, and the patient was approaching a condition of collapse, with much dyspnœa and blueness of the face, eight ounces of blood were taken by venesection, with immediate and decided relief, the pulse recovering itself as the blood flowed, while the distress of the patient was much lessened. The improvement, however, was only temporary. The patient died the following night."

The extreme gravity of the lesion in this case seems to render it worthy of being quoted. If the removal of blood could give relief when one-half of the mitral valve "had lost all valvular action, and swung uselessly from its base," there is hardly any case in which one need despair of its doing good. In the ordinary forms of valvular disease it is often useful, and the relief afforded by it is sometimes maintained for several days, or even weeks, so as to allow time for the operation of other remedies. The application of leeches to the epigastrium relieves the sickness and nausea due to congestion of the stomach; probably they would be still more useful if applied near the anus.

The other method of relieving the engorged pulmonary and venous systemic circulation is by removing, not blood itself, but its watery part alone; in other words by giving purgatives and diuretics. Among the former remedies, the hydragogues are of course to be preferred; jalap, or even elaterium, scammony, salines, &c. As regards diuretics, it has already been observed that one of the principal indications of the favorable action of digitalis is its increasing the flow of urine, sometimes to an enormous extent. Whatever view may be taken of the theory of its action, there is no doubt about the fact. Other remedies which are supposed to act as diuretics in the diseases under consideration are squill, juniper, broom, and cream of tartar. Copaiba is sometimes very useful. I have notes of one case of mitral disease which had previously resisted various kinds of treatment, and in which ascites and anasarca rapidly vanished under the administration of a simple copaiba mixture. I shall never forget the gratification of the patient as the loops of string that held his trousers together soon became unnecessary, and the buttons themselves had to be moved again and again, in adaptation to the rapidly-decreasing girth of his belly. Dr. Wilks has recently found the resin of copaiba no less effectual, as it is certainly more pleasant.

3. The third group of effects of disease of the cardiac valves—the symptoms subjectively experienced by the patient—are frequently capable of great relief by medical treatment, but too often resist all the physician's efforts, and make the termination of a case of this kind almost more distressing and painful than that of any other disease.

[1] Path. Trans. xx. p. 151.

The obvious remedies for dyspnœa, palpitation, and the sense of pressure and weight in the epigastrium, are the ethers and ammonia, especially when combined with digitalis, if the nature of the disease should be such as to indicate its employment. The application of a large belladonna plaster to the cardiac region often gives considerable relief to local pain and to palpitation.

Hyoscyamus is commonly given as an anodyne in these cases; but I have not seen it do very much good. Opium is generally said to be inadmissible, or to be used only with great caution. On the other hand, it would appear that the subcutaneous injection of morphia may be employed with safety, and with the most marked results. Its use has been especially advocated by Dr. Allbutt.[1] He uses the hydrochlorate, in doses of one-tenth to one-third of a grain. It is especially useful, he says, in cases of mitral regurgitation, "when the head is full of venous blood, and distress and stupor seem striving together. An injection of morphia three or four times a week, by tranquillizing the heart, and allowing the circulation to recover its freedom, sets free also the organs that are oppressed. . . . Directly and immediately the injection seems to affect the chest almost alone. The face generally becomes less turgid, and its expression calmer. The heart becomes tranquil and rhythmical. . . . The insufferable præcordial distress ceases. . . . The quick, shallow, anxious cardiac dyspnœa gives way to a deeper, slower, and easier movement. . . . The patient, who has been tossing in misery, feels the first tranquil sleep he has enjoyed for weeks."

The attacks of angina-like pain, which form so important a part of the symptoms in many cases of aortic regurgitation, require essentially the treatment of neuralgias. I have more than once found the regular administration of arsenic able to prevent their recurrence. The paroxysms themselves are often arrested by the inhalation of ten drops of nitrite of amyl, or of a few whiffs of chloroform; or again by the subcutaneous injection of morphia. In one case that I saw—in which all these were used in succession—the patient preferred the morphia, as giving him the highest amount of relief.

ATROPHY OF THE HEART.

By W. R. Gowers, M.D.

Synonym.—Phthisis of the Heart (old writers).

Definition.—Diminution in the size and weight of the heart, consequent on diminution in the amount of muscular tissue contained in its walls. Of these characters the diminution in weight is the most important. An atrophied heart, according to the common use of the term, is one the weight of which is less than the average weight for a person of the same stature. It is said that, in very rare instances, a heart, the total muscular tissue of which is lessened, and the weight below the normal, may be larger than natural, owing to the dilatation of its cavities. The occurrence of such instances is, by some authorities, denied. If they occur, dilatation is their conspicuous feature, and they come more accurately under that head. Diminished bulk remains a character of those forms of atrophy which may most conveniently be considered under this designation. On the other hand, the muscular tissue of the heart may be lessened in quantity, may have undergone atrophy, when there is increase of other elements in the cardiac wall. In such cases the weight of the heart is, as a rule, not diminished, and these instances are considered under the head of the special degenerations. Only those rare examples will be here alluded to in which the weight of a heart so changed is less than normal.

History.—The important functions always attributed to the heart rendered its atrophy a more anomalous condition, in the eyes of the earlier observers, than its enlargement. Accordingly we find that this condition early attracted attention. Pliny states that the kings of Egypt noted its occurrence. Riolanus alluded to it, and ascribed it to deficiency of the pericardial fluid. A well-marked case was recorded by Soumain at the beginning of

[1] Practitioner, iii. p. 342.

the last century.[1] Senac, in 1749, described it carefully in his treatise on the heart,[2] which probably remains the longest monograph yet written on cardiac anatomy and pathology. Allan Burns, in 1809, described some very characteristic examples.[3] It is not mentioned by Corvisart, who wrote nearly at the same time. Mérat, in 1813,[4] alluded to several instances which he had seen, and Bertin, in 1824, gave a full account of it, while by his editor, Bouillaud,[5] varieties were subsequently discriminated, which have since been recognized by most writers on the subject.

Varieties.—Forms of cardiac atrophy have been distinguished corresponding to the varieties of cardiac hypertrophy. Thus, reduction in the weight of the heart due to mere attenuation of the walls, the cavities remaining of normal size, was termed by Bouillaud, *simple atrophy*.

Reduction in size of the heart, with diminution in the size of its cavities, so that they still bear the normal proportion to the heart, is the *concentric atrophy* of Bouillaud and Walshe,[6] the *simple atrophy* of Hayden.[7]

Attenuation of the cardiac walls and diminished weight of the heart, with increase in the size of the cavities, is the *eccentric atrophy* of Bouillaud, Förster, Walshe, and others. These cases, as just stated, came more properly under the head of dilatation. Hayden applies the term "eccentric atrophy" to a condition of heart, examples of which must be very rare, in which the walls are attenuated, the whole heart smaller, but the cavities larger than normal. As "concentric atrophy" he classes hearts which are smaller than normal, have the walls relatively thickened, and the cavities reduced in capacity. This variety was described by Mérat in 1813. It may be doubted whether either of these two varieties has any real existence: they probably represent only states of contraction or relaxation in atrophied hearts. Chomel distinguished two varieties according to the cause of the atrophy—the *congenital* and *accidental*.[8]

[1] Relation de l'ouverture d'une femme presque sans cœur. Paris, 1728.

[2] Traité de la Structure du Cœur, de son action et de ses maladies. Paris, 1749, tom. ii. p. 393.

[3] Allan Burns, Observations on Diseases of the Heart. Edinburgh, 1809, p. 110.

[4] Dictionnaire des Sciences Médicales, Art. Cœur.

[5] Traité clinique des Maladies du Cœur. 2ieme edition. Paris, 1841.

[6] Diseases of the Heart and Great Vessels. Fourth edition. London, 1873, p. 276.

[7] Diseases of Heart and Aorta, 1875, p. 585.

[8] Dictionnaire en 30 volumes.

[Allied to cardiac atrophy, is *atony* of the heart; tending, of course, towards atrophy. This is met with, sometimes, as a result of overwork, producing cardiac exhaustion. During the civil war in the United States, cases of this kind were observed and reported upon, about the same time, by Drs. Stillé, Da Costa, and myself,[1] as seen and treated in U. S. General Hospitals in Philadelphia. Soldiers who had been (particularly in the "peninsular campaign" of McClellan in Virginia) exposed to severe over-exertion in marching, with deficiency both of rest and food, were rendered unfit for duty, without evidence of any organic disease. In these cases, the pulse was small, abnormally rapid (85 to 100 beats per minute) when quite at rest, and greatly accelerated (up to 120 or 130) even by slight exertion, such as walking slowly across a room. Any considerable effort would cause dyspnœa and general distress. On physical examination, the impulse of the heart was found to be feeble. Dulness of resonance upon percussion was not unusually extended. The sounds of the heart were not altered, except in the diminution of the duration and force of the first sound, making it more than normally like the second sound.

From the absence both of symptoms and physical signs to prove the existence of any ordinary form of heart disease, some of these patients were, under medical inspection, suspected of malingering. When sent back to duty, however, a short time sufficed to show their real disability. Rest for a considerable period with good food and tonics resulted in gradual improvement. No fatal case occurred to give opportunity for autopsy.

"Irritable heart" is the expression preferred by some who have studied these cases, to describe their condition. Myers,[2] Parkes, and others have noticed in the soldiers of the British army a greater tendency to functional disorders of the heart than exists in the same class of men in civil life. Faulty accoutrements are reasonably blamed for this. Some of the cases described by these authors recall the history of those just mentioned, as examples of cardiac exhaustion and atony.

It is not hard to account for the pathogeny of such an affection. Increase of the work imposed upon the muscular tissue of the heart is familiarly known, under ordinary conditions, to produce hypertrophy. This follows the general law of muscular exercise and nutrition. But, when overwork of the heart is compelled, as by rapid marching, with accoutre-

[1] Amer. Journal of Med. Sciences, July, 1864.]

[2] Prize Essay on Diseases of the Heart among Soldiers. London, 1870.]

ments, etc., to carry, for many days, perhaps weeks, together, and, at the same time, not only little chance is left for sleep, but food is deficient in quantity and quality, instead of increase of power, exhaustion must result. The time required for recovery of the tone and energy of the heart, under such circumstances may be extended through several months.—H.]

CAUSES.—Smallness of heart may be a congenital or an acquired condition.

A. *Congenital* atrophy is usually well marked. The heart of an adult otherwise free from disease may not exceed that of a child six or seven years old, as in an example mentioned by Allan Burns. The immediate causes of this condition are unknown. Hereditary influence has not, hitherto, been traced. It is said to be more common in women than in men. The subjects of it may be in other respects well formed, but sometimes it has appeared to be part of a more general arrest of development, shown by a childish aspect and defective development of the sexual organs. Parrot[1] doubts the congenital nature of these cases, and believes them to be due to a simultaneous arrest of the growth of the heart and of the sexual organs, occurring at puberty.

B. *Acquired* atrophy may be the result of general or local causes. The chief *general* causes are *chronic wasting diseases*, in which the heart frequently undergoes diminution in size. This may occur in cancer, phthisis, syphilis, chronic suppuration, diabetes. According to the statistics of Quain,[2] the heart is small in about half the cases of phthisis, and the diminution in size is rather more frequent in women than in men. Out of 171 cases, it was small in 53 per cent. of the males, in 67 per cent. of the females. There is no evidence of any special influence exercised by these diseases on the heart. The organ apparently wastes in common with the rest of the body, in consequence of the defective nutrition.

The *local* causes are such as influence directly the nutrition of the heart. *Narrowing of the coronary arteries* is said to be an occasional cause. The influence of this condition is to be more distinctly traced in the production of local degeneration. Walshe, however, regards the influence of pressure in causing local atrophy as due to its effect on the blood supply.

Compression of the heart is apparently, in some cases, a cause of its atrophy. The heart has been found small in long-continued *pericardial effusion*, and the condition has been compared to the contraction of a lung in long-continued effusion into the pleura. *Pericardial adhesions* have been supposed in some cases to have caused cardiac atrophy. The association of the two conditions was first pointed out by Chevers.[1] Hypertrophy and dilatation are more frequent consequences. Kennedy[2] found atrophy in only five out of ninety cases of pericardial adhesion without valve disease. The contraction of tough lymph, resulting from pericarditis, has in some cases been associated with very distinct atrophy of the subjacent portion of the heart.[3] Walshe corroborates this, but believes that the effect is due to pressure upon the arteries. *Compression by fatty tissue* sometimes leads to atrophy of the muscular fibres, especially when the fat is infiltrated among them. The instances of this change in which the heart is smaller than the normal are very rare. Wilks and Moxon mention such a case as an example of "fatty atrophy." The heart weighed only 5½ oz.

Local atrophy, affecting one part of the heart, is due most commonly to the last-described condition, to local infiltration with fat. Occasionally, the limited position of contracting lymph, or narrowing of one coronary artery, may have the same effect.

PATHOLOGICAL ANATOMY.—A heart the subject of atrophy is, as already stated, lessened in weight. The heart of an adult may weigh only six, five, or even four ounces. Quain mentions an instance of the heart weighing only 1 oz. 14 drs. in the case of a girl aged fourteen, who died of phthisis.[4] Its size is also lessened. The circumference at the base may be only six inches. Chomel has recorded an instance in which the heart of an adult did not exceed in size a hen's egg. The thickness of the walls depends chiefly on the condition of the heart, whether contracted or relaxed. The degree of contraction may be estimated by the size of the cavity. In cases of acquired atrophy almost all the adipose tissue has disappeared from the surface, on which the vessels stand out conspicuously. There is often serous infiltration of the fibrous tissue from which the fat has been removed. The texture of the heart may be little changed, or it may be pale in color and softer than natural. On the other hand, it may be dark, dense, and tougher than natural. The change depends on the presence and form of degeneration, whether fatty or fibroid, partly also on the accumulation of pigment granules

[1] Dictionnaire Encyclopédique des Sciences Médicales, 1876, art. Cœur.

[2] Lumleian Lectures, 1872. Abstract in Lancet, vol. i. p. 426.

[1] Guy's Hosp. Reports, vol. vii.

[2] Edin. Med. Journal, 1858.

[3] An observation of this kind was recorded by Malpighi.

[4] Lumleian Lectures, loc. cit.

within the fibres. The microscope shows the primitive bundles to be lessened in size. The fibres are often fattily degenerated; their striation is lessened, sometimes indistinguishable.[1] The fibrous tissue between the bundles may be increased in quantity. Occasionally, especially in the old, brownish pigment may encircle the nuclei of the fibres, or be uniformly distributed through their substance. When it occurs, the pigmentation is usually generally distributed through the heart, and gives its substance a reddish-brown tint. Rindfleisch[2] has described it as a special form of atrophy —"brown atrophy." Friedreich believes that the pigment is derived from the coloring matter of the muscle.

Associated conditions, causing the atrophy, may coexist. The various general conditions, cancer, phthisis, &c., may be present. Pericardial changes, effusion, lymph, plates of calcification, fatty accumulation, may compress the heart, or there may be from some cause obvious reduction in size of the coronary artery. The pericardial fluid is, according to Bamberger, often increased in quantity as a consequence of the cardiac atrophy.

Symptoms.—The physical signs of atrophy depend on the lessened bulk and diminished force of the heart. The extent of dulness, especially the deep dulness, is smaller than normal. To be significant the diminution must be independent of emphysema or any lung condition obscuring the cardiac dulness. The impulse is weak, and felt over a small area. The sounds may be lessened in intensity, or they may be unchanged. The latter has been the case in Walshe's experience. The pulse is small, the patient weakly. When due to a local cause the symptoms of the local causative condition, pericardial effusion, &c., are often present. Palpitation, dyspnœa, and dropsy, are said to occur in cases of acquired atrophy from local malnutrition. The quantity of blood remains unchanged, and the small heart obstructs the circulation. When due to a general state, the heart suffers in common with the blood and the rest of the system, so that the special failing is unnoticed.

The general conditions associated with atrophy of the heart were, in part at least, attributed by the earlier writers to the influence of the cardiac state. Phthisis especially was believed to be entirely due to the small size of the heart, so often found associated with it. It is customary now, as already stated, to regard the small size of the heart as secondary to the general state, and to attribute to it no causative influence.

Diagnosis. — In determining, post mortem, the existence of atrophy, weight should be taken as the test. The error of mistaking contraction for atrophy will thus be avoided. Burns suggested, as a means of avoiding the same error, a comparison between the size of the heart and of the pericardium. The size of the body should always be taken into consideration. It is rarely that atrophy of the heart can be diagnosed during life. It may be suspected when a weak impulse and diminished dulness coincide with signs of cardiac failure and with some recognized causal condition.

Prognosis. — Little can be done to remedy the condition, even when its existence is recognized. The prognosis is therefore unfavorable, but it is always subordinate to that of the condition to which the atrophy is secondary.

Treatment.—The treatment is in the main that of the causal state. In general wasting diseases the atrophy of the heart corresponds to its diminished use, and needs no special treatment beyond general tonics, cod-liver oil, nux vomica, &c. When secondary to local changes, little can be done by treatment beyond the removal as far as possible of the fluid pressing on the heart, or the diminution, by dietetic management, of accumulations of fat.

[1] The "yellow atrophy" of Rindfleisch is fatty degeneration.

[2] Pathologische Gewebelehre, 1875, p. 126.

HYPERTROPHY OF THE HEART.

By W. R. Gowers, M.D.

Synonyms.—Enlargement of the Heart, Dilatation of the Heart (old writers); Active Aneurism (Corvisart); Uniform Enlargement of the Heart, distinguished from dilatation (Allan Burns); Hypersarcosis Cordis (Lallemand).

Definition.—An overgrowth of the muscular tissue which forms the walls of the heart. Besides muscular tissue the heart contains connective tissue and adipose tissue. An increase in either of these constituents may be, and has been, spoken of as an element in cardiac hypertrophy. Thus "fatty hypertrophy" and "connective tissue hypertrophy," or "false hypertrophy," of the heart have been described. It seems more in accordance with the nomenclature applied to other organs to consider these changes as allied to degenerations, and to confine the term "hypertrophy" to increase in the muscular tissue of the heart. Increased thickness of the endocardium and pericardium, which often coexists with muscular hypertrophy, and is sometimes regarded as part of it, is described separately in the articles "Endocarditis" and "Pericarditis."

History.—The earliest allusions to enlargement of the heart appear to be those of Nicolaus Massa in 1559[1] and of Vesalius. Enlargement with thickening of the walls was described in the seventeenth century by Albertini, by our own countryman Mayow, and by Blancard. Its origin in overwork due to obstruction in the circulation was clearly pointed out by Mayow, who in 1674 described the dependence of hypertrophy of the right ventricle on mitral constriction.[2]

Vieussens[1] in 1715 alluded to the origin of hypertrophy of the left ventricle in the overwork caused by constriction of the aortic orifice, and the effect of obstruction in causing enlargement was systematically described by Senac in his treatise published in 1749.[2]

Enlargement from overgrowth without dilatation was mentioned by Morgagni[3] in 1779, by Burserius in 1798,[4] and later by Corvisart in 1806, and distinguished by Allan Burns in 1809, who recorded an example of a heart "weighing several pounds, in which the cavities were not more capacious than natural." Corvisart gave a clear description of the various forms of hypertrophy with dilatation, and recognized the frequency with which the left ventricle is affected. Although he mentioned the occurrence of hypertrophy without dilatation, he did not include it in his account of the forms of enlargement,[5] but described all enlargements of the heart as "aneurisms," classifying them as "active" or "passive," according as there was or was not hypertrophy. Bertin, in a memoir read before the Académie des Sciences in 1811,[6] pointed out

[1] Nicolaus Massa, Anatomiæ Liber Introductorius. Venice, 1559, p. 56.

[2] "Inasmuch as the blood, on account of the obstruction, could not pass freely into the left ventricle, it necessarily happened that the vessels of the lungs, and also the right ventricle, were distended with blood; as a consequence the heart, particularly the right ventricle, would have to contract more violently, in order that it might as far as possible propel the blood through the lungs on to the left ventricle. This again explains why the walls of the right ventricle were so strong and dense, since this chamber, being submitted to more violent action, would be enlarged beyond the rest." Mayow, Tractatus medico-physici, Oxonii, 1674. De Motu Musculari, cap. vii. The translation is that of Cockle, On Insufficiency of the Aortic Valves. London, 1861.

[1] Traité du Cœur, 1715.

[2] Traité de la Structure du Cœur, de son action et de ses maladies, par M. Senac. Paris, 1749. Tom. ii. p. 408.

[3] "Ventriculus dexter corveam quidem secundum naturam, sed crassissimas parietes habebat." De sedibus et Causis morborum. Epist. xvii. art. 22. See also Epist. xxix. art. 20.

[4] The Institutions of the Practice of Medicine, by J. Baptist Burserius, of Kamfeld, 1798. Translated by Cullen Brown. Vol. v. p. 312. Edinburgh, 1803.

[5] This accounts for Laennec's assertion that the occurrence of hypertrophy without dilatation escaped the notice of Corvisart. Bertin pointed out that the condition is described by Corvisart in a case of aneurism of the aorta. "The left ventricle, without being so dilated, had much stronger and thicker parietes than usual." On Diseases of the Heart, Hebb's Translation, p. 283.

[6] Mem. de l'Académie Royale des Sciences, 1811.

the special character of hypertrophy and its isolated occurrence. It was also carefully distinguished by Kreysig in 1816.[1] But in France the nomenclature of Corvisart continued in use by Mérat, Cloquet,[2] and Cruveilhier until, and indeed long after, the publication of Bertin's treatise on diseases of the heart[3] in 1824 gave currency to his distinction of the "concentric," "simple," and "eccentric" forms of hypertrophy. Bertin also demonstrated by microscopical examination that the increase of the heart's substance in hypertrophy depends on an overgrowth of muscular tissue, and also endeavored to show, by a chemical examination of the tissue of the two ventricles, that the quantity of fat in the hypertrophied muscle was less than in the normal portion.[4] He also ably vindicated hypertrophy from some of its supposed consequences.

Avenbrugger in 1763 first employed percussion as a means of ascertaining and estimating enlargement of the heart. The example was followed by Corvisart, who translated Avenbrugger's work. Bertin advocated auscultation as a means of distinguishing the "concentric" and "eccentric" forms. The alterations in the heart-sounds in hypertrophy were, however, first accurately stated by Laennec.[5]

VARIETIES.—The hypertrophy may be *general*, when each portion of the heart is affected, or *local*, when only part of the heart is changed. When the result of the change is a simple increase in the thickness of the wall, without any change in the size of the cavity, the hypertrophy is called "*simple*;" when there is dilatation of the cavity as well as hypertrophy of the walls, the hypertrophy has been termed "*eccentric*." "Hypertrophy with dilatation," or "dilated hypertrophy" are other names which have been applied to this condition. If, on the other hand, the cavity is lessened in size, the hypertrophy has been termed "*concentric*." The existence of this form is doubtful; it is probable that the supposed permanent reduction in the size of the cavity is merely the result of a strong contraction. "Mixed" hypertrophy was the designation given by Bertin to the condition in which one part of a ventricle is thinned and another thickened.

CAUSES AND PATHOLOGY.—A. *Predisposing Causes.*—Strictly speaking, hypertrophy of the heart cannot be said to have any morbid predisposing causes. It is a healthy reaction against a morbid influence, and the conditions which permit its occurrence are those of health. Every divergence from a state of health, which does not immediately excite hypertrophy of the heart, tends to hinder its occurrence. The only general or distant morbid states which are concerned in its production are the antecedents of its exciting causes, and these cannot, strictly, be regarded as "predisposing." Hereditary taint, sex, and age influence the occurrence of the exciting causes of hypertrophy, and render the condition twice as frequent in males as in females (Walshe), and frequent in proportion to age, because men are by occupation and exposure liable to the causes of hypertrophy more than women, and hypertrophy is frequently the result of degenerative changes, the tendency to which increases with age.

Four conditions of health may be considered as especially predisposing to hypertrophy.

(1) General nutritive energy of the system. This influence is shown in the tendency of the normal tissue elements to increase, under certain local stimuli; its defect by their tendency to waste, to degenerate, and give place, under the local nutritive stimulus, to tissue elements of lower vital capacity. This influence is greater in the young than in the old. Its effect in determining the occurrence or the degree of hypertrophy is masked by the greater frequency and greater force of the causes of hypertrophy in later life. It is seen, however, in the rarity with which considerable hypertrophy is developed in old age.

(2) Nutritive quality of blood. The influence of this condition is obvious, and is seen in the distinct increase in hypertrophy which often follows the administration of hæmatinics, as iron, and a good supply of food.

(3) The supply to the cardiac walls of a due quantity of blood. The force of the circulation within the cardiac walls is proportioned to the distension of the aorta.[1] Hence, whatever interferes with

[2] Die Krankheiten des Herzens, Theil ii. Abt. i. p. 460.

[2] Dict. des Sciences Méd., art. Cœur. 1813.

[3] Traité des Maladies du Cœur et des Gros Vaisseaux, by R. J. Bertin. Redigé par Bouillaud. Paris, 1824.

[4] Loc. cit. p. 300.

[5] A Treatise on Diseases of the Chest, Forbes' Trans. 1821, p. 372.

[1] This was very clearly taught by Corvisart. "The heart . . . will have to drive forward, through the narrow artery, too great a column of blood . . . which will necessarily react upon the agent which impels it. . . . Finally, the coronary arteries as well as the capillaries of the heart, remaining in a permanent state of fulness, will supply more nourishing matter to the fleshy substance of this organ; whence arise, without doubt, the increase, at least in part, of its vital energy . . . the greater consistence of the parietes, and the more vigorous action of the organ."—Loc. cit. p. 60.

the quantity of blood entering the aorta lessens, *cæteris paribus*, the capacity of the heart for overgrowth; whatever increases the quantity of blood sent into the aorta, and increases the tension of the blood in it, increases the blood-supply to the heart, increases its capacity for overgrowth. This is no doubt one of the conditions which determines the great hypertrophy so common in aortic regurgitation. The distension of the aorta at the end of the ventricular systole, when the coronary arteries are being filled, is, in that disease, extreme.[1]

(4) The greater (within limits) the proportional amount of rest of the heart, the more perfect is its nutrition. The period available for nutrition is greater when the contractions are infrequent than when they are frequent. The systole is nearly of the same duration at different frequencies; increased frequency in contraction is at the expense of the diastole. Hence infrequent contraction favors the development of hypertrophy when its exciting cause exists. The actual influence of this condition is obscured by the increase in the exciting cause, overwork, which frequency of action involves.

B. *Exciting Causes.*—As far as is at present known muscular hypertrophy has but one immediate cause—increase of work. The operation of this cause, the "physiological stimulus," as it has been termed, may be traced in almost every instance in which hypertrophy is found. Each apparent exception becomes conformable to the rule when the conditions under which the hypertrophy began are accurately known. The over-action of the heart is the cause of its over-growth. Such over-action may be primary, or it may be secondary to an increased resistance to its action. Primary over-action commonly takes the form of increased frequency of contraction. Secondary over-action is in the form of increased force of contraction. But the distinction is not absolute, as will appear immediately.

Other causes have been assumed to account for hypertrophy in cases in which the influence of increased work could not be clearly traced. An irritative influence of the blood on the heart, leading directly to its overgrowth, has been assumed in order to account for some cases of hypertrophy. But there are at present no facts to support the idea that any blood state, any nutritive influence other than the physiological stimulus, ever leads to overgrowth of muscular tissue.[1]

I. *Simple Over-action of the Heart*, the conditions of the circulation and heart entailing no increased resistance, *i. e.* no primary increase of work—is always the consequence of deranged innervation. Its nervous mechanism is at present ill-understood. It is extremely doubtful whether a simple increase in the force, without change in the frequency, of the heart's action, ever results from this influence. Increased frequency is the common result. The more frequent contractions are often apparently more forcible. Such over-action of the heart is well seen in simple nervous palpitation, and most strikingly in exophthalmic goitre. Continuous emotional excitement is a powerful cause of it. It is produced also by the influence of many agents, such as alcohol, tea, and coffee. It is produced also by general muscular effort. Effort acts, it must be remembered, in another way, by causing increased resistance to the movement of the blood.[2]

[1] Milner Fothergill (Diseases of the Heart, p. 65) maintains that the blood-supply to the heart walls is deficient in aortic regurgitation, because the tension in the aorta so soon falls. But, from the short course of the coronary arteries, their distension must be rapid, and related, in degree, to the degree of the tension of the aortic blood, rather than to the duration of the tension.

[1] The conditions of overgrowth in different tissues no doubt vary widely. In some, hyperplasia of the proper tissue elements is induced by any local irritant. This has suggested a generalization which asserts a common basis for hypertrophy and inflammation. The conclusion, true of some tissues, is quite inapplicable to muscular fibres. (*Vide* Moxon, Med. Times and Gazette, Nov. 26, 1870.) But the theory has obtained in Germany wide currency and application, so that a recent writer (Zielonko, Virchow's Archiv, 1872) gives, as an example of hypertrophy of the heart, the enlargement which resulted from the insertion of a seton in its substance, although microscopical examination showed only ordinary inflammatory products as the cause of the enlargement. Henry Green (Clin. Soc. Trans. vol. ii.) has suggested that hypertrophy of the heart may sometimes be due to the irritative influence of the blood in rheumatism, but the evidence which he has adduced is chiefly clinical, and possesses little weight in comparison with the almost uniform significance of pathological facts.

[2] Les mouvemens violents donnent souvent plus de masse au cœur de même que les maladies: nous réduirons ces mouvemens aux exercises fatiguants, à l'agitation qui suit les excès du vin, et à celle qui causent les passions (Senac. loc. cit. tom. ii. p. 400). Corvisart recorded his conviction that the passions were the most powerful cause of organic diseases of the heart, and instanced the influence of the French revolution in causing the malady (loc. cit. pp. 322 and 323). Statistics furnished by Farr, and given by Quain in his Lumleian Lectures, show that the deaths of males at all ages from heart disease have increased fifty per cent. on the increase in

Such increased frequency of contraction tends to cause hypertrophy only in so far as it increases the total work of the heart. It does this, however, in more than one way. (1) Part of the work of the heart consists in the movement of its own mass. No doubt this is but a small fraction of its total labor, but it is a definite quantity, and increases directly as the frequency of contraction. (2) Although simple increase in the frequency of contraction of the heart does not necessarily increase that part of the heart's work which consists in the propulsion of the blood, it does practically effect such an increase. If a heart contracts at twice the normal frequency, and the blood enters the heart at the normal rate, only, say, one-half of the normal quantity of blood will at each diastole enter, and at each systole be discharged. The work of the heart in propelling the blood would thus remain the same. Practically, however, increased frequency of contraction tends to quicken the whole circulation, so that under the circumstances assumed, more than half the normal quantity of blood would at each contraction enter and leave the heart. Hence the tension of the arterial blood becomes increased, and the pulse fuller and less compressible. Reflex relaxation of the peripheral arterioles, the natural effect of increased tension, relieves, but often incompletely, this increased tension. Thus intra-ventricular pressure and the work of the heart are increased. (3) The heart, acting thus with excessive frequency, may act also with excess of force. The increased force may be felt under such circumstances. The heart "thumps" against the ribs. In the pulse the increased force often is unnoticed on account of the smaller quantity of blood which leaves the left ventricle at each contraction. It should be remembered that many circumstances which increase the frequency, also, at the same time, increase the force of the heart's action. Muscular effort is one of these.

This then is the mechanism by which increased frequency of contraction may cause hypertrophy. Its total influence is not, however, great. Increase in frequency of contraction is rarely of long duration under circumstances of due nutritive energy, and it is not often that hypertrophy can be ascribed with probability to simple primary over-action of the heart.

II. *Increased Resistance to the Action of the Heart* is unquestionably the chief cause of its hypertrophy. Such resistance may be in the form of (1) traction from without, or of (2) pressure within the contracting organ.

(1) As a matter of fact pericardial adhesions are frequently associated with cardiac hypertrophy;[1] and, according to Wilks,[2] with hypertrophy of the right ventricle much more frequently than of the left. It is easily conceivable that such adhesions may oppose the diminution in size, and change of shape, which the heart undergoes during its contraction. But for such adhesions to hinder a contracting heart, the external surface of the pericardium must be connected with more than usual firmness to the adjacent structures. It is not certain, moreover, that resistance to contraction applied from without has the same effect as resistance applied within the heart, and the conditions are so complex that it is impossible to trace the direct influence of the adhesions in causing the hypertrophy. Dilatation is invariably, under such circumstances, associated with the hypertrophy of the heart. It would seem to be a more direct result of the pericardial adhesion than the hypertrophy, both as the simple effect of the external traction, and as the result of the weakening of the wall of the heart by the sub-pericardial changes. But dilatation tends in itself, as will be shown immediately, to produce hypertrophy, and the hypertrophy in an adherent heart, without other cause of hypertrophy, is commonly not more than the dilatation might account for. The effect of pericardial adhesions is considered at greater length, in the article on Dilatation of the Heart. Their direct influence in causing hypertrophy must be regarded as possible, but unproved.

(2) Increased blood-pressure within the heart during its systole is the common cause of its muscular over-growth. This is the element which underlies most of the conditions capable of giving rise to hypertrophy. This increased pressure may be due to one of two causes; (*a*) the mass of blood to be moved may be abnormally large; (*b*) there may be an abnormal obstruction to the movement of the blood. The effect of each condition is to augment the resistance to be overcome by the contracting fibres—to increase the work of the heart.

(*a*) The mass of blood to be moved may be abnormally large. This condition exists in all forms of over-distension of the heart. Dilatation cannot exist without an increase in the work of the heart. Hence hypertrophy is its almost invariable concomitant—invariable when the nutritive conditions are such as to render growth of muscular fibre possible.

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population, and that this increase affects adult life almost exclusively (Lancet, 1872, vol. i. p. 392).

[1] As Morgagni, Beau, Hope, and others have especially noticed.

[2] Guy's Hosp. Reports, vol. xvi. p. 202.

The mechanism of over-distension is considered fully in the article on Dilatation of the Heart. It may be direct or indirect. It is direct when a cavity is over-filled by the contraction of an over-distended chamber behind it. Thus in mitral regurgitation the left ventricle is over-filled by the contraction of the over-distended left auricle, and becomes dilated and hypertrophied; or the over-distension may be indirect, the result of a supply of blood to the chamber from a double source—the regurgitation of blood into the chamber and its supply in the normal course of the circulation. Thus the left ventricle becomes over-distended, dilated, and often enormously hypertrophied in aortic regurgitation; and the left auricle becomes dilated and hypertrophied in mitral regurgitation. So, too, in dilatation from the weakening of the wall consequent on pericarditis, hypertrophy commonly ensues. No doubt in these conditions of dilatation the whole of the blood is not always expelled from the ventricle at each systole, but the intracardiac pressure during the systole is still increased and with it the work of the heart.

Plethora has been supposed to cause cardiac hypertrophy. Niemeyer points out that the transient plethora induced by a hearty meal with much fluid may, if habitually repeated, have such an influence. The action of the kidneys commonly prevents any permanent distension of the vessels from this cause.

(*b*) There may be an obstruction to the movement of the blood superadded to that which exists in health. This obstruction may be situated within or without the heart. Within the heart, it may be at the orifice by which the blood leaves the chamber affected. Thus an obstruction at an auriculo-ventricular orifice will cause hypertrophy of the corresponding auricle; obstruction at the orifice of the pulmonary artery will cause hypertrophy of the right ventricle; obstruction at the aortic orifice will cause hypertrophy of the left ventricle. In all these cases dilatation may be conjoined with the hypertrophy, and increase its amount.

The obstruction may be outside the heart. It may be in the larger arteries, the aorta and pulmonary artery. Their calibre may be reduced by pressure upon them (as by an aneurism of another vessel), or by constriction due to changes in their walls.[1] The hypertrophy which occasionally occurs in long-continued displacement of the heart, whether from pleural effusions or deformities of the thorax, consequent on curvatures of the spine, &c., is probably due chiefly to the increased obstruction in the great vessels from their displacement and altered course.[1]

Aortic aneurism has been regarded as a cause of hypertrophy of the left ventricle since the days of Corvisart. The association of the two has frequently been noted, and has been referred by Niemeyer to the law in physics according to which the resistance encountered by a liquid moving through a tube is increased if the tube be suddenly expanded, just as if it be contracted. But it is a matter of considerable doubt whether hypertrophy does occur as a simple consequence of aortic aneurism. Senac long ago expressed a doubt upon the subject.[2] Stokes affirmed that "we have no reason to believe that the existence of aneurism in any portion of the aorta throws additional labor on the heart, and hence we commonly find a small heart coexisting with a vast aneurism."[3] Walshe also regarded the hypertrophy as an occasional consequence, and not invariable even when the sac of the aneurism was situated near the sigmoid valves. The observations of Axel Key,[4] indeed, suggest the question whether hypertrophy of the heart is not more common when the aneurism is far from, than when near the heart. He has recorded eighteen cases of aneurism near the heart, in not one of which was there hypertrophy of the left ventricle. In most of the cases, indeed, the muscle was more or less thinned, with or without slight dilatation, especially of the lower part of the cavity. Considerable dilatation seemed related to disease of the aortic valves, not to the aneurism. In several cases the cavity of the ventricle was positively diminished in capacity, although the walls were thinned. In some instances the muscle of the conus arteriosus was thick, while the rest was thin. The atrophy of the muscular tissue was most marked in some cases in which the aneurism lay near the heart. He suggests as an explanation of this singular atrophy of the left ventricle, the pressure of the aneurism on the pulmonary artery, lessening the amount of blood reaching the left ventricle, and the withdrawal from the circulation of the blood contained in the sac of a large aneurism.

[1] Hypertrophy of the left ventricle has been produced artificially by Zielonko, in the guinea-pig by tying a ligature round the aorta, and thus reducing its calibre. Virchow's Archiv, Bd. 62, Heft I. p. 22.

[1] See Hilton Fagge, Path. Trans. vol. xvii.

[2] "It is certain that the dilatation of these vessels (aorta and pulmonary artery) have not always the consequence (of causing enlargement of the heart)." He goes on to describe a case in which the aorta was dilated to the size of a head, from the arch to the diaphragm, in which the volume of the heart was normal. Senac, Traité, &c., 1749, tom. ii. p. 407.

[3] Diseases of the Heart and Aorta, p. 579.

[4] Nord. Med. Ark. 1869, I. 4, Nr. 22, and Schmidt's Jahrbuch, vol. 150. p. 21.

Degenerative changes in the arteries cause a considerable increase in the total work of the heart, and are effective causes of hypertrophy. The increased resistance which they produce is due to the loss of elasticity in the vessels, their more tortuous course, and the increased friction from roughening of their inner surface. In health the elastic vessels yield before the blood which is thrown into them. When elasticity is lost the vessels approximate to rigid tubes, and the resistance they present is consequently increased. By the increased tortuosity of the vessels, due to the loss of elasticity, their absolute length becomes greater, and the friction of the blood against the wall of the vessel is also increased. These degenerative changes are usually found, in greater or less degree, after middle life, and are probably the cause of the increase in the thickness of the left ventricle, which has been said by Bizot[1] to occur during the later period of life. Degenerative changes may be a consequence as well as a cause of cardiac hypertrophy, the result of the increased strain to which the vessels are exposed. This fact, which will be considered presently, must not be forgotten in estimating the significance of the association.

The obstruction may be situated in the minute arterioles and capillaries. In certain diseases of the lungs obstruction from this cause may be traced. In emphysema many vessels are destroyed, and those which remain are elongated and narrowed by the over-distension of the air-cells. The obstruction to the passage of the blood through the lungs is thus very much increased, and hypertrophy and dilatation of the right ventricle result, and may be carried to a high degree. Hypertrophy of the heart is not infrequent in phthisis ; Quain states that in 171 cases it was present in 25 per cent. of the males, 7 per cent. of the females. The conditions of lung to which it is related have not yet been ascertained, but in cirrhosis of the lung it is especially frequent ; the compression and destruction of the minute vessels by the contracting tissue produce the obstruction. Compression of the lung tissue by pleural effusion is said to have a similar effect. In all these conditions, if long continued, hypertrophy of the right ventricle may occur.

Long-continued muscular effort entails cardiac hypertrophy. As Clifford Allbutt and Myers have shown, the influence of this cause can often be distinctly traced, especially (Milner Fothergill says) among those who work with the arms. Animals frequently afford instances of the remarkable effect which this cause is capable of producing. The most celebrated instance is that of the celebrated Irish greyhound "Master Magrath," the heart of which bore three times its normal proportion to the body-weight, and no cause for the enlargement but extreme and long-continued exertion could be discovered.[1] The increased work in which the hypertrophy arises is probably in part the result of the increased frequency and force with which, in consequence of the respiratory needs, the heart acts. But it is in part the result of the compression of the capillaries of the muscles by the contracting fibres, and also the result of the compression of the arterial trunks by the rigid muscles. The total resistance to the action of the heart is thereby considerably increased. This resistance is not a matter of conjecture. Increase in arterial pressure during general muscular contraction has been demonstrated experimentally by Traube.

During pregnancy the addition of the placental to the systemic circulation involves a considerable addition to the work of the heart. Larcher[2] found, on examination of the hearts of 100 women who died in child-birth, that the wall of the left ventricle was invariably thickened. The average thickness was ·015 m. (about $\frac{6}{10}$ inch). His observations have been confirmed by the clinical investigations of Duroziez, who found that the greater the number of pregnancies the more permanent is the enlargement. He asserts that the enlargement continues through the whole of the lactation period. Friedreich, however, expresses some doubt on the subject.[3]

The remarkable hypertrophy of the heart which is met with in Bright's disease must be considered among those which result from obstruction to the flow through the minuter vessels. It occurs in all forms of chronic kidney disease, most frequently in the contracted kidney, least frequently in the lardaceous form. According to Grainger Stewart, it is invariable in the last stage of the acute inflammatory affection, in which, the disease having assumed a chronic form, the kidney undergoes reduction in bulk.

The hypertrophy which occurs in this condition is confined to the left ventricle, and is often uncomplicated by dilatation. It is frequently considerable in amount. Among such hearts the best examples of simple hypertrophy are met with. After death the heart often remains firmly contracted, and the characters of a concentric hypertrophy are simulated. Dilatation may coexist with the hypertrophy in consequence of coincident degeneration.

[1] Mémoirs de la Société Médicale d'Observ. de Paris, 1836.

[1] Haughton, British Medical Journal, Jan. 20, 1872.

[2] Archives Gén. de Méd., Mars, 1859, and note by Larcher appended to a paper by Menière, Ibid., tom. xvi. 1828, p. 521.

[3] Herzkrankheiten, p. 288.

The association of this hypertrophy of the heart with kidney disease was first pointed out by Bright in 1827[1] as so remarkable that some causal connection between the two must exist, and he afterwards, in 1836,[2] expressed his opinion "either that the altered quality of the blood affords irregular and unwonted stimulus to the organ immediately, or that it so affects the minute and capillary circulation as to render greater action necessary to send the blood through the distant subdivisions of the vascular system." The latter theory is that which has obtained general acceptance, with certain modifications to be alluded to more fully. Modern investigation, while it has extended our knowledge of the conditions under which the hypertrophy arises, has scarcely carried us further in our explanation.

The most important addition to our knowledge is certainly the fact that increased tension of the arterial blood commonly occurs in those cases of Bright's disease in which hypertrophy of the heart is so often found. The hardness of the pulse in such cases had long been remarked, but its significance was not generally recognized until the sphygmograph, by supplying a measure of its degree, drew attention to its importance as supplying independent evidence of an obstruction to the movement of the blood through the smaller vessels.

Traube,[3] who first called attention to the significance of the increased arterial tension, assumed, in effect, that the increased resistance within the kidney was the cause of the obstruction. The theory has been largely accepted in Germany, but its manifest inadequacy has prevented it from meeting even partial acceptance in this country. It is said[4] that Traube himself before his death ceased to hold it in its original form.

In the smaller arteries a remarkable change of structure was pointed out by George Johnson in 1850 as hypertrophy of the muscular coat.[5] First discovered in the kidney, the change was soon found to be general throughout the system. The occurrence of such increased thickness of the walls of the arteries is now generally admitted, and the view that the thickening is due to hypertrophy of the muscular coat has received very wide confirmation. Muscular over-action being the only known cause of muscular hypertrophy, Johnson at first ascribed the vascular change to the same cause as the hypertrophy of the heart—the resistance to the movement of the blood through the capillaries. It was assumed that the arteries by their contraction aided the circulation of the blood, and over-acted to overcome the increased resistance. But with the fall of the theory of arterial propulsion, this explanation became untenable. The function of the muscular coat of the vessels being, as far as is known, the adjustment of the calibre of the vessel, permanent spasmodic contraction became the only explanation of the hypertrophy, and has been for many years ably maintained by Johnson. That such spasm exists is, on Johnson's facts, highly probable, and may, the writer believes, be actually seen in the arteries of the retina in most cases of Bright's disease in which a high arterial tension exists. The effect of such spasm must be an increased resistance to the movement of the blood in the arteries, an augmentation of its tension. Instead of aiding, it thus directly opposes the action of the heart. That it is the sole cause of the increased resistance may be doubted. Even if it were the only cause, the difficulty is not lessened, for we are almost as ignorant of its origin as we are of any obstruction due to the changed composition of the blood. The natural effect of increased arterial tension, increased endocardial pressure, is immediate relaxation of the minute arteries, and freer circulation. The spasm of the vessels under these circumstances is therefore a phenomenon very difficult to explain. Ludwig asserts on experimental grounds that it is due to the action of the retained urinary salts on the vaso-motor centre. [The difficulty of this explanation has arisen from the decline, above alluded to, of the theory of arterial propulsion; and when this theory is, as there is good reason to believe it will be, restored to physiology, the explanation first adopted by G. Johnson will prevail satisfactorily. Admitting, with Sir Charles Bell,[1] and, later, Legros and Onimus,[2]

[1] Med. Reports, p. 23.

[2] Guy's Hosp. Reports, vol. i. p. 397.

[3] Zusammenhang zwischen Herz u. Nierenkrankheiten, Berlin, 1856.

[4] By Milner Fothergill, Diseases of the Heart, p. 286. The inadequate character of Traube's theory led Bamberger into a denial of Traube's facts, relative to the increased obstruction to the movement of the blood, and consequently increased arterial pressure. But these facts may now be considered to be established, and the details of the controversy between Traube and Bamberger have ceased to be instructive.

[5] Med.-Chir. Trans., vol. xxxiii. 1850, p. 107.

[1] Essay on the Circulation of the Blood. I have elsewhere argued this question at length; in Transactions of the American Medical Association (Prize Essay on the Arterial Circulation, 1856; On the Present Condition of Vaso-motor Physiology, 1872), and in the American Journal of Med. Sciences, July, 1868, p. 289.—H.]

[2] Journal de l'Anatomie et de la Physiologie, 1868–70.]

that there is a true *arterial systole*, by which the arteries aid the heart in the onward movement of the blood, it is easy to understand how the same cause (local obstruction) will produce hypertrophy of the muscular tissue both in the smaller arteries and in the heart.—H.]

The existence of the hypertrophy of the muscular coat of the arteries has, however, been denied by Gull and Sutton,[1] who ascribe the thickening to a "fibrosis," and attribute the resistance to the movement of the blood to the obstruction in the vessels due to the inelasticity of this tissue. They do not regard the fibrosis as the consequence of the renal disease, but as a primary general change, of which the affection of the kidney is only one local instance. This theory of the primary general character of Bright's disease accords very well with the phenomena of some cases, but as an explanation of all cases of contracting kidneys it is open to some objections apart from the weight which must be attached to Johnson's observations. In many cases of contracting kidney there is certainly fibroid over-growth to be found widely distributed, but the degree of change in the kidney is incomparably greater than that in other organs, so as to suggest strongly the idea of a primary affection of the kidney. Another fact to be taken into consideration is that, whatever be the cause of the hypertrophy of the heart in the contracted kidneys, a similar hypertrophy results as a remote consequence of kidney disease unquestionably local in its origin. In later stages of an acute nephritis, hypertrophy of the heart is even more frequent than in the primary contracting kidney, and is associated with the same increased arterial tension.

The conclusion then seems to be that hypertrophy of the heart occurs in kidney diseases as a result of increased arterial blood-pressure, the result of some obstruction to the movement of the blood in the minute vessels; that such obstruction is in many cases the indirect consequence of the kidney disease; that it is accompanied in most cases with a morbid state of the smaller arteries, to which it is in part to be ascribed.

Lastly, in some cases, the obstruction causing the hypertrophy of one ventricle may be situated, not in the vessels, large or small, but beyond them, in the other side of the heart. Thus in mitral obstruction, the right ventricle is very constantly hypertrophied; in obstruction in the pulmonary system and right side of the heart, the left ventricle may become hypertrophied. The obstruction may even be on the same side of the heart, and act through both systems of vessels, the pulmonary and the systemic. Thus mitral regurgitation may, as Friedreich has remarked, cause not only congestion of the lungs, distension of the right side of the heart, over-filling of the systemic nervous system, but increase on the tension of the arterial blood, and thus cause an increase in the work of the left ventricle, and an increase in its hypertrophy. It is not easy to understand the mechanism by which this arterial distension is effected, but as a clinical fact it is unquestionable, and occurs especially in cases of mitral regurgitation, in which the left ventricle is greatly dilated and hypertrophied. It is perhaps to be ascribed to the effect of the secondary dilatation of the right side of the heart in augmenting the mechanical obstruction. A tracing from a pulse in such a condition is shown in Fig. 121, p. 777.

It may be convenient to group the causes of hypertrophy which have been described, first, according to their position (Table I.), secondly, according to their effect (Table II.).

[1] Med.-Chir. Trans. vol. lv. 1872.

TABLE I.

EXCITING CAUSES OF CARDIAC HYPERTROPHY.

- Over-action, primary . . . . . → Nervous palpitation, effect of alcohol, tea, coffee, &c., muscular effort (in part).
- Over-action secondary to increased resistance.
  - Exocardial . . . . . → Pericardial adhesions.
  - Endocardial
    - Increase in mass of blood to be moved → Dilatation of heart, primary or secondary to regurgitation, &c.
    - Increase in obstruction to movement of blood, situated,
      - Within the heart, acting immediately, → Contraction of orifices.
      - Outside the heart,
        - Arteries large, → Constriction of aorta, or pulmonary artery, aneurism (?), displacement.
        - Arterial system generally, → Degenerative changes, loss of elasticity, atheroma, &c.
        - Arterioles and capillaries,
          - Emphysema, cirrhosis, pleural effusions, → Pulmonary, acting on right ventricle.
          - Muscular effort (in pt.) pregnancy, Bright's disease, → General, acting on left ventricle
      - Within heart, acting circuitously, through circulation, → Valvular disease, &c.

TABLE II.

CAUSES OF HYPERTROPHY.

AFFECTING—

ALL PARTS OF HEART—

Over-action from nervous and toxic influences.
Dilatation of cavities.
Displacement of heart.

LEFT VENTRICLE—

Mitral regurgitation.
Constriction and regurgitation at aortic orifice.
Constriction or compression of aorta.
Aneurism of aorta (?).
Degeneration of arterial system.
Renal disease.
Pregnancy.
Muscular efforts.
Valvular disease of right side of heart, and all causes of dilatation of right ventricle.

LEFT AURICLE—

Mitral constriction and incompetence.

RIGHT VENTRICLE—

Constriction or regurgitation at pulmonary orifice.
Constriction of or pressure on pulmonary artery.
Degeneration of pulmonary arteries.
Lung diseases, obstructing, compressing, and destroying vessels.
  Chronic bronchitis.
  Emphysema.
  Cirrhosis.
  Pleural effusion.
Affections of mitral orifice.

RIGHT AURICLE—

Regurgitation and constriction at tricuspid orifice.

By what mechanism the increased work leads to muscular over-growth we have little knowledge. The theory has been put forward that the effect depends on reflex dilatation of the coronary arteries. Increased blood-pressure within the heart is known to inhibit, by the depressor nerve, the vaso-motor system, causing dilatation of the minute arteries, and freer circulation. The coronary arteries are believed to participate in this effect, and the readier circulation through them has been thought to be the cause of hypertrophy. That such an action occurs is most probable, and it is probable that thus the nourishment necessary for over-growth is supplied. But that it is not the sole cause is almost certain, from the fact that if the work of a muscle remains the same, a larger supply of blood to it has no power to increase the muscular tissue. We are driven to assume a direct influence of the increased contraction on the growth of the fibre. The average force exerted habitually by a muscle is far below its possible maximum at any moment. It would seem as if this average force, and the bulk of the muscle, were proportioned, that an increase in the habitual force leads, in due nutritive conditions, to muscular over-growth. Whether this over-growth is the result of the direct mechanical stimulus to the contracting fibre, or whether it is the result of a reflex influence exerted through the nervous system, and excited by the increased pressure on the endocardium or by the increased tension on the contracting fibres, we do not know.

One condition is, however, essential for the development of hypertrophy—time. A certain period is necessary for growth of old, or for the development of new tissue. Dilatation may occur quickly; hypertrophy can only take place slowly. Hence the rapidity with which an increased resistance is developed largely influences the resulting condition of heart. Obstruction is usually slowly developed, regurgitation may occur rapidly, and this is one reason why the former entails so much simpler an hypertrophy than the latter. So, too, in the obstruction which is developed in the most gradual manner, that of Bright's disease, uncomplicated hypertrophy is commoner than in any other morbid state. The related conditions of origin of hypertrophy and dilatation are considered more fully in the article on Dilatation.

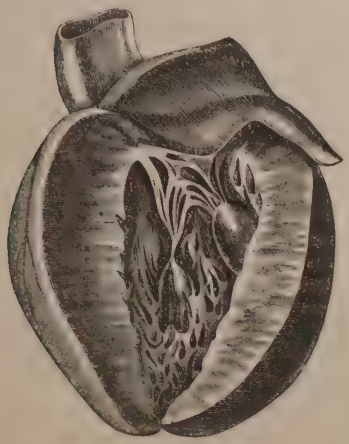
A few cases of hypertrophy have been recorded in which no mechanical cause for the hypertrophy could be discovered. Their proportion to the cases of hypertrophy in which a mechanical influence can be traced is very small, so small that it is probable that some such cause may have existed and have escaped observation. Some of the cases were recorded before the relation of hypertrophy of the heart to kidney disease was well known, and the existence of the latter may easily have been overlooked. In Bristowe's case of "hypertrophy without sufficient cause," recorded in the Path. Trans., vol. v. p. 82, the heart, which weighed twenty-seven ounces, was uniformly enlarged and hypertrophied without local disease, but the kidneys were reduced in size, and granular, and presented atrophy of the Malpighian bodies. In other cases the increased bulk of the organ is due to an increase in the fibrous, as well as in the muscular tissue. Such was the case in a heart weighing forty ounces, preserved in St. George's Hospital Museum, and supposed to be an example of true hypertrophy, until Quain examined it, and discovered its real nature.

Pathological Anatomy. — Hypertrophy may occur in each division of the heart, but varies in different parts, both in the frequency of its occurrence and in the degree commonly attained. The comparative affection of the different parts of the heart depends partly on the frequency and degree with which the causes of hypertrophy affect the different portions, and partly on the amount of muscular tissue each part possesses, and by which it is enabled to resist rather than yield to the internal pressure, which is the cause of the hypertrophy. The left ventricle is that affected most frequently, and in the greatest degree. Next in frequency and degree comes the right ventricle; then the left auricle; lastly, the right auricle. It is rare for the hypertrophy to be general, and to affect all parts of the heart. More commonly it is partial, affecting one part only. The increase in the weight of the heart is the invariable characteristic of hypertrophy. Since the healthy heart consists almost exclusively of muscular tissue, over-growth of that tissue cannot occur at the expense of any other constituent, and must result in an absolute increase in the weight of the heart, proportioned to the hypertrophy.

There is also in most cases an increase in the size of the heart. If the cavities of the heart are unaltered, the increase in its size is proportioned to the hypertrophy. This is the case in the so-called "simple" hypertrophy. The cavities rarely, however, remain unchanged. They are believed by some authorities to be occasionally diminished in size. The heart then may be of normal size, or may be very slightly enlarged. The increased thickness of the walls is at the expense of the cavity, which may become reduced to very small dimensions, it is said incapable of containing a walnut. This con-

stitutes the concentric hypertrophy of Bertin. Lastly, the cavity is, in the majority of cases, dilated, and the dilatation adds greatly to the size of the heart. This constitutes the eccentric variety of Bertin.

[Fig. 120.



Hypertrophy of left ventricle.]

Concentric hypertrophy of the heart has, in most recorded examples, been local, and confined to the left ventricle. Its existence has, however, been the subject of much discussion. It was thought to be common until Cruveilhier, in 1833,[1] pointed out how perfectly its characters were simulated by hearts the subjects of simple hypertrophy and post-mortem contraction. When the heart is at the time of death in systole, the final contraction is fixed by the rigor mortis, the thickened walls are, by their contraction, further increased in thickness, and so remain, and the cavity is reduced to very small dimensions. The resemblance of such a heart to "concentric hypertrophy" is admitted by all authorities. Cruveilhier maintained that all cases of the supposed change are thus explicable, that the cavity of such a ventricle can always be opened out with the fingers, and in this he has been followed by Budd[2] and many later pathologists, who urge further that the contraction of the cavities supposed to occur is incompatible with the absence of symptoms of impeded circulation of the blood. Other authorities believe that concentric hypertrophy does rarely occur. Skoda, Rokitansky, Bamberger, Förster, Walshe are all of this opinion. They assert that hearts are occasionally met with, the cavities of which are so small that the hypothesis of mere contraction is untenable, and is not verified by the effect of post-mortem decomposition, which should relax completely the contraction of rigor mortis. Dechambre and Forget maintained that such hearts could not be expanded, as simply contracted hearts can be.

The balance of recent pathological evidence is certainly opposed to the occurrence of concentric hypertrophy. It is to be noticed that the careful pathological observation of recent years has brought to light few supposed examples of this change. One specimen only has been brought before the Pathological Society.[1] There is in the museum of University College a specimen (No. 2,140) which has been described as itself establishing the existence of the change. But on close examination the characters are far from satisfactory evidence — it is obviously merely an example of the permanent contraction of a heart the subject of simple hypertrophy.[2] The known mechanism of

[1] Dict. de Méd. et de Chir. Prat. art. Hypertrophie.

[2] Med.-Chir. Trans. 1838.

[1] By Wickham Legg. Trans. Path. Soc. vol. xxv. p. 105. The specimen presented contraction of the mitral orifice. Details of measurement and weight of the heart are not given.

[2] The specimen in question has been appealed to as decisive, and illustrates so well the characters which have led to the establishment of this variety, that it is worth detailed description. In the circular glass jar in which it had been preserved for many years it certainly had striking proportions. The walls appeared of great thickness, and the cavity "scarcely capable of containing a hazel-nut." When removed from the jar it appeared considerably smaller. Its weight, with the root of the aorta, is 11½ oz., but it has been kept for many years in spirit. The external length of the ventricle is 4 inches. The heart has been divided transversely midway between the base and apex of the left ventricle. The diameters of the section are antero-posterior 2¼ inches, lateral 3 inches. It is evidently a firmly contracted heart, for the cavity of the right ventricle is a mere curved line. The cavity of the left ventricle is, on close examination, stellate; from the centre three linear branches radiate, and can be opened up. Between them lie the enlarged papillary muscles. On measurement with a wire, the circumference of this stellate cavity, following its branches but excluding the loose papillary muscles, is 4¼ inches. But the most conclusive evidence is afforded by the thickness of the walls measured at the extremities of the radii of the cavity. On the left side the wall measures ⅝ of an inch, in front and behind just ½ inch in thickness. After every allowance has been made for the effect of the spirit, it seems clear that the thickness of the wall is only a little above the normal. The increased weight is proof that the extent of the wall cannot have been below the normal. It is clear also, from the state of the right ventricle, that the heart is

hypertrophy renders the origin of this form of over-growth scarcely conceivable. If hypertrophy is the result of increased work, increased intra-ventricular pressure, the volume of the blood within the ventricle can scarcely have been lessened; but without such lessening, reduction in the size of the cavity cannot have occurred. Thus the increased thickness of the wall and lessened size of the cavity are, on etiological grounds, almost incompatible. Moreover, post-mortem decomposition relaxes hearts, firmly contracted, in a very imperfect manner.

Concentric hypertrophy of the right ventricle has been described as an occasional consequence of some congenital malformations of the heart.

Eccentric Hypertrophy.—The thickening of the wall in eccentric hypertrophy is not always conspicuous. The cavity is dilated, and the superficial area of the wall increased, and the increase in tissue may be only enough, or even not enough, to maintain the normal thickness of the wall. Thus the wall of the left ventricle may be so hypertrophied as to lead to a considerable increase in the weight of the heart, and yet may be only of the average thickness. In estimating the presence and amount of hypertrophy, therefore, the size of the cavity must always be taken into consideration. In one of the heaviest hearts recorded, a heart much dilated, the thickness of the wall of the left ventricle was not more than is common in less dilated hearts of only half the weight.

The increase in the size and weight of the heart is often very considerable. In estimating them it should be remembered that the normal average weight varies according to the sex, age, size of the individual. These are considered elsewhere (art. "Size and Weight of the Heart."). A heart which exceeds 9 oz. in a man or 8 oz. in a woman, probably possesses an excess of some constituent, in most cases of muscular tissue. A common weight for hypertrophied hearts is 12–16 oz. Hearts are occasionally seen of much greater weight, especially when dilatation extends the area, and hypertrophy the thickness of the cardiac walls. Under these circumstances the enormous "bovine" hearts are met with. Walshe has met with one weighing 40 oz.; Lancisi mentions one which weighed, emptied of blood, two pounds and a half; Croker King one of 44½ oz.; Austin Flint one of 46 oz., while hearts weighing 46½ oz. have been shown by Bristowe and by David at the Pathological Society. The enormous weight of five pounds, mentioned by Lieutaud, must be regarded as doubtful. How much more then the "quinze livres" of Marchetis![1]

The shape of the heart is altered according to the part affected. If one ventricle is more affected than the other, that which is the more hypertrophied forms a larger share of the apex than in health, and the chief enlargement of the heart is on the side of the affected ventricle. Thus in simple hypertrophy of the left ventricle, the extremity of that ventricle extends beyonds the other, so as alone to constitute the apex, while increased width results from the lateral enlargement. The resulting shape resembles an obtuse-angled triangle when the heart is relaxed, an elongated ovoid when partially contracted. In hypertrophy of the right ventricle the extremity of that ventricle extends to the apex of the heart, but does not usually pass beyond the other. Hence the apex is much rounder than in health, and may be indistinguishable. When dilatation is joined to the hypertrophy, the width of the heart is much increased, and the transverse may exceed the vertical diameter. This is especially the case when the right ventricle is affected, when the heart may assume an almost spherical shape. Hypertrophy of the auricles is never sufficient to alter the shape of the heart; the effect of their dilatation is considered elsewhere.

The increase in the thickness of the wall is in direct proportion to the amount of hypertrophy, but in inverse proportion to the amount of dilatation. The hypertrophy is usually so much in excess of the dilatation as to cause an absolute increase in the thickness of the wall. This is commonly greater in the outer wall than in the septum. In the ventricles the trabeculæ and papillary muscles usually participate in the hypertrophy, and, it is said, to a greater extent in the right than in the left ventricle. Sometimes they are thinned, when the heart is dilated.

In health the thickness of the ventricular wall varies considerably in different parts. The average thickness of the wall of the left ventricle is about half an inch

firmly contracted, and also that the circumference of the inner surface of the left ventricle—the test of the actual reduction in size of the cavity—is little, if any, less than the normal. It seems, therefore, to be merely an example of firm contraction in a heart the subject of moderate simple hypertrophy. The history of the specimen is not known.

[1] Quoted by Senac, loc. cit. tom. ii. p. 408. Bellingham is said, by several writers, to have met with a heart weighing 80 oz. The only ground for the assertion seems to be that Bellingham states that he had seen a heart preserved in the museum of St. George's Hospital which was said to weigh five pounds. This seems to refer to the large heart alluded to by several writers and mentioned above (p. 772) as lately examined by Quain and found to weigh 40 oz.

in men, rather less in women. The measurement should be always exclusive of the papillary muscles, and the place at which the measurement is made should always be specified. The increase is usually greater towards the base than towards the apex. Hope pointed out that the greatest thickening is a little above the middle of ventricle, at the place where the columnæ carneæ are inserted. Thence it decreases suddenly towards the aortic orifice, gradually towards the apex of the heart. Occasionally the reverse obtains, especially in aortic regurgitation (Walshe), and the wall is thicker towards the apex than towards the base.

When the wall of the left ventricle measures three-fifths of an inch in thickness it may be considered hypertrophied. An increase in the average thickness to three-quarters of an inch, or an inch, is not uncommon. An inch and a quarter is occasionally reached, and it is said, an inch and a half, or even two inches. The larger dimensions were probably in cases in which there was little dilatation, and the heart was contracted. In the large heart described by Bristowe, the weight of which was forty-six and a half ounces, the wall of the left ventricle was only $\frac{7}{8}$ths of an inch in thickness at the base; the length of the cavity of the ventricle being six inches.

The right ventricle yields readily to internal pressure, and presents a marked increase in the thickness of the wall much less frequently than the left; simple hypertrophy is very rare. The average normal thickness of the wall is two-and-a-half lines in men, two lines in women. When hypertrophied it is often a third, or half an inch in thickness, and even in rare instances three-quarters of an inch, an inch, and even, it is said, an inch and a quarter (Bertin, 88th case, "eleven to sixteen lines.") The numerous columnæ carneæ are commonly much thickened. The cavity is usually enlarged, but may be lessened in rare cases; probably, however, only in cases of malformation. When the ventricle is thus hypertrophied, and the left ventricle is dilated, the two may, as Morgagni and Bertin remarked, seem to have become transposed.

The left auricle is not unfrequently hypertrophied. The average normal thickness of its wall is one line and a half; where hypertrophied it may reach two to three lines. The right auricle is rarely hypertrophied, and always in least degree. The average thickness of its walls is one line; when hypertrophied it may attain one-and-a-half or two lines. The auricles have never been found to present contraction of their cavity.

In pure hypertrophy the part changed is of firm consistence, firmer than the normal heart, so that the walls do not collapse when cut across. It presents little deviation from the normal color, it is sometimes a little darker. Such unmixed hypertrophy is rare. More commonly the tissue has undergone degeneration, and is paler and softer than normal, sometimes generally, sometimes partially. Rokitansky points out that in the hypertrophy of the ventricles the changed wall of the right ventricle is always tougher than that of the left.

The hypertrophied wall of the heart usually contains more fibrous tissue than the healthy wall. The tissue lies between the primitive bundles, separating them, and here and there forms more extensive tracts. This change, when more considerable, is considered as "fibroid degeneration." It is more abundant in the wall of the right ventricle than in that of the left, and doubtless is the cause of its greater consistence.

The nature of the change in the muscular fibre in hypertrophy has been the subject of much discussion. Does the increase in the size of the heart depend on an increase in the size, or in the number of fibres? The evidence, in some degree conflicting, is on the whole strongly in favor of the view that the increased thickness of the wall is due solely to increase in the number of the fibres constituting it, *i. e.*, to the formation of new fibres. The chief evidence of an increase in the size of the fibres is obtained from the measurements of Hepp,[1] still quoted as authoritative, and who asserted, and gave measurements to show, that the average thickness of the fibres in hypertrophy is about four times the thickness of the fibres in health. This conclusion, however, by itself suggests a fallacy, since the average thickness of the wall in hypertrophy is less than double the average normal thickness. Vogel and Henle, Rindfleisch and Walshe conclude that there is no increase in the size of the fibres, while Robin thinks that there is a slight increase, although not enough to account for the increased size of the heart. Wilks and Moxon are convinced that the chief share in the increase in size is due to increase in number. Considerable weight must be attached to the careful observation of Zielonko,[2] who finds that the average of a large number of measurements of the fibres of hypertrophied hearts is a little *less* than the average of the normal fibre. His observations also corroborate the fact (long before stated by Förster) that the normal fibres are smaller in early than in later life, and are increased in size by good general nutrition. The writer has found on direct enumeration of the fibres in a transverse section of the wall that

[1] Zeitschrift für rat Méd. 1854, p. 257.
[2] Virchow's Archiv, Bd. 62, Heft. I. p. 29.

their number is in the main proportioned to its thickness. The conclusion appears justified that there is no increased size of the fibres in hypertrophy, that the overgrowth of the heart is entirely dependent on the development of new and less perfectly nourished tissue elements. Rindfleisch suggests that they may arise by fissuring of the pre-existing fibres. He has observed that the square cells, of which the muscular fibres of the heart have been shown to consist, contain several nuclei, instead of a single nucleus, as in health.[1]

Symptoms.—Cardiac hypertrophy gives rise to certain distinctive physical signs, and may be accompanied by certain definite symptoms. These signs and symptoms depend on the increased size of the heart, and on the increased force with which it acts. They vary according to the part of the heart which is affected, and according to the amount of dilatation which is associated with the hypertrophy. It will be convenient to consider separately the symptoms of the change in each division of the heart, beginning with the left ventricle. In it the change is carried to the greatest degree, and gives rise to the signs and symptoms commonly understood as those of hypertrophy of the heart.

When hypertrophy is considerable, the heart, unless fixed by adhesions, lies, in consequence of its greater weight, lower in the thorax than in health. The weight of the base is said to increase the natural obliquity of the organ, so that it may assume a nearly transverse position.

Left Ventricle.—*Physical Signs.*—The increased bulk of the heart may cause precordial bulging, noticeable chiefly in the area between the nipple and the left edge of the sternum. The intercostal spaces are widened, and the surface of the chest is more prominent than is the corresponding part on the opposite side. This bulging is most marked in hypertrophy occurring in early life.

The area of dulness is increased. The superficial dulness is usually more extensive, the deep dulness invariably larger, and the increase is chiefly to the left. The left edge of the deep dulness, instead of passing from the middle of the third left cartilage to the apex, extends from the inner extremity of the third rib to the nipple, or even to the anterior axillary line, one, two, or even three inches outside the nipple. It may also, although less commonly, extend upwards to the second interspace. Its shape is thus usually more oval than in health.[2] Resistance on percussion is greater than in health. In extreme enlargement the resonance in the left back is defective, and Walshe has even known the dulness to be so marked, and respiration so weakened by pressure upon the lung, that pleural effusion was simulated. The apex-beat, marking approximately the limit of the heart, is moved outwards and downwards, with its enlargement, into or outside the vertical nipple line, and into the sixth or seventh interspace, into the latter probably only in dilated hypertrophy (Walshe).

The increased form of action manifests itself by increased impulse.[1] The area of impulse is increased; it may be felt in the fourth, fifth, and sixth interspaces. A larger portion of heart comes in contact with the chest wall, and the increased force aids also in producing a more extensive impulse. In pure hypertrophy a maximum apex-beat is still perceptible, bearing a normal proportion to the rest of the impulse. But the impulse is not only more forcible, it presents a special change; it is slower, more deliberate as well as more forcible, and hence has been for long termed "heaving." In dilated hypertrophy the impulse is more abrupt than in simple hypertrophy, in which the slow heave is carried to its greatest degree. The extension of the impulse is often visible, and the whole left front of the chest may be raised by it. It was spoken of as "jarring" by old writers, and still is occasionally so described. But a "jar" implies vibration, and although a vibratory character is often felt in the impulse of a hypertrophied heart, it is due to coexisting valvular disease, not to the over-action of the heart itself. Occasionally a double impulse can be felt with

[1] Pathologiste Gewebelehre, Vierte Aufl. 1875, p. 193.

[2] The deep cardiac dulness is, except in the presence of extreme emphysema, an accurate and convenient measure of the enlargement of the heart. By many authorities it has been strangely undervalued. Niemeyer's assertion that percussion often fails to reveal hypertropy of the left ventricle is comprehensible only in consequence of the guide employed being the superficial dulness, which depends much more on the state of the lung than on the state of the heart. For a very full and clear account of the relations and significance of the diminished resonance caused by the heart in its various conditions, see Balfour, Clin. Lect. on Diseases of the Heart, 1876, Lect. 1.

[1] According to old writers, Fernel, &c., the impulse of a hypertrophied heart had been known to fracture the ribs! All the instances, however, seem to have occurred in convents or monasteries. Cæsalpinus and others assert that two ribs of St. Philip de Neri were torn from their cartilages by the palpitation of his heart. Senac wisely doubted the occurrence of such fractures, unless the ribs had been previously weakened by disease.

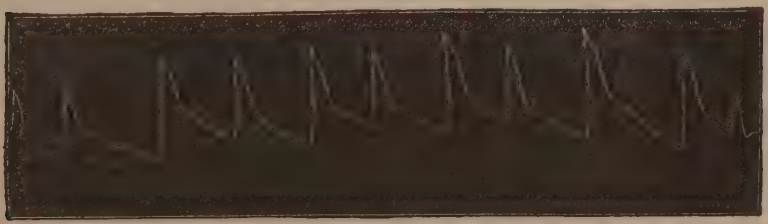
each beat of the heart. Rarely it is a double systolic impulse (Walshe), the origin of which is obscure. More commonly the second and slighter impulse corresponds with the commencement of diastole, at the end of the "sinking back," as Hope expressed it, who first pointed out the phenomenon. He explained it as due to the sudden filling of the ventricles with blood. Hayden, who adopts a similar explanation, has pointed out the coincidence of this second impulse with the second sound. Walshe remarks that the movement is rather a succussion than an impulse against the chest walls. This character, and the obvious coincidence with the second sound, have, in several cases, suggested to the writer the probability that the impulse is really due to the shock communicated to the whole heart by the closure of the aortic valves, a closure rendered more forcible by the greater distension of the aorta. It is in accordance with this explanation that, as Hope and Walshe both point out, this second impulse may occur in simple hypertrophy, but is most marked in dilated hypertrophy (in which the distension of the aorta is greatest), and that it is absent in simple dilatation.

The sounds of the heart are altered. The first sound is rendered less loud, but longer, the change being especially marked over the ventricle. The sound may be normal at the base and ensiform cartilage (Walshe). Sometimes the muffling of the sound amounts almost to extinction. The second sound is usually loud. When dilatation is added to the hypertrophy the first sound becomes louder and clearer. The post-systolic silence is shortened, as Laennec noted, in consequence of the lengthening of the first sound. Laennec thought that this lengthening may amount to a faint murmur, apart from valve disease or hæmic state, and Walshe corroborates the opinion. During attacks of palpitation the first sound may be much more distinct than when the heart is acting uniformly.

Reduplication of the first sound is occasionally met with in hypertrophy: rarely according to Walshe; almost invariably in eccentric hypertrophy, according to Hayden. It is certainly frequent in some forms of hypertrophy, especially in that due to Bright's disease (Sibson). Irregularity in force is not common, in frequency very rare, except in association with dilatation and degenerative changes.

Symptoms, proper.—A great number of morbid phenomena have been ascribed to the influence of cardiac hypertrophy. The list, however, has been shortened according as the symptoms of the causes of hypertrophy and of the other associated consequences of those causes, are distinguished from the symptoms directly due to the hypertrophy itself.[1] Almost all the consequences of dilatation of the heart were formerly ascribed to the conjoined hypertrophy. The credit belongs to Bouillaud of having first vindicated hypertrophy from its supposed influence in causing dropsy and other consequences of cardiac failure.

Fig. 121.



Tracing from pulse in great hypertrophy and dilatation of left ventricle in a case of mitral regurgitant disease, with general venous distension and ultimate increase in arterial tension. Artery large and incompressible. Tracing taken at very high pressure, which did not modify its character.

Subjective symptoms of cardiac hypertrophy may be absent, when the hypertrophy is moderate, with little or no dilatation, and is adequate to overcome the obstruction which has produced it. In such cases, however, the varying force of the heart's action, the varying amount of the obstruction, and the common conjunction of relative weakness with absolute strength, lead to sensible evidence of derangement.

Consciousness of the increased force with which the heart acts is a more or less frequent symptom in all except the slightest forms of hypertrophy. Under excitement the conscious beating may amount to "palpitation." Slight irregularity may increase the discomfort, but

[1] Senac, in speaking of this subject, says: "Rien n'est plus ordinaire que les bévûes des observateurs dans la recherche des causes, tout ce qu'ils trouvent dans les cadavres ils attribuent souvent à la dernière maladie, ou à celle qui a attiré leur attention." Loc. cit. tom. ii. p. 398.

much irregularity or considerable palpitation is rare, except in dilatation, and to that the symptom is to be ascribed. Pain, as Walshe points out, is extremely rare in simple hypertrophy, and anginal attacks are almost confined to cases in which the dilatation is considerable. The force with which the left ventricle contracts has an immediate effect on the arterial pulsations. The carotids throb visibly. The pulse is large, full, hard, and sustained. When dilatation is conjoined with the hypertrophy, the pulse is still full, but is softer and more compressible and less sustained. The sphygmogram shows these characters in a sudden and high rise, and, where the hypertrophy is simple, there is a high and often sustained tidal wave. Where there is coexisting dilatation, the tidal wave may not be sustained in consequence of the imperfect emptying of the ventricle (Fig. 121). Aortic obstruction may, however, modify considerably these characters, rendering the pulse smaller, while it remains hard, sustained, and incompressible. If considerable, it also renders the contraction slow, and the percussion stroke may be lost in a slowly rising tidal wave, as in the accompanying tracing:—

Fig. 122.

Tracings from infrequent and slow pulse of aortic obstruction, with coexisting mitral disease, and hypertrophy of the left ventricle. The slowness of the contraction had been increased by the administration of digitalis. Taken at a high pressure; pulse small but almost incompressible.[1]

The force with which the blood is driven into the smaller vessels may modify the function of certain tissues and organs. The face is often flushed. Tinnitus aurium, flashes of light, and muscæ volitantes may be complained of. Headache and mental dulness are sometimes observed, but as a rule the intellect is unaffected. The general nutrition also suffers little. Organic functions are little interfered with. Increased arterial pressure might be supposed, as Walshe remarks, to modify considerably the urinary secretion, increasing the quantity of water. The urine presents, however, no distinctive change. Swelling of the bronchial mucous membrane, and increased secretion are connected by Niemeyer with the active distension of the bronchial arteries. Shortness of breath on exertion is common, and is by Walshe connected directly with the hypertrophy. True cardiac dyspnœa is rare; and any considerable shortness of breath is probably to be ascribed to the cause of the hypertrophy, or to concurrent dilatation. The pressure exerted on the lungs by an enlarged heart may cause some interference with their function and increase the dyspnœa.

Consequences of Hypertrophy. — A long train of evils which are met with in more or less frequent association with hypertrophy, were formerly regarded as its consequences. Many of them are in no way related to its occurrence, but are the result of the dilatation, or remotely of the cause of the hypertrophy. Such are œdema, capillary engorgement, venous congestions, passive hemorrhages. These were enumerated by Hope as consequences of hypertrophy. Bertin long before taught clearly that they cannot be regarded as such, since they are absent when hypertrophy exists in its most simple form, and occur in proportion as the hypertrophy is complicated by other conditions, such as valvular disease, dilatation of the ventricle, &c., themselves capable, without hypertrophy, of causing the symptoms.

Not only does hypertrophy not produce these effects, but its tendency is to prevent their occurrence. Its power of arresting the mechanical effects of its causes is very great, and proportioned to its degree. Disease of the aortic orifice, for instance, as long as the hypertrophy is great and unweakened by degeneration, produces no backward effect. So in mitral obstruction, hypertrophy of the left auricle may for a long time save the lungs from passive congestion. So, too, hypertrophy of the right ventricle may prevent any over-distension of the venous system from obstruction to the circulation through the lungs or the left side of the heart.

The only morbid effects of hypertrophy which can accurately be thus regarded, are those which result from the greater force with which the blood is driven into the arterial system. These consequences are seen best when there is no impediment to the escape of blood from the ventricle, and especially when the cause of the hypertrophy is occasional or is situated in front of the arterial system. The tendency is for the due proportion between the contents of the arteries and the veins to

[1] These two tracings were taken for me by Mr. H. R. O. Sankey.

be disturbed, for the arteries to become over-filled, the veins and the pulmonary system under-filled with blood. It has been said that the whole circulation is accelerated, but this can only be the case when the action of the heart is for the time being more than enough to overcome the resistance which has evoked it.

It has been supposed that the increased supply of arterial blood may lead to the overgrowth of organs, but the conjecture is unsupported by observation.

A more direct effect upon the vessels may often be traced. When the obstruction is situated beyond the arteries, their walls are exposed to a greatly increased pressure. The same effect occurs when the obstruction is at the aortic orifice, and the action of the heart is from any cause (as dynamic excitement, or the cessation of another cause) in excess of the obstruction. Both the large and small arteries suffer under these circumstances. The increased pressure on the aorta may cause its dilatation, although, as Senac observed, the enlargement from this cause is not often considerable. A more frequent consequence of the pressure to which the arteries are exposed is seen in the degeneration of the vessels.[1] Modern observation has established the frequent association of so-called endarteritis deformans (atheroma) with increased strain. The change is seen in the aorta, in the pulmonary artery, and in the smaller vessels, especially in those in which the relative pressure is the greatest, as in those at the base of the brain.

But degeneration is not the only effect of the increased strain upon the vessels; they not unfrequently give way, and hemorrhage results. Hemorrhage into the brain is, on account of its frequency, magnitude and importance, that form to which attention has been chiefly directed. The frequent association of apoplexy and enlargement of the heart led Corvisart first to assume a causal relationship between the two.[2] In this he has been followed by most subsequent writers—Bertin, Hope, Bouillaud, Andral, Burrows, and others. But the conclusions of the earlier observers require some abatement in the light of modern knowledge of the frequency with which apoplectic attacks result, by another mechanism, from cardiac disease. Embolism may give rise to symptoms not unlikely those of cerebral hemorrhage, and embolism is almost constantly associated with hypertrophy of the heart. But even when these cases are eliminated from the discussion, the pathological evidence of the association of apoplexy and hypertrophy of the heart is still impeachable. In sixty-five cases of apoplexy collected by Quain[1] the heart was enlarged in two-thirds, and in one-half there was no valve disease. The significance of the latter fact is that in these cases the cause of the hypertrophy was probably situated away from the heart, in or beyond the arterial system, which would thus have to bear the whole force of the over-acting heart. But this is the condition in which arterial disease is produced; the small vessels degenerate, and, becoming weaker, are less able to bear increased pressure to which they are exposed. This is the case, notoriously, in Bright's disease, especially in the contracted kidney, with which cerebral hemorrhage and cardiac hypertrophy are so constantly associated. In primary degenerative changes in the smaller vessels the same result is seen—a like obstruction may cause hypertrophy, and a like weakness yield before the increased pressure. The same sequence is sometimes seen when the cause of the obstruction is situated beyond the arteries and capillaries, and acts, it may be, through both systems of the circulation. Mitral disease may lead to extreme blood tension in the arterial system, as the sphygmographic tracing on page 777 shows. Cerebral hemorrhage sometimes occurs in such cases, even in the young, from the rupture of an overstrained artery.

All authorities are agreed as to the causal relationship between hypertrophy of the heart and the rupture of diseased vessels. But to this some, as Watson, Eulenberg, Rokitansky, would limit the connection. It must be considered still doubtful whether an over-acting heart can rupture a healthy artery. It is true the large arteries of the brain are often found healthy in cases of cerebral hemorrhage, but this affords only slight evidence of the condition of the smaller vessels of the cerebral substance. These are frequently diseased when the vessels at the base of the brain appear healthy.

Statistics on this point corroborate, but do not extend, the conclusion from iso-

[1] Pointed out by Kirkes in 1857, Med. Times and Gaz. No. 370, 371.

[2] "Where apoplexy takes place in a person in whom there is an excess of muscular substance and strength in the heart, it is easy to conceive that the resistance of the vessels of the brain is not in unison with the extraordinary impetus which the heart impresses on the blood; it necessarily follows that the smaller vessels of the brain become more permeable to this fluid, or that they give way and cause effusion and apoplexy." Corvisart, l. c., p. 164. It, however, would seem to have been first suggested by the death of Malpighi, who died from cerebral hemorrhage, and whose heart was found greatly hypertrophied, "the parietes of the left ventricle were two fingers in thickness." (Baglivi.)

[1] Lumleian Lectures, loc. cit.

lated observations. Quain found that diseased vessels are more common in cases of cerebral hemorrhage when the heart is healthy than when it is hypertrophied. They are present in two-thirds of the former, and only in about half of the latter. The inference suggested is that, since extensive disease of vessels shown by the implication of the larger trunks is less common, in cerebral hemorrhage, when the heart is hypertrophied than when it is healthy, an over-acting heart needs less diminution in the strength of the vessels, in order to effect their rupture, than a healthy heart.

The occasional, though rare, occurrence of cerebral hemorrhage in the young does not help to decide the question. Disease of the cerebral vessels is now known to be not uncommon in early life, and some of the cases occur in the subjects of heart disease, in whom there exists circuitous increase of arterial tension just described. Moreover in such subjects cerebral aneurisms, perhaps from imperfect embolism, are frequent, and in many cases it has certainly been by the rupture of such an aneurism that cerebral hemorrhage has occurred.

Concentric Hypertrophy of the Left Ventricle.—The symptoms are as uncertain as the existence of the malady. Theoretically, the signs of simple hypertrophy might be expected, and with them some dyspnœa in consequence of the impediment which must be presented to the passage of blood through a heart so much lessened in capacity.

Right Ventricle.—Considerable increase in the size of the right ventricle causes prominence of the lower part of the sternum, epigastric fulness, and often bulging of the lower left cartilages adjacent to the sternum. The superficial cardiac dulness is little changed, but the deep dulness extends further to the right than normal, the right edge being one or two fingers' breadth to the right of the sternum. This dulness is partly dependent on the enlargement of the ventricle, partly on over-distension of the auricle, which always accompanies the change in the ventricle. Pulsation may be felt at the epigastrium. The apex-beat is in its normal situation, or moved a little to the left, extended as far as, but not beyond, the nipple line. It is frequently changed, being obscured and diffused when the right ventricle lies in front of it. A distinct impulse may be felt over the right ventricle, *i. e.*, over the lower part of the sternum,[1] and in the adjacent left interspaces. In health a distinct impulse is very rarely to be felt in this situation. In hypertrophy the impulse may have considerable strength, but it is generally quick, rarely of the slow, heaving character which hypertrophy of the left ventricle produces. It may sometimes be felt as far as the base. Little alteration in the sounds of the heart is caused by hypertrophy of the right ventricle. The pulmonary second sound is usually intensified by the increased tension within the pulmonary artery. Sometimes the second sound is reduplicated. Jugular pulsation has been associated with hypertrophy of the right ventricle by Lancisi, Laennec, Hope, and others. It is doubtless due to actual regurgitation through the tricuspid orifice, and coexisting dilatation of the ventricle is necessary for its production.

Few symptoms proper can be associated with the condition. The pulse is natural. The venous system shows no signs of engorgement. It is remarkable how completely hypertrophy of the right ventricle will prevent the development of dropsy, and other signs of venous stagnation, by an obstacle in front of it. The lungs or left side of the heart usually present evidence of the condition causing the hypertrophy, emphysema, disease of the mitral orifice, &c. Dyspnœa is common, as Walshe points out, but is more frequently the result of the cause of the hypertrophy, than of the hypertrophy itself.

Consequences of Hypertrophy of the Right Ventricle.—The immediate effect of over-action in the right ventricle is to over-distend that part of the pulmonary vascular system which lies between the ventricle and the obstruction which has caused the hypertrophy—the pulmonary arterial system, when obstruction is in the tissues of the lung, the pulmonary veins also, when the obstruction is on the left side of the heart. Atheroma of the pulmonary artery frequently exists in conjunction with this condition, and has been regarded as causal, but in few cases have the two been observed except in conjunction with some other recognized cause of such hypertrophy, and it seems more reasonable to conclude that the degeneration is the result of the increased strain to which the pulmonary artery is exposed. Where the degeneration is considerable and of old standing, as in cases in which the artery is found calcified, it may be the only discoverable cause of a moderate hypertrophy of the ventricle.

Pulmonary congestions, œdema, and especially pulmonary apoplexy, have, since the days of Bertin, been commonly ascribed to hypertrophy of the right ventricle. Where the obstruction causing the hypertrophy is situated on the left side of the heart, the increase in the strength of the right ventricle will add considerably to the strain upon the distended pulmonary vessels, and may constitute the efficient cause of their rupture.

[1] This was pointed out by Burggrave in 1754 (Act. Acad. Nat. Cur. vol. x. p. 140).

Modern pathological research, however, has shown that diseases of the right side of the heart frequently cause pulmonary apoplexy in another way, by leading to pulmonary embolism. We are only beginning to learn how large a proportion of pulmonary apoplexies is due to this cause. When such embolism occurs, hypertrophy of the right ventricle will increase very much the strain on the collateral circulation, and, in consequence, will augment the amount of hemorrhage.

AURICLES.—*Hypertrophy of the Left Auricle* is usually attended with evidence of its enlargement, *i. e.*, dulness, commonly relative only, in the inner part of the second left interspace. Less frequently a distinct impulse is to be recognized in this situation, preceding in time the ventricular impulse and due to the auricular systole. Evidence of mitral disease is commonly present, a systolic or presystolic murmur. Dilatation of the auricle invariably coexists. No symptoms are known to be associated with the hypertrophy. Its tendency is to prevent the mitral mischief from influencing the pulmonary circulation.

Hypertrophy of the Right Auricle is very rare, and is always associated with dilatation. Its signs are dulness to the right of the sternum in the third and fourth interspaces, and, in very rare cases, an impulse, presystolic in rhythm, in this situation. It is often attended with marked jugular pulsation, and with the evidence of disease of the right ventricle or of the tricuspid orifice.

DIAGNOSIS.—The diagnosis of hypertrophy depends on the recognition of increased force of impulse, and especially, in the case of the left ventricle, by the deliberate heaving character which indicates the contraction of a large mass of muscle. In the case of right ventricular and of auricular hypertrophy, the increased force is indicated by impulse, where in health none is present. Evidence of enlargement of the heart, by percussion dulness, or by movement of the apex-beat, or by extension of the impulse, is usually also obvious. In left ventricular hypertrophy the character of the pulse assists the diagnosis. Where doubt remains, the presence of a morbid state, capable of causing the hypertrophy, may afford evidence of its probable existence.

In judging of the existence and degree of hypertrophy the condition of the lungs must always be taken into consideration. Considerable emphysema may conceal all the signs of a hypertrophy of high degree: The impulse may be imperceptible, the dulness masked, and the heart-sounds weakened. The existence of hypertrophy must then be inferred from the condition of the arterial system.

There are certain conditions from which hypertrophy has most frequently to be distinguished.

Undue Exposure of the Heart in very flat- or narrow-chested persons, with small lungs, may simulate hypertrophy. The heart comes into more extensive contact with the anterior wall of the chest than in health. Its impulse is felt over a larger area, and may appear to have undue force. A maximum apex-beat is still preserved. The superficial dulness is more extensive than in health. The distinction from hypertrophy rests on the absence of a heaving character in the impulse, on the normal or nearly normal position of the apex-beat (it is never outside, though it may be in the nipple-line), on the natural extent of the deep dulness, on the unchanged pulse, on the absence of any causal condition, and on the recognition of the short antero-posterior or transverse diameter of the chest. The difficulty of diagnosis in such cases is sometimes increased by the presence of an exocardial murmur, produced by the undue friction of the heart against the bony chest wall, and, by the circumstance, that patients with very flat chests are often weakly and anæmic, and suffer from shortness of breath and extreme consciousness of any dynamical heart-disturbance.

Dynamical Disturbance of the Heart may be mistaken for hypertrophy. Under excitement the heart may beat with apparently increased force, and be brought into abnormal contact with the wall of the chest, so that there is an increase in the area as well as in the force of the impulse. Sometimes the increase in force is more apparent than real, and the pulse is small and weak. Frequently, however, the rapidly-acting heart distends the arteries, and the pulse becomes hard and full. There is an entire absence of the deliberate heave of hypertrophy, and of evidence of permanent change in the form of the heart; there is no bulging, no increased dulness. Rest in the recumbent posture soon reduces the impulse to the normal. It must not be forgotten that an hypertrophied heart readily palpitates under excitement, and any irregularity in the excited action is ground for suspicion.

Displacement of the Heart may lead to an apparent extension (really a movement) of the impulse and dulness in a given direction. Displacement to the left, moving the apex outside the nipple line, may simulate hypertrophy of the left ventricle; and displacement downwards, rendering the impulse of the right ventricle perceptible at the epigastrium, may resemble dilated hypertrophy of the right ventricle. But under these circumstances

there is no alteration of the character of the impulse as there is in hypertrophy; the opposite boundary of the heart may be found to have undergone a corresponding change of position, and a cause of the displacement will be discoverable.

Dilatation of the Heart resembles hypertrophy in causing increase in size, shown by extension of dulness and increased area of impulse. The impulse is, however, diffuse and weak; the proportional intensity of the apex-beat is lost, the pulse is soft, and the action of the heart often irregular. The distinction between the two conditions can rarely be absolute, since they are usually, in varying degree, conjoined.

Pericardial Effusion may cause bulging and an increase in the area of dulness. The impulse, however, is less, instead of more forcible; and the apex is raised, instead of being moved outwards or downwards. The dulness extends upwards in the pyramidal form, and its left boundary is beyond, instead of corresponding to, the left limit of the impulse. Apart from the auscultatory signs, the acuteness of the symptoms in pericarditis, sudden pain, dyspnœa, fever, will usually prevent an error in diagnosis. In auricular hypertrophy, the extension of dulness above the normal limits of the cardiac dulness is usually slight. If sufficient to stimulate pericardial effusion, a pre-systolic impulse will, in most cases, be detected.

Aneurism has been confounded with hypertrophy, but the conditions under which such a mistake could arise must be very rare. The double centre of pulsation usually affords a sufficient distinction.

Local diagnosis of the part of the heart affected with hypertrophy has been alluded to in describing the symptoms produced by the change in the several chambers of the heart. The chief difficulty arises in some cases of ventricular hypertrophy. In hypertrophy of the right ventricle, slight hypertrophy of the left ventricle may be concealed or simulated by the strong impulse of the anterior right ventricle and the displacement outwards of the apex-beat consequent on the enlargement of the right side. The diagnosis of the state of the left ventricle must then depend on the character of the apex-beat—on the presence or absence of a distinct heaving impulse. On the other hand, considerable hypertrophy of the left ventricle may cause an impulse over the position of the right ventricle. In such a case the impulse of the left ventricle possesses great force, and the diagnosis must be based on the relative proportion of the impulse over the two ventricles.

In all cases a comparison of the extent of causal lesion, with its mechanical effects, will often suggest an accurate opinion as to the existence and degree of hypertrophy when the part affected is not accessible to physical examination. For instance, congestion of the lung is the necessary mechanical effect of mitral constriction. The absence of such congestion, when considerable mitral constriction exists, is valid ground for suspecting compensatory hypertrophy of the left auricle. So, too, we sometimes find that such compensation has not occurred—that the lungs are constantly overloaded with blood, but that the general venous system has not suffered; the jugular veins are undistended: there is no anasarca or albumen in the urine. In such a case we may be sure that there is hypertrophy of the right ventricle.

Prognosis.—The difficulty of extricating hypertrophy from the various conditions with which it is associated has led authorities to entertain very different opinions regarding its influence on the life and well-being of the patient. The gravest consequences of hypertrophy (as formerly described) are now known to be those of its attendant conditions; the "conservative" character of hypertrophy, as a healthy reaction against a morbid influence, is generally recognized, and its prognosis is admitted to be, as a rule, favorable; any unfavorable element being due rather to coexisting dilatation, or to other effects of the cause of the hypertrophy, than to that condition itself.

It is rarely that evil results can be traced directly to the overgrowth of the heart. The unpleasant sensations attending the action of a hypertrophied heart suggest many possible evils which experience rarely justifies. It may produce hemorrhage, especially into the brain, when vessels are rotten, but probably does not rupture healthy vessels; it may render inflammations more severe, but never initiates them. Most observers will share Walshe's profound doubt whether in its most extreme forms, hypertrophy can never *per se* lead to death.

Does hypertrophy ever diminish or disappear? It is probable that hypertrophy lasts as long as its cause exists. Many facts on record support the opinion that, if the cause of simple hypertrophy cease to act, the heart gradually resumes its normal size. Atrophy may occur in an hypertrophied as readily as in a normal heart. Whether a heart the subject of dilated hypertrophy ever regains its normal volume is doubtful. The occurrence of the so-called concentric atrophy, in which the size of the cavities lessens, and also the disappearance of the dilatation of atony, support the idea that a moderately-dilated heart may regain its normal size.

The prognosis in hypertrophy must, therefore, largely depend on the extent to which its cause is removable. For practical purposes the work of the heart in these cases may be divided into three categories: (1) that which is required to carry on the healthy circulation, the body being at rest; (2) superadded work, temporary and variable, such as is involved in muscular exertion, emotional excitement, local inflammation, pregnancy, &c.; (3) some permanent abnormal resistance to the movement of the blood, increasing the pressure within the cavity affected. The second of these is alone amenable to treatment. The chance of removing or curing hypertrophy depends on the extent to which causes of this class constitute the work of the heart. Where hypertrophy is developed when the work of the second class is as slight as possible, where no avoidable exertion is made, and where no occasional causes of obstruction exist, the chance of removing or lessening hypertrophy is small. In the rare cases in which the whole increase over the normal work of the heart depends on causes of the second class, the prognosis is the most favorable. Such cases are sometimes met with among athletes, as in an instance Walshe records.

The probable permanence of the hypertrophy on the one hand, the likelihood that it may give place to dilatation on the other hand, must influence the prognosis in any individual case. This probability must be estimated by the degree to which the causes of dilatation are, or are likely to be, in operation. Impaired general health, or the presence of degenerative tendencies, local or general, render the prognosis less favorable.

Where the cause of hypertrophy is permanent, the influence of the hypertrophy varies, and with it the prognosis. In certain conditions the increased force with which the heart acts may lead, directly or indirectly, to evil consequences, and in such cases the presence and degree of the hypertrophy may entail, *per se*, a corresponding increase in the gravity of the prognosis. In all forms of valvular disease in which the hypertrophy depends on direct obstruction to the escape of blood from the cavity, the hypertrophy is purely beneficial in its effect; it secures a due supply of blood to the parts beyond the obstruction; it saves the vessels and organs behind from suffering from the impediment. It is only when hypertrophy is due, in part, to a variable cause beyond the obstruction, that it may be occasionally in sufficient excess to produce prejudicial arterial distension.

In cases of regurgitation, in which the heart has to exert undue force in the propulsion of an undue quantity of blood, the hypertrophy is less simply beneficial in its influence. The muscular force with which the ventricle contracts tends to increase the amount of blood regurgitated, and so increase its own repletion. This is the case directly in aortic regurgitation, indirectly in mitral regurgitation. The degeneration of the arteries is hastened by the strain to which they are exposed by the action of the hypertrophied ventricle in aortic regurgitation; while in mitral regurgitation, although the stronger action of the ventricle may drive a larger quantity of blood into the aorta, it also increases the amount regurgitated through the incompetent valves. But it must be remembered that in these cases the hypertrophy is a substitute for dilatation, and may be accepted as the less of the two evils; or it counteracts the influence of dilatation which coexists.

When the obstruction causing the hypertrophy is situated in the vascular system, pulmonary, or systemic, whether the consequence of degeneration, Bright's disease, &c., the hypertrophy is also less simply beneficial, since the increased strain to which the vessels are subjected increases their liability to degeneration and rupture.

In Bright's disease this danger reaches its height, since degeneration of the strained vessels is very apt to occur and renders their rupture easy. In senile changes cardiac and vascular degeneration often correspond, and the hypertrophy which at first is evoked by the change in the vessels yields to dilatation, by which the blood-tension is lessened. But this retro-compensation is not without new risks.

In all cases, however, it is still true that the prognosis of the hypertrophy is subordinate to that of the lesion causing it, and also to that of coexisting dilatation. Once established as the result of a permanent cause, it usually increases, and bears a simple proportion to its cause. It is extensively employed in prognosis, but is used rather as an indication of the extent and gravity of the lesion causing it than as affording in itself much information. As far as it goes, its presence renders the prognosis of the causal lesion better. Compensatory in its action, it wards off evil and promotes health.

TREATMENT.—Current opinion as to the treatment of hypertrophy has undergone great changes, in accordance with the altered ideas of its relation to the common consequences of organic heart disease. When most of these were considered to be the direct effects of the over-acting heart, every effort was made to diminish its over-action and to lessen its over-growth. Low diet and frequent

bleedings are the measures which Albertini and Valsalva handed down to a long series of their successors; and the effects of their doctrine is even now to be traced, although perhaps rather in the pages of text-books than in practice. Even after the purely consecutive nature of hypertrophy was clearly recognized by Bertin, the same treatment was advocated.

The judicious management of hypertrophy depends on the recognition of the fact that it is sometimes purely beneficial, usually welcome as a substitute for its too frequent associate, dilatation, and rarely directly prejudicial. No universal rule for the treatment of hypertrophy can therefore be laid down, since the proper course may be sometimes to foster its occurrence, sometimes to lessen its excess, or, failing that, to prevent its increase.

Hypertrophy of the heart being the result of two factors, nutritive activity and increased work, its increase may be to some extent prevented, and its amount diminished, by lessening each factor in its production. The nutritive activity of the heart can be lessened only by diminishing that of the general system by low diet, bleeding, &c. But to attempt this while the causes of hypertrophy continue, is to substitute dilatation for hypertrophy. The system has been advocated, however, in conjunction with causal treatment, from the time of Bertin. It may be questioned whether the causes of established hypertrophy can ever be sufficiently reduced to permit the safe employment of "antiphlogistic" measures. Moreover they can rarely be necessary. We see in the voluntary muscles that reduction of work is invariably followed by reduction in size of muscle. Every analogy suggests that cardiac hypertrophy will rapidly subside when the condition which excited it has lessened or has ceased. It is not often that this result can be proved to occur in the case of the heart, but instances are not infrequent in which it seems to take place. The reduction of the causes of hypertrophy, *i. e.*, the work of the heart, to a minimum, constitutes, then, the main object in the treatment of hypertrophy. This work is partly of a constant, partly of an occasional nature. The normal work of the circulation must be carried on; the permanent organic cause of the hypertrophy can rarely be lessened; but the occasional addition to the heart's work involved in violent muscular exercise, increased frequency of contraction from alcohol or emotion, increased obstruction from remediable states of blood or local inflammations, may all be to a large extent removed. Rest of body and mind is therefore the first and most essential element in treatment. All exercise which quickens the pulse must be absolutely forbidden.[1] Emotional tranquillity must be as far as possible secured. The utmost temperance in food and alcohol should be enforced. A fair amount of nitrogenous food, and a very little light wine with it, constitute the best diet. If food is well taken without alcohol, the latter may often with advantage be prohibited. The digestive organs should be carefully attended to. Nothing disturbs the action of the heart so readily as a distended stomach. Food must be moderate in amount, and every cause of transient plethora avoided. The secretions must be carefully regulated, and impaired action of the kidneys or the skin must be supplemented by mild purgation or diuresis. Local inflammations, bronchitis, &c., must be carefully guarded against, and when they occur, removed as speedily as possible.

Too often, however, the amount of obstruction which can by these means be removed bears but a small proportion to the total against which the heart has to contend. Can this permanent obstruction be further reduced? To some extent the work of the heart can always be lessened by reduction in the total quantity of the blood. This formed an important element in the old system of treatment, and it was partly with this object that frequent and repeated bleedings were recommended. Their condemnation in the present day is superfluous. It may be doubted whether occasional leeching, which still finds advocates, is justified by its ultimate results, although its immediate effect is to give relief to the heart. Restriction of fluids has been suggested. It is at any rate a harmless measure; but the rapidity with which urinary secretion regulates the volume and density of the blood renders it doubtful whether more than a very transient effect is produced.

It will be gathered from these statements that the conditions under which an attempt at the removal of hypertrophy is indicated are very rare. Whenever the hypertrophy can act immediately on the causal resistance, its influence is always, on the whole, and sometimes entirely beneficial. Only when the over-action of the heart is primary, or is due to a cause which has ceased to operate, is it to be attacked directly. In the rare instances in which violent exercise has called out persistent hypertrophy, or some obstruction has been removed, the condition may call for immediate treatment to reduce its effect. Where the obstruction is situated far from the heart, and degenerated ves-

[1] "On doit regarder le repos comme un remede préservatif; mais ce repos n'exclut pas un exercice modéré, lorsque les grands accidents sont calmés."—Senac, l. c. p. 419.

sels are interposed which have to bear the full force of an over-acting ventricle, as in Bright's disease, the question also sometimes arises of the chances of evil from vascular rupture, on the one hand, and from a weakened heart on the other. The certain, slow, but sure evil of a weakened heart will generally counterbalance the possible catastrophe, and any attempt to lessen the cardiac strength will be avoided.

The use of drugs in hypertrophy is a subject on which various opinions have been held.[1] Most observers agree with Walshe, that the reduction of the bulk of the heart is beyond the direct power of any drug. The chief *rôle* of medicine lies in regulating the cardiac contractions and in freeing the circulation from removable causes of embarrassment. Frequent action involves a great increase in the work of the heart. Force is needed, it has been stated already, to move the heart, apart from the movement of the blood. The minimum frequency consistent with the due supply of blood to the system gives the heart its best conditions of action. Moreover, very frequent action may fill the arteries to repletion, and so increase their distension as greatly to augment the intra-cardiac pressure. Lastly, frequent action lessens the total rest of the heart, and favors degeneration. No remedy has been discovered which lessens the undue frequency of the action of the heart so effectually as digitalis. But digitalis strengthens the cardiac action, and hence its use in hypertrophy has been discountenanced by most modern writers, and by some strongly condemned.

The experience of clinical observers is not, however, in complete accord with theoretical conclusion. By many the value of digitalis in hypertrophy is strongly asserted. One explanation for this may lie in the fact that hypertrophy is so rarely simple. Almost invariably, dilatation is conjoined with it. In dilatation, digitalis is of extreme value, and its use in hypertrophy is to a great extent proportioned to the existence and amount of dilatation. Moreover all irregular action of the heart involves waste of force, involves useless work. Too frequent contraction does the same. Each may generally be controlled by digitilas. Even where there is no irregularity and little dilatation, the cardiac action may be below the actual needs of the system; the compensation is insufficient, and the additional strength of contraction imparted by digitalis is purely useful. The dose of digitalis needed in these circumstances is smaller than that required in dilatation. Five minims of the tincture, or a drachm of the infusion, three times a day, will usually effect all that is required. A larger dose, is, as Milner Fothergill states, much more frequently deleterious than in dilatation, in which large doses are borne, not only with impunity, but with advantage. In pure hypertrophy, digitalis is rarely necessary. Veratrum viride has been used, especially in America, to reduce the strength of the heart, when in hypertrophy its force appears beyond the present need of the system. Doses of five minims of the tincture may be given three times a day. Both the force and frequency of the heart's action are reduced. Inunction of Ung. Veratriæ has also been employed for the same purpose.

Where hypertrophy is not pure but is great, and acts directly on the vascular system, or tends to increase its cause (as in aortic regurgitation), it may be necessary, by similar measures, to reduce the force of the heart to a minimum necessary for the work of the circulation. Digitalis has been employed in small doses and recommended strongly by B. Foster, but most authorities discountenance its use under these circumstances, and Ringer[1] points out that the same end may be attained by small doses of aconite. A combination of aconite and veratrum is recommended by H. C. Wood.[2]

The consciousness of the cardiac contractions, which constitutes so troublesome a symptom of hypertrophy, is only in part due to the force with which the heart acts. It is much more frequently the result of irregular or too sudden contractions, and related to coincident dilatation rather than to hypertrophy. It is commonly controlled by rest and digitalis.

For the relief of cardiac pain, direct sedatives may be needed. Opium, or morphia, is very effectual. Aconite is strongly praised by Walshe. Belladonna, Indian hemp, hydrocyanic acid are also useful in some cases. The Virginian prune bark, which contains hydrocyanic acid, is sometimes useful, but its tonic properties render it more suitable for dilatation. Cold locally applied to the cardiac region is strongly recommended by Niemeyer.

The treatment of hypertrophy of the right ventricle must be conducted on the same general principles as that of the left. It is almost always united with dilatation, and is never excessive. Hence it needs as far as possible to be strengthened, both absolutely by tonics, digitalis, &c., and relatively by diminishing its work, by lessening as far as possible the

[1] Their possible use seems to have occurred to the French school of physicians at the beginning of this century, although the chief cardiac medicine, digitalis, had long before been employed in this country.

[1] Handbook of Therapeutics, fifth edition, p. 427.

[2] Philadelphia Med. Times, 1874, Nov. 14 and 21.

obstruction to the movement of blood through the lungs, and by the avoidance of over-exertion, &c. Hypertrophy of the auricles rarely calls for special treatment. Never simple, the conjoined dilatation always predominates.

The more detailed treatment of dilated hypertrophy is described in the next article, on Dilatation of the Heart.

DILATATION OF THE HEART.

By W. R. Gowers, M.D.

Synonym.—Enlargement of the Heart (old writers); Aneurism of the Heart (Baillou, Lancisi); Passive Aneurism, or Passive Dilatation (Corvisart); Herzerweiterung (Freysig); Cardiectasis (Jaccoud).

Definition.—Increase in the size of one or more of the cavities of the heart. Such increase in size may or may not be attended with obvious thickening or thinning of the cardiac walls.

History.—Dilatation of the heart received much attention from the earlier pathologists, being rightly regarded as the chief cause of its enlargement. In the middle of the sixteenth century, Vesalius gave an account of a heart, the left ventricle of which contained two pounds of blood, and Baillou[1] mentioned one that equalled in size a man's head. Harvey[2] also in 1628 alluded to this condition. Dilatation of the auricles was described by Willis. Dilatation of the right ventricle and left auricle, as the result of mitral constriction, was described by Mayow in 1674. Vieussens,[3] in 1715, described a case, observed in 1695, of extreme dilatation of left ventricle, the consequence of aortic regurgitation. Peyer, Lancisi, and all successive writers alluded to, or related instances of the condition. The first systematic account of its mechanism and causes was given by Senac[4] in 1749, who distinguished dilatation with and without thickening of the walls. Morgagni,[5] in 1779, described very clearly its origin, and effect on the circulation. Several cases were related by Ferriar,[6] in 1792, and the general causes and symptoms of dilatation were described by Allan Burns, in 1809. In France, after the writings of Lancisi had given the word currency, Baillou's term "aneurism," had been used to designate enlargements of the heart, as well as of the great vessels. Corvisart, in his description of dilatation in his work, published in 1806, designated the two varieties described by Senac, "active" and "passive" aneurism, with a subprotest against the application of the term to conditions with such different tendencies. He described accurately, as far as the description went, the different symptoms and tendencies of the two conditions, and pointed out the association of dropsy, or the "serous diathesis," with dilatation, rather than hypertrophy. A further account of dilatation of the left auricle as a mechanical consequence of mitral constriction was given by Abernethy in 1806.[1] Dilatation consequent on carditis, and associated with adherent pericardium, was described, as the result of articular rheumatism, by Sir W. Dundas in 1808.[2] Its varieties were recognized a little later by Kreysig. Bertin, in 1811, distinguished the conditions and processes of dilatation and hypertrophy (in the sense in which the words are now used), and Laennec's work on Auscultation, published in 1819, gave the terms authoritative use. In Bertin's systematic treatise, edited by Bouillaud in 1824, the chief varieties were distinguished which have since been generally recognized.

The detection, by percussion, of enlargement of the heart, of which dilatation is the chief cause, is due to Avenbrugger (1763); that of the altered impulse by palpation, to Corvisart (1806); that of the auscultatory signs, to Laennec (1820).

Varieties. — From the condition of the cardiac walls, their increase or dimin-

[1] Epidemics et Ephemerides, 1574. Yvaren's Trans., Paris, 1858, p. 289.

[2] De motu cordis et sanguinis.

[3] Traité du Cœur.

[4] Traité de la Structure du Cœur, &c., tom. ii.

[5] De Sed. et Caus. Morb., Epist. xxvii.

[6] Medical Histories and Reflections, by John Ferriar, M.D., vol. i. 1792, p. 144.

[1] Med.-Chir. Trans. vol. i. p. 27.

[2] Ibid. p. 37.

ution in thickness, certain varieties have long been distinguished.

(1) *Dilatation with Hypertrophy* (active aneurism of Corvisart), in which the walls are increased in thickness, as well as the cavities in size.

(2) *Dilatation with Attenuation* (passive aneurism of Corvisart), in which the cavities are increased in size, while the walls are reduced in thickness. To these Bertin proposed to add that of *simple dilatation*, in which the dilated walls preserve their normal thickness, and *mixed dilatation*, in which the walls are in one place increased, in another diminished, in thickness. These varieties have been adopted by most subsequent writers. The name, "simple dilatation," cannot, however, be considered an accurate designation of the condition which it denotes, dilatation without hypertrophy of tissue. If a heart be dilated only, its walls, extended in area, are necessarily lessened in thickness. For the normal thickness of the walls to be preserved when the cavity is dilated, overgrowth of tissue must occur. Thus the condition of "simple dilatation" necessarily produces dilatation with attenuation, while the state to which the term is applied is really dilatation with moderate hypertrophy: this was shown clearly by Stokes. Forget applied to the condition the term *hypertrophie dilatoire*. Many writers have suggested, and Wilks and Moxon maintain, that pure dilatation never occurs, that hypertrophy is the invariable accompaniment, as the increased weight testifies, and that recorded examples of hearts dilated and not increased in weight have been examples only of relaxation. They prefer the simple distinction into dilatation with thickening, and dilatation with thinning.

Other varieties which have been distinguished are those of *general dilatation*, in which all four cavities of the heart suffer, and *partial dilatation*, in which the change is confined to one or some of them. It has also been proposed by Hayden[1] to designate those cases in which an obvious active cause of dilatation can be distinguished, *consecutive*, and those in which no such causes exists, *primary*. Lastly, dilatations have been classified as *temporary* or *permanent*. Bertin suggested that the latter only should be included under the term, the temporary forms being rather examples of distension than of dilatation.

CAUSES.—The maintenance of the normal size of the heart ultimately depends on the existence of a due proportion between its elastic and contractile force, and the blood-pressure to which it is exposed in passive resistance and active contraction. A disproportion between these two forces is the ultimate cause of its dilatation; such disproportion may result from a change in the amount of either factor, an increase in the blood-pressure, a decrease in the cardiac strength. Often the two conditions are conjoined; a weakened heart yields before an increased pressure, and thus becomes over-distended; and the conditions being permanently dilated. To these two causes must, probably, be added the effect of traction from without, which acts by lessening the effect of the contractile force of the heart, and so corresponds in its action with the weakening of the wall.

Thus diminished strength of the walls of the heart constitutes a predisposition to dilatation, and the causes of that weakening may be considered as the *predisposing causes* of dilatation; the endocardial pressure being regarded as the *exciting cause* of the dilatation. But, as is the case with many predisposing causes of disease, the weakness of the wall of the heart may be the only morbid antecedent. Moreover, the action of these two causes of dilatation is not simply predisposing and exciting. It will be convenient, however, to consider the mechanism of their action after they have been described in brief detail. The antecedents of the predisposing and exciting causes may be spoken of as the *remote causes* of dilatation.

It must be remembered also, that increased endocardial pressure is the immediate cause, not only of dilatation, by its mechanical effect, but of hypertrophy, by the vital reaction which it induces. Its effect in producing dilatation is influenced in part by the existence of the predisposition (weakness of the cardiac wall), in part by the conditions under which it acts, and which may be regarded as *determining causes*. Commonly, however, the double tendency of the increased pressure results in the double effect, and hypertrophy and dilatation are conjoined. We have thus four classes of causes to consider, the remote, predisposing, exciting, and determining causes.

(A.) *Remote Causes.*—The general conditions of hereditary influence, age, sex, occupation, previous illness, etc., enter largely into the causation of dilatation of the heart, as the antecedents of the conditions on which it immediately depends. They can only be fully understood when the immediate causes are known. *Hereditary taint* has a powerful influence in disposing to special degenerations and to certain diseases, such as acute rheumatism, on which the immediate causes largely depend. *Age* has a similar influence. Degenerative changes are concerned in the production of both causes of dilatation, and hence the disease increases in frequency with advanc-

[1] Diseases of Heart and Aorta: Dublin, 1875, p. 558.

ing years. *Sex* influences the occurrence of dilatation by determining exposure to one of the commonest causes of increased endocardial pressure, muscular exertion. Degenerative changes in the vascular system are largely due to the same influence, and are causes of dilatation. Hence the disease is more frequent in men than in women. *Occupation* has a similar influence: all those occupations which involve considerable effort tend to cause dilatation of the heart.

(B.) *Predisposing Causes.*—Conditions of weakness of the cardiac walls may consist in acute or chronic changes in the muscular fibres, or in destruction of those fibres and their replacement by tissue elements which yield more readily to the pressure of the blood. Morbid states of the muscular fibres are, (1) Atony, in which the relaxation of the fibres at rest is more absolute, their contraction less complete. (2) The granular degeneration of acute disease. (3) Fatty degeneration, resulting ultimately in the actual destruction of fibres. (4) Fatty overgrowth, in which the muscular fibres undergo secondary atrophy. (5) Fibroid degeneration, the sequel to an acute inflammatory change or the result of a chronic perversion of nutrition. (6) Special degenerations and growths. (7) Weakening of the fibres due to the state of dilatation. Beau pointed out that the fibres common to the two ventricles may be so weakened by dilatation of one, as to lessen considerably the contractile force of the other ventricle, and so to aid its dilatation. (8) Lastly, it has been stated by Niemeyer[1] that the muscular fibres may so lose their contractile power as to permit dilatation when no structural change in the cardiac wall, or in the fibres themselves, can be detected by the microscope. Seitz[2] has lately advocated the same view. In all the recorded examples, however, over-exertion has been the exciting cause of the dilatation, and the cases appear to have been characterized rather by insufficient power to react against the augmented pressure, than by any primary degeneration.

The conditions by which these pathological process are produced constitute the predisposing causes of dilatation. The most important of these conditions are: (1) Anæmia and chlorosis, in which the general mal-nutrition results in atony, and, it may be, granular degeneration of muscular fibre throughout the body. (2) Acute febrile diseases, especially rheumatism, erysipelas, pyæmia, typhus, typhoid fever, &c., having a similar effect. (3) Inflammation, primary or secondary to endo- or peri-carditis, the inflammation in the latter case invading the adjacent layer of the heart. (4) Obesity, with local overgrowth of fatty tissue. (5) Chronic degenerative changes in the system, as yet ill-defined, but often due to chronic alcoholism, and causing fatty and fibroid degeneration of various organs, including the heart. (6) Derangements of the blood-supply to the walls of the heart. Chronic and intermitting passive congestions cause, as Sir William Jenner points out,[1] degeneration of the heart, toughening its walls and lessening its contractile power. Diminished blood-supply is a common cause of fatty and granular degeneration. It may be due to imperfect distension of the coronary arteries in consequence of the defective distension of the aorta, or it may result from narrowing of those vessels by the contraction of lymph outside the heart, or by degeneration, atheromatous and calcareous, of their walls. (7) Defective nerve-power probably in some cases leads to inefficient contraction and dilatation of the heart. Dr. Dobell believes that sexual excesses are powerful causes of cardiac weakness.

Traction from without, the result of pericardial adhesions, is sometimes a cause of dilatation of the heart. The two conditions are constantly found associated, but in the majority of cases there exists also endocardial mischief sufficient to account for the dilatation. Hence, Morgagni and many subsequent writers doubted whether the state of the walls was not always the consequence of the coexisting valvular disease. But cases are not infrequent in which dilatation exists, and no morbid condition can be found to explain its occurrence except an adherent pericardium. Beau,[2] arguing from a small number of such cases, inferred that dilatation was the invariable result of pericardial adhesion. The same view was very strongly maintained by Hope.[3] Wider observation showed, however, that adhesions were frequent enough with no morbid state of the heart's walls. Laennec, Bouillaud,[4] Barlow, Stokes, and others maintained, therefore, that pericardial adhesions have no direct effect in causing dilatation. The same view has been still more recently maintained by Hayden.[5] Gairdner,[6] however, emphasized the fact that in a minor-

[1] Text-book of Practical Medicine, American Trans. vol. ii. p. 320.

[2] Zur Lehre von der Ueberanstrengung des Herzens. Deutsches Archiv für klin. Med. 1873, xi., xii.

[1] On Congestion of the Heart, Med.-Chir. Trans. vol. xliii.

[2] Arch. Gén. de Méd. ser. ii. tome x. 1837, p. 425.

[3] Diseases of the Heart, p. 192.

[4] Traité Clinique, &c., 1835, p. 454.

[5] Diseases of the Heart and Aorta, p. 363.

[6] Edin. Med. Journal, February, 1851.

ity of cases no other cause can be discovered for the changes in the walls of the heart. At the same time he showed that, in other cases, the adhesions not only do not tend to cause dilatation, but they do not prevent the reduction in size which accompanies chronic wasting diseases. The most extensive statistical evidence on the question is that furnished by Kennedy,[1] of Dublin, who collected ninety cases of adherent pericardium without valve disease, and found that the heart remained healthy till death in thirty-four, and was enlarged in fifty-one. But some of his cases were from museums, into which hearts of the normal size would be little likely to find their way, and it is probable, therefore, that his proportion of healthy hearts is too small. Dr. Hayden has collected twenty-three cases of adherent pericardium, without valve disease, and found that in seven there was enlargement without any other discoverable cause.[2] Putting together these facts, and those recorded by other authorities, it seems fair to conclude that adherent pericardium causes enlargement of the heart in one-third of the cases.

The difference in the effect of the adhesion is not to be explained by difference in its extent. The most marked hypertrophy and dilatation was due, in one of Gairdner's cases, to a firm adhesion of very limited extent, near the apex of the left ventricle. In other cases in which no influence was exerted, the adhesion was universal. Dr. Wilks[3] has pointed out that, when general adhesion is associated with dilatation, the effect is more marked on the right ventricle than on the left. This is no doubt due to the thinness of the muscular wall of the right ventricle. In estimating the effect of pericardial adhesions it must be remembered how frequently they are associated with another cause of dilatation, the damage to the subjacent portion of the cardiac wall by the extension to it of the pericardial inflammation. For the settlement of the question of their influence more facts are needed which shall embrace not only the state of the heart's walls, and the fact of adhesions, but the extent, firmness, and probable duration of the latter, the extent to which the pericardium is connected with parts around, and the extent to which the muscular fibres of the heart have suffered from the inflammation.

Dr. Gairdner[4] has maintained that when the expansion of the lungs is interfered with by their atrophy, the inspiratory efforts to distend them, which he regards as the great cause of emphysema, may lead to over-distension of the heart. He believes that it is by this mechanism that emphysema is associated with dilatation of the heart, and appeals in support of the theory, to the fact that the dilatation is not confined to the right side, but affects in slighter degree and a little later in time the left side also. This view depends for its probability on the inspiratory theory of emphysema. If, with Sir William Jenner and most modern authorities, emphysema is believed to arise chiefly, not from primary atrophy of the lung, but from its over-distension during expiratory efforts, this explanation of the origin of dilatation of the heart falls to the ground. No dilating influence by traction can result from violent expiratory efforts, and when emphysema is once established the inspiratory effort which can be made is far less than in health. If the dilatation of the right ventricle in these cases is referred, as is generally taught, to obstruction to the flow through the lungs, the simultaneous affection of the left side can be explained in another way.

(C.) *Exciting Causes.*—Increase in the endocardial blood-pressure has been mentioned as the chief exciting cause of dilatation of the heart. Such increased pressure opposes the contraction of the heart, and leads, by a mechanism to be presently described, to its dilatation. It depends on increased resistance to the movement of the blood, the result of an increase in its mass, or an obstruction in the orifices or vessels through which it flows. This increased pressure leads to two results, directly to dilatation, indirectly to hypertrophy. The causes of the increased pressure, which are more fully considered in the article on hypertrophy, are as follows:—

(1) Increase in the mass of blood to be moved, consequent on over-distension of the heart. Thus regurgitation through an orifice causes dilatation of the chamber behind. Thus, too, the dilatation tends to its own increase, a process which is only arrested by the occurrence of hypertrophy.

(2) Resistance to the movement of the blood in consequence of narrowing of the orifice by which it leaves the affected chamber. The influence of this condition in causing dilatation is not great. The obstruction is gradually developed, and unless associated with weakness of the cardiac walls, the latter become hypertrophied to overcome the increased resistance. In aortic obstruction, for instance, dilatation is rare.

(3) Resistance to the movement of the blood through the vascular system is a powerful cause of dilatation, and is most effective when suddenly developed or in-

[1] Edin. Med. Journal.
[2] Loc. cit., table on p. 362.
[3] Guy's Hosp. Rep. vol. xvi. p. 202.
[4] British and Foreign Medico-Chirurg. Rev., July, 1853, p. 212.

termitting, and especially when the condition in which it arises is such as to impair the nutrition of the walls of the heart. Disease of the large vessels, aorta and pulmonary artery, rarely causes dilatation. Obstruction of the smaller vessels is a more effective cause, and especially those forms of obstruction which affect the pulmonary circulation alone, or in conjunction with the systemic vessels.

Long-continued and severe muscular efforts are, as Ferriar[1] pointed out, a powerful cause of dilatation and hypertrophy. The resulting condition of heart depends largely on the existence of the conditions which favor the occurrence of one or the other state. The effect of effort is to obstruct the circulation through both the general and pulmonary system. Its influence on the left ventricle has been described in relation to hypertrophy. Clifford Allbutt has especially pointed out the direct effect on the right ventricle and the influence of undue smallness of lungs on its occurrence. The obstruction to the pulmonary circulation by the pressure of the air on the inner surface of the air cells obstructs the escape of blood from the right side of the heart. The compression of the heart itself interferes with the entrance of blood from the veins, tends to their over-distension, and when the pressure is removed, to the over-distension of the right auricle and ventricle. Thus the intermitting obstruction causes intermitting over-distension of the right side of the heart, and that intermitting congestion of the walls of the heart which leads to the degeneration of its substance and renders dilatation permanent.

It is by a similar mechanism, according to the views generally accepted, and fully stated by Sir William Jenner in the present volume of this work, that emphysema of the lungs causes dilatation of the heart. Intermittent distension results, as just described, from the violent expiratory efforts with closed glottis, which constitute the efficient cause of emphysema; and as the latter condition is developed, degeneration, elongation, and destruction of capillaries render the obstruction permanent, which before was occasional. The right side of the heart undergoes dilatation, sometimes to an extreme degree. Hypertrophy is usually also produced. The congestion of the cardiac wall disposes the left ventricle to yield before the increased pressure of the aortic blood, which is an ultimate effect of the venous distension acting through the capillary system.

Other forms of pulmonary change have a slighter tendency to cause dilatation of the heart than emphysema. An exception must, however, be made for cirrhosis of the lung, which produces, in a large number of cases, hypertrophy and dilatation of the right side. Such a change was present in one-third of the cases of cirrhosis collected by Bastian.[1]

Mechanism.—The consideration of the mechanism by which dilatation is effected is necessarily, in the main, theoretical. It has, perhaps on this account, received little attention, and has even been sometimes dismissed as useless. But any clear conception of the way in which a morbid state is related to its causes, if correct, must afford a clearer view of its pathological significance, and of the way in which by treatment it may best be met.

The dilatation of the heart is produced, in every case, by its over-distension with blood. Just as the various causes of hypertrophy involve, as the efficient cause, overwork, so the various causes of dilatation involve over-distension. The immediate cause of this over-distension is, in each case, the existence at the end of the diastole of an endocardial pressure disproportioned to the resisting power of the wall of the heart, and before which the wall yields. The act of dilatation thus occurs during the diastole of the heart. This circumstance lessens the simplicity of the relative action of the exciting and predisposing causes of dilatation, since, as will be immediately explained, each may act by producing a similar effect.

Three sources of over-distension may thus be recognized. (1) The mass of blood entering in the normal course of the circulation may be abnormally large; *simple over-distension.* (2) Blood may enter the cavity from an abnormal source (regurgitation), and being added to that entering it in the normal course of the circulation, increases the mass of blood and so the distension of the chamber: *over-distension from regurgitation.* (3) The whole of the blood previously in the chamber may not be expelled from it during contraction, the residual blood being added to that entering from behind increases the distension of the chamber; *over-distension from imperfect contraction*, or *residual over-distension.*

(1) *Simple over-distension* is the result of over-distension of the source from which the blood enters the affected chamber. It is well seen in the effect of mitral regurgitation on the left ventricle. The over-distended auricle drives an abnormal quantity of blood into the ventricle, into which probably an increased quantity has already passed in consequence of the heightened tension of the blood within the auricle. It is probable that a large quantity of blood enters the ventricle during diastole, enough to equalize, or almost to equalize, the pressure within

[1] Med. Hist. and Ref., vol. i. 1792.

[1] Art. Cirrhosis of Lung, vol. iii.

the ventricle and within the auricle,[1] before the auricular contraction effects the actual distension or over-distension of the ventricle. The pressure to which the inner suface of the ventricle is exposed at the end of the auricular systole is very great, for in accordance with the well-known law of hydrostatics it is multiplied directly as the area of the inner surface of the ventricle exceeds that of the auriculo-ventricular orifice. Simple over-distension may occur, especially in the auricles, in conditions of acute weakening of the cardiac walls. The lessened tone of the muscular fibres allows them to yield unduly before the pressure of the incoming blood, and as the current is continuous, they thus become directly over-distended. Similarly the flaccid ventricles may yield unduly before the current which enters during diastole, and the systole of the auricles may over-distend the ventricles. This mechanism has been described by Beau[2] as *dilatation sans asystolie*. But the conditions are those under which contraction is imperfect, and the small pulse renders it probable, in many cases, that such imperfection occurs. Residual over-distension will then increase the dilatation.

(2) *Over-distension from regurgitation* is one of the most efficient causes of dilatation. The cavity is filled with blood from a double source. That which enters into the normal course of the circulation is added to that which has regurgitated into the cavity, and over-distension results. In aortic regurgitation, for instance, it is the addition of the contents of the auricle to the blood regurgitating into the ventricle from the aorta, which actually distends the chamber and dilates it until, ultimately, the dilating process is met by compensating hypertrophy. In permanent patency of the semilunar valves the intra-ventricular pressure at the end of the auricular systole must be very great, since the pressure of the aortic blood will be added to that produced by the contraction of the auricle.

(3) *Over-distension from imperfect contraction; residual over-distension.*—Whenever, from any cause, systole is incomplete, blood must remain in the chambers and render the entrance of the normal quantity of blood an over-distending agent. Incompleteness of contraction is theoretically possible from two causes, diminished contractile force, and increased resistance to contraction. It is probable that each of these does actually prevent complete contraction, since each is found to be an efficient cause of dilatation.

(*a*) The various conditions which weaken the cardiac walls, already considered, must tend to render the heart incapable of overcoming all the resistance that is opposed to it, whether that be normal or increased. Hence the contraction is imperfect, and the residual blood is the ultimate cause of over-distension. To this condition Beau gave the name of *asystolie*. This weakening of the wall not only leads to over-distension, it also renders the effect of the over-distending force greater in degree and in duration, for the weak wall yields more to the increased pressure, and the yielding of the degenerated wall is permanent. Among the conditions weakening the heart must also be reckoned the state of dilatation. The dilated heart has increased work, for it has to move an increased mass of blood, to overcome a greater pressure. To this it is even less competent than a healthy heart. Hence the dilatation itself renders the contraction additionally incomplete, and is thus perpetuated and increased. The influence of dilatation is of course here considered apart from that of the hypertrophy commonly conjoined with it, and to some extent counteracting its effect.

It is by interfering with contraction that pericardial adhesions must be considered to exert whatever influence they possess in causing dilatation of the heart. Connections of the pericardium with parts around, consequent on the extension of inflammation to its outer surface, may cause the adhesions to the heart to oppose considerably the reduction in size during systole, and thus to render the contractions incomplete.[1] Moreover, a similar effect may be produced by the interference with the approximation of different parts of the surface during contraction, which must occur if a thick inelastic membrane covers the heart. Such an influence will interfere chiefly with the contraction of the thin-walled right ventricle, and this may be one reason why it suffers most.

[1] That this is the case is highly probable, from a phenomenon sometimes to be observed in cases of mitral constriction. When diastolic and presystolic murmurs are both present, the former due to the slow passage of blood through the orifice in consequence of the tension of the blood within the auricle, the latter due to the contraction of the auricle, there may be, during an occasional prolonged diastole, an interval of silence between the two murmurs. When the diastolic murmur is loud, this silence can only be explained by a cessation, or almost cessation, of the flow of blood, which means, of course, an equalization of the pressure in the two cavities.

[2] Beau, Traité d'Auscultation. Paris, 1856.

[1] Thus in Gairdner's case, already mentioned, in which marked hypertrophy and dilatation of the left ventricle were associated with, as the only discoverable cause, a local adhesion near the apex, a corresponding adhesion connected the other side of the pericardium with the left lung.

The effect will be to cause a residual over-distension, just as does the simple weakening of the cardiac wall with which the adhesions are so often associated.

(*b*) Increased resistance from some obstruction to the circulation is another cause of incomplete contraction. Such increased resistance may interfere with the contraction of a healthy heart, but probably rarely does this unless great and suddenly developed. The reserve of power usually prevents imperfect contraction, and compensating hypertrophy gradually renders the heart efficient. But when suddenly developed, or when the nutrition of the heart is interfered with, the chamber dilates. This dilatation was formerly, and is still by some, ascribed to the direct effect of the increased pressure on the contracting fibres.[1] It was compared by Senac to the effect of an extending force in elongating a cord.[2] Niemeyer[3] pointed out that such an explanation is entirely inapplicable to the conditions of the phenomenon. Increase in the capacity of a contracting chamber from increased pressure within it during contraction, is inconceivable. Such increased capacity can only be explained by an increased quantity of blood entering it under a pressure sufficient to overcome the resistance of its walls. The influence of increased resistance to contraction may be to weaken the muscular fibres, to lessen the elasticity of the walls, and to render over-distension easier, but more than this it cannot directly effect.

(D.) *Determining Causes.*—The exciting causes of dilatation and hypertrophy are thus to some extent the same; the occurrence of the result is influenced not only by the predisposition already described, but also by certain determining conditions.

(1) The rapidity of the development of the increased blood-pressure, *i. e.*, the rapidity with which the valvular disease, or the systemic or pulmonary obstruction is produced. Time is necessary for the production of that hypertrophy which alone can prevent dilatation, and a suddenly-developed obstruction invariably leads to dilatation.

(2) The small amount of muscular tissue normally existing in the wall of the affected chamber of the heart. This is naturally proportioned to the work of each segment of the heart, *i. e.*, to the blood-pressure, to be by it passively resisted and actively overcome. The extra pressure induced by the abnormal obstruction or regurgitation bears no necessary proportion to the normal blood-pressure, and before absolute equal increments of pressure, the smaller the normal amount of muscular tissue, the more readily does dilatation occur, because the systole is the more readily rendered imperfect, and residual over-distension produced.

Each cavity of the heart affords an illustration of these influences, and although our knowledge is still very imperfect, we can understand something of the origin of the condition found in each instance, and it is worth while to recapitulate briefly the way in which the different results are brought about.

In aortic obstruction, the left ventricle is commonly hypertrophied, less commonly dilated. The left ventricle, containing the greatest amount of muscular tissue, possesses a large reserve of force, and can overact so as to overcome a moderate increase in resistance, and so prevent residual over-distension and dilatation. The development of obstruction is usually slow, and thus there is time for hypertrophy to occur. In aortic regurgitation there is always dilatation, and usually much, often very much, hypertrophy. The regurgitant blood causes the ventricle to be overfilled, and the patent aortic orifice transmits to the interior of the ventricle, during its passive state, the intra-aortic pressure. The regurgitation is usually slowly developed, and the muscular tissue of the ventricle considerable, and hence hypertrophy occurs. This is favored by the abundant blood supply to the heart, consequent on the great distension of the aorta.

In mitral disease, obstructive and regurgitant, the left auricle undergoes dilatation and hypertrophy, the former predominating in regurgitation, from the direct over-distension and frequently rapid development of the pathological state. Hypertrophy of the auricle is usually more frequent in obstruction from the slowness with which the lesion is developed. Dilatation is always, however, conjoined, from the ease with which the contraction of the weak auricle is rendered imperfect by obstruction. In mitral regurgitation, the left ventricle is hypertrophied and dilated, and the dilatation is usually considerable, in consequence of the direct over-distension of the chamber, and perhaps also of the

[1] Lately by Chirone in Lo Sperimentale, August, 1874.

[2] "La contraction qui resserre les ventricules est peut-être l'instrument qui augment les dimensions, que le sang soit en trop grande quantité dans ces réservoirs; qu'il trouve quelque barrière que l'empêche d'en sortir avec la liberté qu'il a ordinairement, l'action des fibres sera plus forte: or cet excès de force doit nécessairement les allonger: un raccourcissement forcé produit le même effet qu'une action qui tire et qui tend une corde, ses éléments doivent nécessairement s'ecarter, et mê se séparer, s'ils sont tirés avec trop de violence."—Senac, Traité, &c., 1749, tom. ii. p. 397.

[3] Loc. cit. vol. ii. p. 316.

imperfect distension of the coronary arteries and consequent damaged cardiac nutrition.

The right ventricle, in disease of the left side, usually undergoes dilatation, from its small amount of muscular tissue, but often is also hypertrophied, in consequence of the slowness with which the obstruction in the left side tells back upon the right. The hypertrophy is usually less and the dilatation as much marked, when the obstruction is situated in the pulmonary system, in consequence of the directness with which such obstruction affects the ventricle, the rapidity with which it is frequently developed and increased, and the damage to the cardiac nutrition, which results from the extreme and sudden passive congestion to which the heart is, in these cases, very often liable.

In obstructions to the systemic circulation, hypertrophy is the common change in the left ventricle, and often, especially in Bright's disease, is wholly unattended with dilatation. The extreme slowness with which the obstruction is developed is, no doubt, a chief factor in determining the occurrence of hypertrophy rather than dilatation. Occasionally, however, dilatation occurs instead of hypertrophy. Such cases are perhaps instances of simultaneous cardiac and vascular degeneration, in which the increased blood tension is the result of the latter, and the damaged heart is incapable of resisting the abnormal pressure.

The sequence of the conditions of hypertrophy and dilatation varies under different circumstances. It is certainly not uniform, as has been maintained by some writers. When an increased resistance or a cause of over-distension is suddenly developed, dilatation results at once, and hypertrophy slowly, when time allows overgrowth to occur. This is frequently seen in aortic and mitral regurgitation. The order is the same when the initial state is one of defective power in the walls of the heart; dilatation precedes and is the cause of hypertrophy—as in that which results from carditis. On the other hand, dilatation may be secondary. Degeneration occurs in the hypertrophied tissue more readily than in the healthy heart. Nutritive influences fail from impaired health or advancing years.[1] Again, the coronary vessels suffer from undue strain, degenerate, and lessen the blood supply. This, as pointed out by Mauriac, is a frequent occurrence in aortic regurgitation. Under all these conditions the degeneration weakens the cardiac wall, and dilatation occurs at a later period than the hypertrophy.

PATHOLOGICAL ANATOMY. — Dilatation may affect all the chambers of the heart or only some of them. It has been a subject of rather unprofitable discussion whether general or partial dilatation is the more common. It is rare for one chamber to suffer considerably alone. When the cause of the dilatation is disease of an orifice, the chambers behind the orifice are usually alone affected. An exception is mitral regurgitation, in which the cavity in front of the orifice is dilated also. The chamber immediately behind the diseased orifice commonly suffers more than the others. In mitral constriction, for instance, the left auricle is most dilated. In all diseases of the left side of the heart, the right side may ultimately become dilated. Hence the most widely distributed change occurs when the obstruction is in front of the left ventricle, and affects each part of the heart successively. In aortic regurgitation, for instance, enormous hearts are met with, in which every cavity is dilated. Occasionally a similar result follows obstruction in the aortic system.

The dilatation, as already stated, is rarely simple. Hypertrophy is usually present, and varies in amount according to the conditions described in the last article. From the variations in the amount of dilatation and associated hypertrophy very different effects on the form and size of the heart are produced.

The amount of dilatation is estimated by comparison with the normal capacity, by measurement of the external size of the heart, the thickness of the walls, and the length and mid-circumference of the cavity. In estimating it, regard must be had to the age of the patient, and to the state of the body. The capacity of the heart naturally increases with age. In decomposition the relaxation of the heart is extreme, the cavities present their maximum capacity, the walls their minimum thickness. The existence of decomposition, which in some cases is very rapid, must therefore induce caution in inferring actual dilatation from a flaccid and apparently dilated state of the heart.

A heart, the subject of general or partial dilatation, is increased in size and altered in shape. The increase in size may be considerable; the circumference being two, three, or four times the normal. Occasionally the left ventricle is so large as to be "capable of containing another heart"—a favorite comparison since the time of Malpighi. The left auricle may be dilated, in disease of the mitral orifice, to very large dimensions. In a case recorded by Cruveilhier, it had four times its normal dimensions. The greatest di-

[1] Niemeyer pointed out how frequently from this cause the hypertrophy which results from senile vascular degeneration gives place to dilatation.

latation, however, occurs on the right side. Both ventricle and auricle may be very large. The right auricle, as Burserius[1] remarked, may undergo greater dilatation than any other part of the heart. Stokes[2] mentions a case in which the auricle was so capacious as to contain a pound of blood.

The shape is altered according to the part of the heart affected. In general dilatation the heart is increased in width, so that it has a more globular shape. This depends especially on the dilatation of the right chambers, and is marked when these alone are affected. Considerable dilatation of the auricles may alter considerably the normal shape of the heart. Thus in the case mentioned by Stokes, the dilated right auricle "formed a vast purple tumor, which concealed the whole of the anterior portion of the right lung."

In pure dilatation the weight of the heart is normal. Instances of this are, however, to say the least, very rare. As a rule the weight of the dilated heart is greater than normal, in consequence of the almost invariable coexistence of hypertrophy.

The walls of the heart the subject of simple, or nearly simple, dilatation are flaccid, and collapse when cut across. They are thinner than normal in proportion to the amount of dilatation, and to its freedom from accompanying hypertrophy. In most cases the attenuation, however considerable, is the result of the extension of the wall. In rare cases the wall may actually be atrophied. In the ventricles the thinning is most marked towards the apex. The wall of the left ventricle may be reduced to one-sixth of an inch at the middle and one-twenty-fifth of an inch at the apex. The walls of the auricles may, in extreme dilatation, be reduced to an almost membranous condition. Very frequently, coexisting hypertrophy prevents noticeable diminution in the thickness of the walls, even when the dilatation is very great. The thickness of the wall may even be above the normal, notwithstanding the dilatation, especially when the latter is moderate in degree.

The muscular tissue is sometimes normal in appearance, sometimes pale or mottled. Under the microscope it usually presents evidence of degeneration, especially when the dilatation is comparatively pure. The muscular fibres present indistinct striation, or granular or actual fatty degeneration. The connective tissue between the fibres is often increased, and may also present granular degeneration. The endocardium may be thicker or thinner than normal; it is often irregularly thickened and opaque, especially in the auricles. The pericardium is stretched in proportion to the dilatation, and is also often unduly opaque.

The orifices participate in the dilatation of the cavities of the heart. The auriculo-ventricular orifices undergo the greatest extension, especially when the cavities on each side of them are dilated. The ultimate result is that the valves become incompetent to close the orifice, in consequence of the disproportion between their area and that of the enlarged orifice. This effect is increased by the removal of the bases of the papillary muscles to a greater distance from the orifice, in consequence of the extension of the wall. For a time the incompetence may be averted. The segments of the valves may undergo some amount of dilatation so as to close the enlarged orifice, and the papillary muscles may undergo at their apices transformation into fibrous tissue, which, being incapable of contraction during the systole, effects a practical elongation of the muscle, and so helps to counteract the effect of the removal of their points of attachment. Ultimately, however, the dilatation of the orifice exceeds the influence of these compensations, and incompetence of the valves results. This is the case especially in the right side of the heart, in which the dilatation of the two cavities is usually simultaneous and considerable, and is the common cause of tricuspid incompetence.[1]

In dilatation of the auricles, the large venous trunks opening into them, unprotected by valves, commonly participate in the dilatation, and may be greatly enlarged, so that their openings into the auricle may be hard to determine. The auricular appendices are also much dilated.

Certain associated conditions are commonly met with in cases of dilatation. Some of these are causal, such as valvular disease, pericardial adhesions, emphysema of the lungs, kidney disease. Others are sequential, such as passive congestion of organs, and its consequences in alteration in their texture.

CONSEQUENCES.—From the incompetence of the valves due to the dilatation of the orifices, regurgitation of blood with all its consequences, results. Before, however, sequential regurgitation is developed, the same consequences, although in less degree, may result from the diminished power of propelling the blood. The

[1] The Institutes of the Practice of Medicine, 1798. Cullen Brown's Trans, vol. v. p. 312.

[2] Diseases of Heart and Aorta, p. 275.

[1] This was first insisted on by Forget, Gazette Médicale de Paris, 1844, p. 657. The dilatation of the orifice was pointed out by Corvisart, loc. cit. p. 154.

resistance of a larger quantity of blood has to be overcome, and the power of moving it is absolutely diminished by the dilatation. Hence, unless compensatory hypertrophy assist, less blood leaves the dilated chambers at each systole. The amount of residual blood may be so large that the quantity which can enter in the normal course of the circulation is less than in health. Hence, as Morgagni pointed out,[1] the dilatation acts as an obstruction to the onward movement of the blood, the vessels behind (venous system) become overfilled, the vessels in front (arterial system) underfilled.

The effect of dilatation of a cavity may thus come to be the same as that of obstruction at the orifices of the heart by which the blood should enter the chamber. If the chamber affected be a ventricle, the first effect is the over-distension of the corresponding auricle, and its consequent dilatation and perhaps hypertrophy. The veins by which the blood enters the auricles are over-distended, and when valvular incompetence is added, the pulmonary and larger systemic veins may be enormously dilated. I have known the right internal jugular to be so large, in dilatation of the right side of the heart, as to be mistaken for an aneurismal dilatation of the common carotid artery. Pulsation may be communicated to the veins as a result of the valvular incompetence (see article on Diseases of the Valves). The venous congestion affects alike the general tissues, causing various dropsies into the cellular tissue and serous cavities, and the organs, especially the lungs, brain, liver, portal system, and kidneys. Lastly, the other side of the heart may be overloaded and dilated, and ultimately even the side of the heart first affected, by the transmission of the influence through both systems of circulation. The last effect, which occurs only when the primary disease is at the mitral orifice, is perhaps due to the secondary dilatation of the right side. The effect of this venous congestion is to overload the venous radicles of the organs with blood, and cause their permanent dilatation. The proper tissue-elements of the organs undergo atrophy, or it may be granular, or fatty degeneration, partly in consequence of the pressure upon them of the distended veins, partly from the imperfect supply of arterial blood. Lastly, the connective tissue of the organs overgrows, and their consistence is thereby increased. The effect of these changes is somewhat modified by the characters of the organ affected.

The heart itself may suffer from the mechanical congestion of its walls, the consequences of which have been already pointed out. The mechanical congestion, however, it is believed, affects the heart later and less than the other organs, in consequence of the obliquity of the opening of the cardiac veins which produces a valve-like effect.

The lungs are overloaded with blood, and serosity exudes from their walls into the air-cells and minute bronchi, and probably blood corpuscles migrate into the parenchyma. Ultimately the capillaries becomes varicose,[1] the blood-pigment collects in the cellular elements of the lung, giving it a brown color, and the connective tissue is increased in quantity,[2] augmenting considerably the consistence and to a slighter extent the size of the lung, and producing ultimately the condition of "brown induration."

The brain undergoes slighter changes, no doubt in consequence of the effect of gravitation in opposing the movement of the blood. Its venules are enlarged and the distension of the surface veins may be very great. The pressure of the distended vessels in the interior may lead to their rupture into the perivascular sheaths, or to atrophy of the adjacent brain substance. The consistence of the brain is often lessened. Induration does not result. Corvisart maintained that rupture of large vessels and cerebral hemorrhage might result from venous congestion, but his opinion has not received much confirmation.

The liver is congested, in a very high degree, from the directness with which the hepatic vein suffers from increased distension of the inferior vena cava. The organ becomes uniformly enlarged, first and mainly from the distension of the radicles of the hepatic vein, and afterwards by fatty degeneration of the liver tissue, or by fibroid overgrowth around the vessels and between the lobules, by which the organ may become indurated. On section, the distended venules are very conspicuous, and their enlargement is such that the hepatic tissue is compressed between them, and the appear-

[1] Morgagni, speaking of a case of aortic regurgitation, says: "Some portion (of the blood) returned into the left ventricle of the heart when the ventricle ought to receive the blood that was coming in from the lungs, it would necessarily happen that the returning portion, as well as the portion which had not been extruded just before, must occupy some part of that space which, from the design of nature, was entirely due to the blood that was coming in from the lungs, which circumstance finally could not but overload the lungs and heart." De Sedibus et Causis Morborum, 1779, letter 23, art. 12. As translated by Cockle, loc. cit.

[1] Buhl, quoted by Wilson Fox, vol. iii. art. Brown Induration of the Lung, p. 801.

[2] Rokitansky, Wilson Fox, loc. cit.

ance is produced of lobules lying between the distended venules, and thus a portal congestion is simulated. The liver tissue is frequently pale from fatty or fibroid degeneration, and, contrasting with the dark vessels, the so-called "nutmeg liver" is produced. Ultimately the liver may undergo reduction in size from atrophy of the proper elements and contraction of the fibrous tissue (Murchison).[1]

The flow through the liver capillaries is necessarily impeded, and thus the obstruction is transmitted to the portal system. The spleen is enlarged, and, like the liver, may be the seat of fibroid overgrowth, causing its induration. The peritoneal and intestinal vessels are distended, and fluid may be effused into the peritoneal cavity. The fibroid overgrowth in the liver may ultimately lead to compression of the portal venules, and consequent portal congestion, out of proportion to the congestion which results simply from the cardiac state.

The kidneys suffer similar congestion, and present the appearance which was produced artificially by ligature of the hepatic vein, by Robinson.[2] They are enlarged, smooth, and dark in color from the venous distension. The cortical and pyramidal portions preserve their relative proportions. At first their consistence may be lessened, and the capsule separate readily; after a time fibroid overgrowth occurs and the kidneys become indurated. Ultimately this tissue may contract, the organs becoming smaller and harder, their surface slightly granular, and the capsule unduly adherent.

The veins of the body generally are also over-distended. Serum escapes from them into the connective tissue and accumulates in the more depending parts. Usually the condition comes on gradually, and the œdema commences in the legs. It is first noticed in the evening, and disappears during the night, when the legs are raised; but it continues increasing, until, although lessened, it is not removed by the horizontal posture. If the patient be in bed it may be first noticed in the lower part of the back. It may increase until the distension of the legs is extreme, and the skin, if not relieved, may slough. Lastly, coagulation may occur in the distended veins, but this accident is not common. The amount of congestion varies from time to time in dependence upon accidental causes of increased obstruction, due sometimes to variable cardiac strength, more frequently to variations in the cause of the dilatation in the lungs, &c. Again, the manifestations of venous congestion are not uniform in different cases. An accidental cause, a local inflammation, may determine a large effusion of serum in some special position, as the pleural or peritoneal cavity. Some accidental obstruction may lead to local œdema. A special predisposition to disease in some one organ, as the liver or the kidney, may cause that organ to suffer in undue degree and give a special character to the symptoms. Moreover a vicarious action is often observable between the vessels of the organs and of the limbs and cellular tissue. The extreme affections of organs, the very large livers, the extreme albuminuria, are often seen where the general œdema is slight; whereas when the anasarca is extreme there may be even to the last only a trace of albumen in the urine, and the enlargement of the liver may be trifling. Fibroid overgrowth in organs may hinder the distension of their vessels, and so throw an additional strain upon those of the general system.

The over-distension of the venous system, on which so many of the symptoms depend, can only be in part ascribed to the dilatation of the heart. It is in large part due to the cause of the dilatation. Dilatation of the right ventricle permits the obstruction in the lungs, which exists in emphysema, to tell back upon the venous system. But it also adds to the obstruction. When due to no increased resistance, but to muscular degeneration, it will give rise to similar symptoms. So in the latter case, degeneration of the cardiac wall, the weakness in its contractile power which permits dilatation, is itself, as Niemeyer pointed out, a cause of the impaired circulation. The resulting dilatation, by its mechanical influence, intensifies what may be called the potential obstruction which results.

Symptoms.—The existence of dilatation is declared by certain symptoms and physical signs. Some difficulty in their determination has arisen from the circumstance that pure dilatation is so rarely met with; dilatation is usually accompanied by hypertrophy. But pure hypertrophy is not uncommon, and by comparison of these cases with those in which dilatation coexists, and especially with those in which dilatation predominates, the symptoms of the latter condition have been ascertained. They are most marked and characteristic in general dilatation.

The *Physical Signs* depend on the increased size and lessened strength of the heart. The area of dulness, both deep and superficial, is increased. The deep dulness may extend from the anterior axillary line, to two fingers' breadth to the right of the sternum, even in rare cases as far as the right nipple; upwards it may reach to the first rib, and down-

[1] Clinical Lectures on Diseases of the Liver, 1868, p. 120.

[2] Med.-Chir. Trans. 1843, p. 51.

wards to the seventh rib. It inclines to squareness of outline, in consequence of the lateral increase in the size of the heart. The greater the dilatation, the greater is the lateral increase in the dulness. The impulse is perceptible over an abnormally large area. It may be felt from the epigastrium to the axilla. It is also diffused. A maximum apex-beat may or may not be perceptible. It is always less distinct than in health. When it cannot be felt it may sometimes be seen (Walshe). The impulse is weak and sudden in proportion to the amount of dilatation and to its purity, *i. e.*, its freedom from associated hypertrophy. It may be somewhat undulatory in character, in consequence of different parts of the heart striking the chest wall successively, not simultaneously. Successive beats may be unequal in strength, and may also strike the chest-wall at different points. Bulging of the chest-wall is slight in dilatation, and is said to be always absent when there is no hypertrophy; now and then in a large dilated and slightly hypertrophied heart it is very distinct. Displacement of organs occurs in the hypertrophied form, the lungs are pushed out of the way, the liver may be displaced downwards, so that its rounded upper surface is visible beneath the ribs.

The sounds of the heart are weakened, the first sound is shortened and its tone raised. As Flint[1] puts it, the valvular element in the sound predominates. When there is coexisting hypertrophy, the first sound may be clear and ringing, but the sound becomes weaker in proportion to the amount of dilatation. The shortening may cause the first sound to resemble in its characters the second sound, so that, as Stokes[2] pointed out, it may not be easy to distinguish between them. Laennec taught that clearness of the first sound is a sign of dilatation. Stokes and Gairdner[3] showed that this clearness exists only when hypertrophy is combined with the dilatation. Reduplication has been noticed in some cases, and may be due to the asynchronous contraction of the two ventricles.

In dilatation of the ventricle, especially of the left ventricle, a systolic apex murmur is frequently heard. In a large number of cases it depends on incompetence of the auriculo-ventricular valves, primary (in the case of the mitral valve), or due to the extension of the orifice in the dilatation of the heart. In many cases, however, no incompetence can be discovered after death, although a systolic apex murmur was heard during life. But the post-mortem tests for incompetence of the mitral valve are not very satisfactory. Slight inefficiency may remain undetected, and on the other hand, slight regurgitation cannot be accepted as conclusive evidence of functional incompetence. In each case the action of the papillary muscles during life may vitiate the post-mortem conclusion. Hence some authorities believe that such a murmur, when heard in dilatation of the ventricle, is always due to auriculo-ventricular regurgitation. Others, among whom are Stokes[1] and Walshe, believe that a murmur is occasionally to be heard in cases in which the post-mortem evidence of valvular competence is so conclusive that regurgitation is a very improbable explanation. They consider that the contraction of the ventricle alone may throw the blood into audible vibrations. The conditions are certainly such as to render the result conceivable. It is probable that the systole of a dilated ventricle is never complete. A considerable amount of blood remains in its cavity. The spaces between the various projections into the cavity,—the trabeculæ, papillary muscles, the cuspid valves,—are larger than in health, and remain unobliterated at the end of the ventricular contraction, and the eddies into which the blood is thrown must be considerable. Moreover, the irregularity of the blood current is no doubt sometimes increased by irregularity in the contraction of the ventricles. By these means it seems probable that a murmur may be produced within the ventricle, the consequence and the sign of dilatation only.

The *pulse* is weak in proportion to the amount and purity of the dilatation. It is sometimes of moderate size, sometimes small; its size is largely influenced by the condition of heart to which the dilatation is secondary. It is often quick, and is unduly quickened by exertion. Sometimes it is infrequent, either because the heart's action is infrequent, or because the irregularity in force is so great that every systole does not influence the pulse. Thus the effect of intermission is produced. Actual intermissions may also occur.

Dilatation of the *left ventricle* alone, is attended by the changes in the impulse which have been already described as among the most conspicuous signs of general dilatation. The impulse is diffused, and both impulse and dulness are extended to the left. The first sound is weak; the pulse presents the characters just described. Sooner or later the mitral orifice is stretched to incompetence of the valves; then general dilatation, with all its symptoms, quickly follows.

Dilatation of the *left auricle* may lessen the resonance at the inner end of the

[1] On Diseases of the Heart, second edition, p. 86.

[2] Op. cit. p. 260.

[3] Edinburgh Medical Journal, July, 1856, p. 56.

[1] Diseases of the Heart and Aorta, p. 261.

second left interspace, and a feeble presystolic impulse may be perceptible there. Pressure on the left bronchus may interfere with the expansion of the left lung (Barlow).

Dilatation of the *right ventricle* causes pulsation to be transmitted to the epigastrium, and extension of dulness to the right of the sternum in the fifth and sixth interspaces; the apex of the heart is in the normal position. Jugular fulness is common, and pulsation consequent on tricuspid incompetence is not rare, and, as tricuspid incompetence is rarely due to any other cause, it affords additional evidence of the existence of dilatation of the right ventricle. The pulse may, as Lancisi pointed out, be little changed.

Dilatation of the *right auricle* causes dulness to the right of the sternum, where pulsation may sometimes be detected, generally presystolic, rarely systolic in consequence of the tricuspid insufficiency (as in a case of Dr. Stokes,[1] in which an aortic aneurism was simulated). Jugular pulsation, systolic in rhythm, occurs, and may be in rare cases diastolic also.

Symptoms.—Dilatation of the heart affects, secondarily, almost every organ in the body, and its symptoms, direct and indirect are very numerous. They vary widely, however, in distribution and degree, in different cases.

Cardiac discomfort is frequently present; it varies from mere uneasiness to acute pain, constant or paroxysmal (pseudo-angina). Palpitation is very common. The sudden contraction of the enlarged heart is perceived unduly by the patient, especially when irregularity in force or rhythm is superadded. The heart is easily excited to frequent contraction by slight causes—muscular exertion, emotional excitement, or mechanical disturbance, as by a distended stomach.

The general strength is always lessened. The patient complains of lassitude and languor and faints easily.

All parts of the general system present evidence of passive congestion. The venous stasis is seen in the distended superficial veins and the cyanotic tint. Subcutaneous œdema is often present and may be considerable. Its occurrence is influenced, not only by the cardiac obstruction, but by the state of the blood. In anæmic persons the normal blood-pressure may suffice to cause slight œdema of the feet, and a similar state of blood assists very much the effect of the increased venous pressure in cardiac dilatation. The local dropsies, effusions into the pleural, pericardial, or peritoneal cavities are attended by their special symptoms. Their occurrence may alter the character, and add much to the gravity of the symptoms present in a given case.

Special symptoms result also from the congestion of organs. The congestion of the lungs is indicated by cough, dyspnœa, cyanosis. Cough is often a very troublesome symptom. It may be paroxysmal and independent of any bronchial secretion, or a small amount of mucus may excite an excessive cough. Secretion is often, however, abundant enough from the congested vessels, and the sputa may be abundant, watery, or mucous, often stained with blood. The congested bronchi are liable to inflammation, by which all the symptoms are increased.

Dyspnœa is a very constant symptom, due chiefly to the imperfect pulmonary circulation and deficient aëration of the blood. At first it is slight, and is felt only when exertion increases the need for oxygen: especially on ascending a hill or stairs. Later on it may be constant, and be increased when the body is recumbent (probably because the descent of the diaphragm is impeded by the weight of the abdominal viscera). Respiration may be quickened to thirty or forty acts per minute, and is panting in character, with noisy expiration. It varies in intensity, sometimes in correspondence with cardiac failure, sometimes without apparent cause. The patient, never free from a sense of want of breath, may from time to time start up in an agony of dyspnœa, undo the clothes upon his chest, and grasp convulsively at any object within his reach. Often even the reclining posture with the head backwards cannot be borne, and the sufferer can only rest or sleep sitting up with his forehead supported. Sometimes a rhythmical character may be observed in the dyspnœa, analogous to, though not identical with, the Cheyne-Stokes breathing. Brief attacks of panting dyspnœa commence suddenly, and gradually subside to comparative, perhaps dozing, calm, with which they alternate. These spasmodic forms of dyspnœa may be singularly out of proportion to the interference with the aëration of the blood, as estimated by the amount of cyanosis.

The congestion of the brain causes frequent headaches. Vertigo is common. The patient sleeps and dreams much. He dozes during the day, and at night is disturbed by restless starts. Corvisart pointed out that the passive congestion sometimes causes a "sub-apoplectic" state during the last hours of life. Delirium is not uncommon, and may be violent; a state of approaching chronic mania sometimes results.

The congestion of the liver is indicated by an icteric tint of skin, by pain and weight in the right back, right shoulder,

[1] Diseases of the Heart and Aorta, p. 275.

or hepatic region, and by abdominal discomfort due to the increased size of the organ. Frequently the enlargement can be both seen and felt. Pulsation may be felt in it, either communicated to it directly by the heart, or, it is said, transmitted through the venous system. The liver is very constantly depressed as well as enlarged. More urgent symptoms result from the transmitted obstruction in the portal system. The functions of the stomach and intestine are interfered with by the mechanical congestion of their walls. Vomiting is a common, and often a most troublesome, symptom. It is probably due to the mechanical congestion of, and direct pressure upon, the stomach. Possibly, in some cases, it may, as Walshe suggests, be the reflex result of an irritation of the pneumogastric nerve. It sometimes results from a catarrhal condition, which is easily excited in the congested organ. The distended vessels may give way, and hæmatemesis result. Piles are common. The hemorrhage from them may relieve the congestion and prevent other symptoms. In other cases, from the mechanically congested vessels, serum escapes into the intestinal canal, or the peritoneal cavity, causing diarrhœa or ascites. In the former the stools are copious and watery, and give little pain. Such diarrhœa may constitute the earliest symptoms of cardiac mischief. All these symptoms of portal congestion may, in the later stages, be intensified by an increase in the obstruction due to secondary changes in the liver itself.

The mechanical congestion of the kidneys produces changes in the urine, which becomes scanty, dense, high-colored, often loaded with lithates, and may contain albumen. The quantity of albumen varies, and does not always correspond, as might be expected, with the amount of venous congestion. Roberts[1] suggests that it depends on the pressure to which the arteries are exposed in the congested state, and he points out that it is often greater, the stronger the force with which the heart acts. Tube-casts are frequently present in the urine, and are generally hyaline or slightly granular, and of medium size.

The ultimate effect of general dilatation is to act through the venous and capillary system on the arteries and the left ventricle, increasing the tension of the pulse and the effect on the left side of the heart. The variation in the amount of obstruction at different times produces great alterations in the organic symptoms. As Stokes pointed out, attacks of dyspnœa due to cold, &c., may be accompanied with a rapid increase in the size of the liver, which will descend in a short time far into the abdomen, partly from the enlargement, partly from displacement, and on the subsidence of the attack will return to its ordinary volume. The albumen in the urine may undergo a similar modification, although in less simple dependence on the venous stasis.

DIAGNOSIS.—The essential sign of dilatation, by which its existence and degree may best be ascertained, is the diffusion of the cardiac impulse, its comparative uniformity over the whole area in which it can be felt. In proportion to the purity of the dilatation the first sound is toneless, high pitched, and short and weak; the pulse is small and feeble, and the lungs and general system suffer from the secondary consequences of the cardiac failure.

Obscuration of impulse may simulate diffusion, and thus lead to a mistaken diagnosis of dilatation. A thin layer of overdistended lung may intervene between the heart and the chest-wall, and so render the apex-beat indistinct and apparently diffused. The increased resonance over the cardiac area will indicate the cause of the indistinctness. Dilatation may also be simulated, as Niemeyer pointed out, when the apex strikes against a rib, and the impulse is felt equally in the interspace above and below the point of contact. This is most frequent in narrow-chested persons, whose ribs are near together. A mistake may be avoided by noticing this conformation of thorax, and by observing that the apex-beat is nearly in the normal situation, and that the apparent diffusion is vertical only; there is no lateral extension of the impulse.

From *hypertrophy* the diagnosis can rarely be one of absolute distinction. Some hypertrophy usually coexists with dilatation, and often confers on the diffused impulse increased force, and sometimes the pathognomonic "deliberate," heaving character. In proportion to the predominance of the dilatation, the impulse is weak and sudden, the precordial region is not bulged, the cardiac dulness is increased laterally rather than vertically, the impulse is extended laterally rather than lowered, and the pulse is weak rather than strong.

From *pericardial effusion* dilatation is principally to be distinguished by the direction of the increase in dulness which occurs in each condition—in dilatation laterally, in pericardial effusion upwards. The pyramidal apex of the latter, when distinct, is not simulated by the dulness of the dilated heart. The impulse of the heart and the dulness are conterminous, to the left at all events, in dilatation; while the dulness of pericardial effusion may extend beyond the impulse. The apex-beat is not raised in dilatation, and

[1] On Urinary and Renal Diseases, third edition, p. 356.

the sounds of the heart are as loud over the precordial region as at the top of the sternum, where in effusion they are most distinct (Walshe). Lastly, there is no friction-sound, and far less displacement of organs or precordial bulging, than in pericardial effusion. But precordial bulging and obliteration of intercostal spaces may be present in dilated hypertrophy, and in extreme dilatation the sounds may be much weakened. In a case recorded by Evans the right ventricle was actually tapped under the idea that it was a pericardial effusion.[1]

From *fatty degeneration* dilatation may be distinguished by the evidence of enlargement of the heart, by the diffusion of its impulse, and by the proportion between its diffusion and its weakness. In fatty degeneration, when it exists alone, there is no enlargement of the heart, and the change in the impulse is a simple weakening without diffusion. Often the two conditions are conjoined.

Prognosis.—The prognosis in dilatation of the heart is always grave. Unless compensated for by hypertrophy, its direct effect is to interfere with the function of the heart, and to lead to those serious results to which death is often due. Hence the gravity of the prognosis is proportioned (1) to the purity and extent of the dilatation; (2) to the existence of a tendency to degeneration rather than to growth, and of states of general malnutrition, defective food-supply, &c., which interfere with the occurrence of hypertrophy; (3) to the extent to which the dilatation is due to causes beyond control, to the amount of irremovable work which the heart has to perform.

Must the state, once established, be regarded as permanent? The *relative* amount of dilatation may certainly be lessened by the development of hypertrophy. There is some reason to believe that apart from the development of hypertrophy a dilated heart may lessen in size. It was long ago asserted by Beau and Larcher that dilatation is sometimes temporary when due to a temporary cause, and it has been said that a similar diminution may occur when, by absolute rest, the work of a recently dilated heart is reduced to a minimum. Individual cases have conveyed this idea very strongly to careful and unbiased observers. Milner Fothergill has lately brought forward strong evidence to show that such reduction in size may occur. He has shown that diminution in the cardiac dulness may correspond with the disappearance of the symptoms of dilatation, and afford evidence that the condition is itself diminished. The same conclusion is indicated by the completeness with which the acute dilatation of adynamic diseases, such as fever, may pass away.

[1] Clin. Soc. Trans. 1874–75.

Treatment.—The object of treatment in dilatation of the heart must be to restore as far as possible the disturbed balance between the cardiac work and the cardiac strength. The increased blood pressure, to which the dilatation may be primarily due, must be reduced to the minimum compatible with the work of the circulation. Accidental causes of obstruction must be removed. Bronchitis must be got rid of as soon as possible. Especially, exertion must be avoided. Rest, mental, moral, and physical, is of the greatest importance. Muscular exertion involves a large increase in the work of the heart, and its cessation will often suffice to restore the disturbed balance. In extreme dilatation, confinement to bed or the couch for a time is a wise measure, and will not seldom remove most of the troublesome subjective symptoms, and even some grave objective signs of dilatation. Where this cannot be secured, or is unnecessary by reason of the moderate degree of dilatation, the rigid avoidance of all needless and severe exertion should be enforced.

The blood-pressure may also be lessened by the reduction of the total volume of the blood. This may be accomplished in more than one way. The most ready method is by the abstraction of blood by venesection or cupping. The relief which it affords is often immediate and striking. The ultimate effect, however, is that the volume of the blood is soon reproduced, while the heart is permanently weakened. Hence it must only be employed when the need for immediate relief is paramount, and renders the danger of the ultimate damage a secondary consideration—that is to say, when the patient is in imminent danger of death. It is especially useful when the right heart and venous system are overloaded. The quantity of blood taken need not be large. In less urgent cases the same end may be obtained by other means, by purgation and diuresis. The former must not be severe, or the subsequent depression is not easily rallied from. Diuresis is often of great service in these cases, even where there is no dropsy.[1] The amount of fluid taken as drink should be small.

The power of the heart should be increased so that it may resist the blood-pressure, and may contract completely, so as to expel the whole of its contents. To this end the general nutrition must be,

[1] "In morbis pectoris, semper ducendum esse ad vias urinæ." Baglivi, quoted by Ferriar.

as far as possible, improved. A dry bracing air is useful, and gentle exercise should be taken which does not increase materially the work of the heart; food must be nutritious and easily digested. Iron is of great service, and seems to aid directly the production of the needful hypertrophy.[1]

Excited action of the heart must be calmed by avoiding the causes of excitement, and by sedative medicines. Moral emotion must, as far as possible, be avoided, and the sources of gastric disturbance guarded against or relieved. A distended stomach easily excites an attack of palpitation.

Of drugs having a direct action on the heart, none is so useful as digitalis, which increases the tone of the heart, lessens the frequency and increases the force of the contraction. There has been much discussion as to the action of digitalis, and the condition of heart in which it is of most service, but there is at present a consensus of opinion that its action is tonic, and that in dilatation its most marked beneficial effect is produced.[2] The heart's action is reduced in frequency and increased in force; irregularity in force and rhythm is lessened or removed. The sphygmographic tracing shows this effect. The grave consequences of dilatation are lessened, venous congestion, dyspnœa, and œdema, general or local, are all diminished.[3]

Concerning its *modus operandi*, there is still some difference of opinion. The lessened frequency of contraction probably lessens the work of the heart by diminishing that part which consists in moving its own mass, and at the same time the longer periods of rest probably conduce to the perfectness of the cardiac nutrition. Frequency of action is at the expense of rest, for the length of the systole remains nearly the same at various degrees of frequency of contraction, increased frequency being obtained at the expense of the diastole. It has been calculated that the time of rest to the heart which is contracting 144 times per minute, is increased by one-third if the pulse is reduced to 72.[1] Moreover, the smaller cardiac vessels, arteries, and veins, as well as capillaries, must be emptied of blood during the cardiac systole.[2] A certain time must elapse on each diastole before the capillaries can be filled with blood and transudation of nutritive fluid through their walls can take place. This period will be nearly the same in every diastole, and hence the total period of rest available for cardiac nutrition, will on this account also be greater the less frequent the contraction.[3]

Digitalis appears to act also by increasing the completeness of the contraction of the heart. Under its influence the heart of an animal becomes firmer at the end of systole. Such contraction insures the expulsion of the whole of the blood contained in the chamber. Every approximation to this is, in dilatation, a direct gain. It not only assists directly the circulation, but it arrests a process which is probably the main mechanism of the origin and increase of dilatation, viz., the over-distension of the chamber in consequence of the addition of residual blood to that which enters it from the ordinary source. Increased firmness of contraction will not only lessen the tendency to further dilatation, but will improve the condition of the cardiac walls,[4] and in-

[1] Chalybeate waters were recommended by Senac in commencing dilatation (Traité, 1769, t. ii. p. 330), and his recommendation was endorsed by Ferriar (Med. Hist. and Ref., vol. i. 1792, On Dilatation of the Heart, p. 168).

[2] Withering pointed out that digitalis "seldom succeeds in men of great natural strength," but does much good "if the pulse be feeble or intermitting, the countenance pale, the lips livid, the skin cold." An Account of the Foxglove, &c. Birmingham, 1785.

[3] "I do not intend to say how this medicine (digitalis) acts, but I can, from observation, declare, that it has a very powerful effect in obviating the urgency of the symptoms in dilatation of the heart" (Allan Burns, 1809, loc. cit. p. 57). Ferriar had previously largely used digitalis in dilatation. The verbal accord between these writers and those of the present day is more complete than is that of their meaning. Dilatation of the heart was to the former synonymous with its enlargement and over-action, and they valued digitalis for (and believed that it did good by) its supposed power of lessening such over-action, when it was really strengthening the heart's defective power.

[1] Milner Fothergill, Diseases of the Heart, p. 4.

[2] Harvey observed that the substance of the heart becomes pale during its contraction.

[3] Assuming, for instance, that the period required for the vascular distension of the heart, and not available for nutrition, to be uniform at different frequencies of contraction, and to amount at each contraction to one-tenth of a second, the total period then available for nutrition would be increased about three per cent. from this cause only, by a reduction in the frequency of the pulse from 100 to 80. But it is probable that the time needed for the vascular distension of the heart is shorter the greater the distension of the aorta, and hence that it is shorter the less frequent the contraction, and the actual increase in the period available for nutrition will be rather greater than is represented by the above estimate.

[4] Partly, no doubt, by rendering perfect the expulsion of the blood from the cardiac veins, as Dr. H. C. Wood points out (Phil. Med. Times, 1874, Nov. 14, 21).

crease the tendency to compensatory hypertrophy.

Digitalis acts also by steadying the heart, diminishing its irregularity. Dr. Ringer[1] suggests that its main effect in dilatation of the left ventricle accompanying mitral regurgitation is thus produced. By preventing irregular contraction it arrests that part of the regurgitation which depends on the irregular action of the papillary muscles, and so relieves the over-distension of the auricle, and indirectly of the ventricle.

Five to fifteen drops of the tincture of digitalis may be given with advantage three times a day. Most observers have found the tincture convenient and reliable, but the infusion is believed by Ringer to be a surer preparation, in doses of one or two drachms. Much larger doses have been given, but these should be employed with caution. Ringer recommends strongly that the minimum effectual dose should be employed in the first instance, since an increase after a time is often necessary.

The Virginian prune has long been employed as a cardiac tonic in America, and was introduced into this country by Clifford Allbutt,[2] who has found it very useful in cardiac dilatation. I have found its power as a tonic, although marked, inferior to digitalis; but it is of much value for the relief of continuous cardiac discomfort, and may with advantage be given for a time, while digitalis is omitted. Twenty or thirty minims of the tincture may be given three times a day. Nux vomica and strychnia are also useful in improving the cardiac tone. Arsenic has been recommended for the same purpose.

Treatment of Special Symptoms.—Cardiac discomfort, in various forms, whether as pain or palpitation, is the source of much distress. The tranquillizing influence of digitalis on the heart relieves much of the pain. Aconite is of use in the same way, and is of most service when "extreme irritability of contraction coincides with great weakness of beat" (Walshe). Half a minim or a minim may be given; its effects being watched. Drawing a few deep breaths will often arrest an attack of palpitation (Brown-Séquard). Belladonna may be given internally in doses ℥v to ℥xv three times a day, and is often of much service. Belladonna plasters have been condemned by some authorities as useless, but they certainly give relief to the cardiac discomfort. Patients constantly ask for their repetition. The tincture of the Virginian prune sometimes gives very marked relief to continuous pain, and will sometimes stop for a time slight pseud-anginal seizures. Opium has the same power over cardiac as over other pains. Hypodermic injections of morphia give quick relief, but their use has been discountenanced in grave heart diseases, on account of the fear of too profound a sedative influence on the heart. But Clifford Allbutt and Ringer have employed them extensively, and assert that $\frac{1}{10}$ or $\frac{1}{8}$ of a grain may be injected with perfect safety, even in grave dilatation. The relief to the patient is certainly in many cases most striking. A very small quantity will sometimes procure sleep, in cases of cardiac insomnia, when sedatives given by the mouth fail altogether. Tolerance of sedatives by the mouth in these cases does not always imply a corresponding tolerance of the hypodermic injection, and the first injection should therefore always be very small.

For paroxysmal pains, antispasmodics may also be given. Nitrite of amyl in inhalation, so useful in true angina, also gives relief to the pseud-anginal seizures, and to the sense of suffocation, which is sometimes troublesome. If necessary, it may be employed diluted with spirit. A few drops of chloroform, or what is more convenient, half a teaspoonful of chloric ether, inhaled with steam, is also useful.

Attacks of increased cardiac failure need general stimulants and antispasmodics. Alcohol, given with hot water, is one of the most rapidly diffusible stimulants. Sal-volatile and ether, with tincture of lavender, are the most convenient and most effective drugs. Stimulation of the ends of the pneumogastric nerve in the stomach seems to have some influence in exciting the heart's action, and effervescing drinks and carminatives are useful in that way.

In syncopal seizures the head should be placed low, and the remedies just enumerated should be employed. Active respiratory movement should be restored as quickly as possible. It is thus, and by arousing consciousness and will, that cold affusions and stimulating applications to the nostrils are of service.

The lung complications of dilatation of the heart, bronchitis, œdema, congestion, need the most stimulating special treatment for each variety. Stimulating expectorants, as ammonia, are necessary for the bronchitis; and congestion is best relieved by the cardiac tonics and stimulants already described, and by mild counter-irritation.

Cough is often an exceedingly troublesome symptom in these cases, it may be paroxysmal or constant, and out of all proportion to the expectoration. Morphia is generally necessary to control it; one-twelfth of a grain may be given by the mouth, and with it a few minims of the tincture of belladonna.

[1] Handbook of Therapeutics, 5th ed. p. 421.

[2] Medical Times and Gazette, Feb. 16 and March 2, 1867.

Dyspnœa is among the most obstinate, as well as the most distressing, symptoms of dilatation. Its chief treatment is that of the cardiac failure, and the same diffusible stimulants are needed. The paroxysmal form is relieved most effectually by more direct sedatives: opium, Indian hemp, belladonna, lobelia inflata. Dry-cupping, and a few leeches to the precordial region, are recommended by Walshe when there is palpitation as well as dyspnœa. Posture is important; the head should be well raised, and Walshe recommends an attitude leaning forward, with the forehead supported by a sling. When the dyspnœa is dependent on pulmonary œdema, relief is often only to be obtained in the sitting posture.[1]

Headache is not often a troublesome symptom except in dependence on the cough. It is best relieved by posture, and by bathing the forehead with hot water. Sleeplessness is often a distressing symptom. Rest is disturbed by sudden starts, or the patient wakes up in a sudden fright with a sense of great distress. Such symptoms may usually be removed by the administration of the third dose of digitalis at bedtime in combination with bromide of potassium. Indian hemp (gr. ½ of the extract or ℥x. of the tincture), will also, though less uniformly, give relief. Actual insomnia may be relieved by chloral, chloral and bromide, and morphia by the mouth or skin, employed with caution.

The congestion of the liver may be lessened to a marked extent by mercurials. Every relief given to the portal congestion no doubt lessens immediately the pressure upon the hepatic lobules.

Vomiting is sometimes a very troublesome symptom. Effervescing ammonia, with bismuth and hydrocyanic acid or morphia, is the most useful. The amount of ammonia need not be large; gr. x of the carbonate with gr. xj of citric acid is sufficient. Ice should be sucked, and food given in small quantities. Counter-irritation to the epigastrium is sometimes useful. Any portal congestion must be relieved, the bowels being kept open. Diarrhœa sometimes demands treatment, and should be moderated rather than restrained. If constipation is present, moderate doses of hydrogogue purgatives are most useful, as Püllna or Hunyadi Janos water, colocynth, or podophyllin. Flatulence is often very troublesome, and adds much to the cardiac and general distress. Hot fomentations externally and carminatives internally give most relief; sal-volatile, peppermint, chloric ether, spirit of horse-radish, are all useful. The relief which is afforded to the sufferers from dilatation of the heart by the removal or diminution of their gastric troubles is often very great. Dobell has lately drawn attention specially to this subject.[1]

The scanty urine consequent on the kidney congestion may call for treatment. Mild diuretics, with digitalis for its double action, often suffice to relieve the kidneys. Often, however, this long-continued congestion induces tissue changes in them, and dry-cupping to the loins, or stronger diuretic treatment—broom, juniper, &c.—may be necessary. Stokes[2] remarks that diuretics often succeed after a mercurial, where they have previously failed.

Dropsy is almost invariably a troublesome symptom in the later periods of a case. It is dependent partly on the blood-state, favoring osmosis, partly on the mechanical congestion, increasing the pressure of the blood in the small vessels, and increasing it to the greatest extent in the most depending parts, where gravitation aids the cardiac failure. It can only be effectually combated by treating each of these causes, first by strengthening the heart, and secondly by improving the blood-state. Hæmatinic and cardiac tonics are needful for this. But it may be lessened by other measures. No remedy can promote, directly, the absorption of the effused fluid from the cellular tissue back into the bloodvessels, but reduction in the volume of the blood exercises a marked influence. The abstraction of blood will be necessary only when acute pulmonary œdema threatens life, and then cupping on the chest is preferable to venesection. Often purgation is sufficient, and hydrogogue cathartics, bitartrate of potash, elaterium, jalap, are the most effectual. Where there is evidence of enlargement of the liver, a dose of a mercurial is stated by Hayden to increase the effect of the purge upon the dropsy. Diuresis occupies a position hardly second to purgation for the removal of dropsy; copaiba, iodide of potassium, nitrate of potash, juniper, broom, nitric ether, and especially digitalis, may be given. The dose of digitalis for the removal of dropsy by its diuretic action needs to be larger than when its tonic action alone is needed, ʒij or ʒiv of the infusion, or ℥x or xx of

[1] A reclining-chair, with a cross rest on which the forehead can be supported, has been for some years in use at University College Hospital, and a "heart-bed" for the same purpose is described and recommended by Dobell.

[1] On Affections of the Heart, 1872. The value of carminatives has long been known. Albrecht relates that Sylvius removed all symptoms in a case of cardiac dilatation by their use. According to Pliny, the Egyptians believed the juice of horse-radish to be the only cure for atrophy of the heart.

[2] Loc. cit. p. 263.

the tincture, may be given twice a day. Dry-cupping over the kidneys sometimes, it is said, increases the effect of the diuretic.

In severe cases all these means, successful at first, may ultimately fail to remove the dropsy, and it becomes necessary to relieve the distended limbs, or sloughing will occur. It is necessary to anticipate this and to scarify or puncture the skin, and allow the limb to drain. Scarification is the more effectual, but is said to be attended with greater risk of erysipelas. Erysipelas will rarely occur when the precaution is taken to wrap up the limbs in flannel, wrung out of warm water, immediately after the scarification, renewing it every two hours during the first two days. A harelip pin is a convenient instrument for the punctures.

Jaccoud[1] recommends, as a substitute for punctures, friction each day with croton oil: in a day or two the characteristic eruption is produced, and from it the serum escapes abundantly. The frequency with which a slight inflammation is the starting-point of a slough makes it difficult to believe that the risk of gangrene is lessened by this method.

In all cases of dropsy, as little fluid as possible should be taken. When the kidneys are underacting from congestion, its effect is, as Milner Fothergill[1] has insisted, only to throw an increased strain upon the heart.

[The propriety of so absolute a dictum in regard to fluid, may be questioned. Dilution of the blood, by lessening its stimulating quality, tends to diminish congestion of the kidneys, and thereby to favor secretion. Of all agencies employed in practice for diuretic effect, much experience shows that water is, ordinarily, the most potent; and hardly any diuretic will act well unless considerably diluted with water. Thirst is, no doubt, usually the best guide in this matter.—H.]

FATTY DISEASES OF THE HEART.

By W. R. Gowers, M.D.

Fatty degeneration of the heart consists in the substitution of fat for its muscular substance. This result may be reached by two processes of different pathological and clinical relations. The one process effects simply the molecular substitution of fat for the proper substance of the muscular fibres. The other consists in the overgrowth of the normal fatty tissue of the heart among the muscular fibres, so as to compress, and ultimately to destroy and replace them. The former process needs the microscope for its demonstration; the latter is obstrusively conspicuous to the naked eye. The one is an indication of diminished vitality, and may be its initial stage, a necrosis; the other is at first a growth. Some varieties of the two processes present common pathological features, and their effect on the function of the organ is the same; but their diverse conditions of origin and anatomical characters need separate description. In pursuing this course the example is followed which was set by Dr. Quain[2] in a memoir on the subject, which has served as a model for most subsequent writers. The hypertrophic form of "fatty infiltration" will first be described, and then the "necrobiotic" process of fatty degeneration.

Fatty Overgrowth.

Synonyms.—Fatty Infiltration (Rokitansky); Fatty Growth, Fatty Hypertrophy (Quain); Adipose Cardiaque, Surcharge Graisseuse, Obésité du Cœur (French writers).

Definition.—An abnormal development of adipose tissue on and in the substance of the heart. Fatty tissue is always present on the surface of the heart, and varies in amount according to the age, and the nutritive conditions and tendencies of the individual. In abnormal development this fat may become so excessive that mechanical interference with the function of the organ is the result. It is a local "instance of the extension into

[1] Pathologie interne, 4th ed. vol. i. p. 621.

[2] On Fatty Diseases of the Heart, Med.-Chir. Trans. vol. xxxiii.

[1] The Progress of Heart Disease, Lancet, vol. i. 1875.

the domain of disease of the physiological process of growing fat."[1]

History.—Some of the symptoms of obesity, which are in part cardiac, were among the earliest medical observations. Hippocrates noticed the tendency of fat persons to earlier death than others, and both he and Celsus observed the dyspnœa which is associated with obesity. Harvey described an excess of fat around the heart of Old Parr. Since that time almost every writer on diseases of the heart has alluded to or described a similar condition. Kerkering,[2] in 1717, noted its occurrence at so early an age as two years. Senac[3] in 1749 described carefully the normal variations in the quantity of fat according to time of life, &c. Morgagni[4] recorded examples of hearts so loaded with fat that no muscular tissue could be seen. Portal[5] noted the concurrence of fatty overgrowth in the heart with a similar condition in the voluntary muscles. The state was fully described by Corvisart, who suspected that it might be a cause of sudden death. Morgagni thought that the muscular fibres of the heart suffered in this condition of fatty growth. The microscope, long after, showed that this is actually the case. It was inferred, however, from the apparent substitution of the fatty for the muscular tissue, and from the evidence of cardiac weakness. Hearts subject to this mixed change were described, and the disease ably discussed, by Duncan (1816), Cheyne (1818), Townsend (1832), and R. W. Smith (1838).[6]

Causes.—Excess of adipose tissue on the heart is usually associated with excess of adipose tissue elsewhere, and is due to the same causes. Quain found that in almost every case of fatty growth about the heart there was general obesity. The converse holds good to a less extent. King Chambers[7] records that of thirty-six corpulent persons a considerable excess of fat at the base of the heart was found in twelve. On the other hand, in 165 bodies not remarkable for fat, there was excess of fat about the heart of four only.

Hereditary predisposition exercises a marked influence on the occurrence of obesity, and no doubt also on the occurrence of fatty infiltration of the heart. In two-thirds of the cases of general obesity it is found that hereditary or collateral obesity exists. Sex also influences its occurrence. If the statistics of Quain[1] be combined with the cases of fatty growth contained in the valuable tables of Hayden,[2] we have thirty-five cases of this condition of which twenty-five were men and ten women. The condition is therefore more than twice as common in the male as in the female sex. Age also exercises a distinct influence. At birth, as Senac pointed out, the heart is free from fat, and the amount increases as years go on. After six years fat is always present between the auricles and the ventricles. Fatty infiltration follows the same law and commonly occurs after middle life.[3] The combined cases of Quain and Hayden illustrate this very clearly. Under 20 there was but one case, between 20 and 30, three cases; between 30 and 40, none; between 40 and 50, four; between 50 and 60, eleven; between 60 and 70, nine; and over 70, seven cases. Sedentary habits increase the tendency to this condition. Food is an effective agent if the disposition to grow fat exists. Starchy, saccharine, and fatty foods are the chief fat-forming elements. Their effect is the supply to the system of a quantity of carbon in excess of the respiratory needs, and this carbon is stored up as fat. But if the oxygen supplied be in considerable deficiency, nitrogenous food may yield fat by its imperfect oxidation. Alcoholism also exercises a remarkable influence. Malt liquor seems to be more effective in causing fatty growth than spirits, but any form of alcoholism conduces to it; the blood in chronic alcoholism has been found to contain far more fat than in health. Sudden changes in the conditions of the system seem sometimes to determine the overgrowth of fat, general and local. An acute illness, and confinement to bed owing to an accident, are among the causes which Chambers mentions as having set the obese tendency in operation which has continued after the cause ceased to act.

Pathological Anatomy.—The fat normally present on the heart exists chiefly in the auriculo-ventricular and inter-ventricular sulci, extending thence on to the ventricles, especially on to the surface of the right ventricle. When excessive it may conceal from view almost the whole of the muscular tissue of the heart. It may remain confined to the surface,

[1] Hayden, Diseases of Heart and Aorta, p. 596.

[2] Opera Omnia Anat. 1717, p. 134.

[3] Traité, &c., vol. i. p. 187.

[4] De Sed. et Caus. Morb. Ep. iii. Obs. 20; xxvii. 2; xxx. 18.

[5] Mém. de l'Acad. des Sciences, 1784.

[6] See Fatty Degeneration—History.

[7] On Corpulence, 1850, p. 92.

[1] Loc. cit.

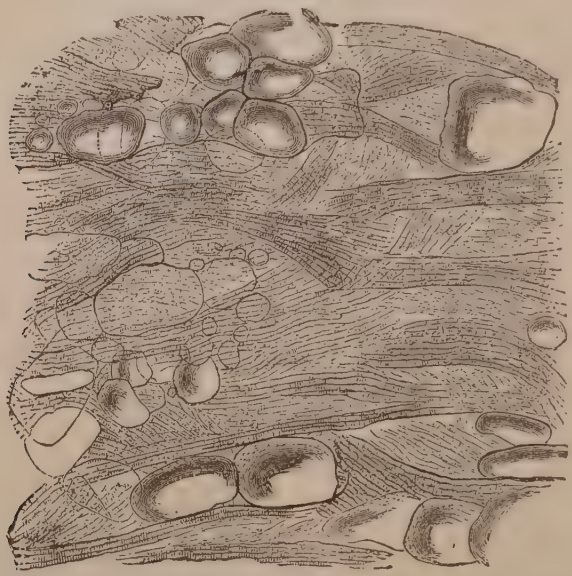
[2] Diseases of the Heart and Aorta, p. 648, et seq. These Tables are compiled from the Transactions of the Pathological Societies of London and Dublin, and from Dr. Hayden's own case-books.

[3] The case which is described by Kerkering stands almost alone.

but when considerable it usually invades the substance of the heart, passing in between the muscular fibres. On section the muscular substance appears narrowed, its junction with the surface fat being much nearer to the inner surface of the wall than in health. Streaks of fat may extend into the muscular tissue. Sometimes the latter is reduced to a thin endocardial layer, and even, as Laennec pointed out, the papillary muscles may appear to arise from a mass of fat. The muscular fibres are not really destroyed so completely as they appear to be: under the microscope they may still be seen in considerable number among the fatty tissue by which they are separated and displaced, and often pressed upon. Fatty degeneration of the fibres does, however, occur in a very large majority of the cases. Of the twenty cases contained in Hayden's table, in two only were the muscular fibres stated to be healthy.

The fat does not, however, always form such extensive and continuous layers. A follicular variety of fatty overgrowth was described by Laennec, and has since been generally recognized. In it the fatty tissue occurs in minute areas, which can be seen as specks in the substance of the

[Fig. 123.



Fatty Infiltration of Heart.—A section from the more external portion of the left ventricle of the heart showing the growth of fat *between* the muscular fibres. The fibres are in some places atrophied and commencing to undergo fatty metamorphosis. × 200. (Green.)]

heart, especially beneath the endocardium. In all conditions of fatty growth the fat is contained in oval and round cells, having an average diameter of $\frac{1}{1000}$ inch, and very similar to those which contain fat elsewhere. In cases of fatty overgrowth upon the heart there is usually also an excess of fat outside the pericardium.

Symptoms.—A considerable increase in the amount of fat upon the surface of the heart may be unattended by morbid signs. This was remarked by Corvisart and Laennec, and their observations have since been abundantly confirmed. Where the fat has invaded the substance of the heart, is infiltrated in the muscular tissue, the effect is a simple weakening of the heart, identical with that presently to be described as the result of the fatty degeneration, which is so frequently combined with the fatty growth. The impulse and sounds of the heart are weakened. The apparent weakening is greater than that which actually exists, because the subcutaneous and mediastinal fat obscures the impulse and dulls the sound. From the same cause the slight increase in the size of the heart which commonly exists is rarely to be detected. The actual diminution in the strength of the heart is often considerable. The pulse is weak, but may be perfectly regular even to the last. Dyspnœa is frequent, and syncope, and even rupture of the heart, may occur. The tendency to sudden death is very marked. Out of thirty-four cases in which the character of the death was noted it was sudden in twenty-four. Of these a third died from rupture of the heart, and another third from syncope. In every case of rupture, and in most of those in which syncope occurred, there was fatty

degeneration of the remaining muscular fibres.

DIAGNOSIS.—The diagnosis of fatty overgrowth in this condition depends on the recognition of the association of cardiac weakness and general obesity. The signs and symptoms are those of fatty degeneration. With such signs, if general overgrowth of fat is present, we are justified in suspecting the existence of fatty overgrowth and infiltration of the heart.

TREATMENT.—The treatment is essentially that for the general obesity of which the local overgrowth is but a part. The main object is to lessen the supply of the fat-forming hydrocarbons, and to increase their consumption in the system. The amount of food taken, if excessive, should be restricted; and fat, starch, and sugar should be, as far as possible, excluded from the diet. As much exercise should be taken as is practicable without putting undue strain upon the weakened heart. It is doubtful whether drugs possess any power of lessening the local accumulation of fat. Alkalies are believed by Chambers to diminish general obesity: whatever beneficial influence they exercise on general obesity they will also exert on the local state.

In other respects the treatment of fatty overgrowth is the same as that of fatty degeneration of the heart presently to be described.

FATTY DEGENERATION.

SYNONYMS.—Ramollissement (Corvisart, Laennec), Softening of the Heart; Carditis (Bouillaud); Greasy Degeneration (Hope); Fatty Metamorphosis (Rokitansky); Atrophie Graisseuse (Parrot); Steatose Parencymateuse (Blachez).

DEFINITION.—A change in the muscular fibres of the heart, by which the transverse striæ are at first obscured, and afterwards disappear, being replaced by granules and globules of fat. This granular and fatty degeneration of the heart, as far as we at present understand it, has nothing in its nature of specific character, but is simply the expression of defective nutrition of the proper substance of the fibre. Hence, as might be expected, the conditions with which it is associated, and to which, directly or indirectly, it is due, are widely different in their nature and mode of action. So diverse are they that it is evident that the condition of the heart is rather a common consequence, than a special disease. It has, however, its own symptoms and its own consequences, and so needs special description.

HISTORY.—Fatty degeneration was a late discovery in cardiac pathology. Morbid appearances, such as are now known to result from fatty degeneration, were mentioned by Robert Fludd[1] in the beginning of the seventeenth century, and by Lancisi a hundred years later, but received little attention. Overgrowth of the surface fat, and its invasion of the muscular tissue, were indeed described by Morgagni and many other writers, as has been already stated, and there can be little doubt that in some of the instances recorded, true fatty degeneration of the remaining fibres was present. Such a change in voluntary muscles was discovered by Haller and Vicq d'Azyr; and some French pathologists at the beginning of the present century suspected that a similar process might be the cause of some morbid appearances in hearts which did not present the ordinary characters of fatty growth. Corvisart,[2] who was perfectly familiar with the latter, had heard of this opinion, and considered the explanation plausible, although he had not himself seen the change referred to. In 1816, Andrew Duncan[3] described a heart which was probably an example of the mixed change, fatty growth and degeneration, and a similar case was recorded by Cheyne[4] in 1818. The naked eye characters of the simple degeneration were first accurately distinguished by Laennec[5] in 1819, who described the change in a limited area in very exact terms, recognized its identity with the degeneration described by Haller and Vicq d'Azyr, and gave to it the definite name of "fatty degeneration of the heart."[6] Bertin,[7] quoting Laennec's description, believed he had noticed the change in question, but admitted that he had confounded it with chronic softening, "of which," he said, "it is perhaps only a variety." General softening of the heart was described by Bertin as the effect of carditis.[8] It was

[1] Senac quotes from Fludd an account of a heart so soft and brittle that the fingers could be placed in its substance. It is said that the man from whom it was taken had played at cards two days before his death. (Senac, Traité, &c., vol. ii. pp. 382, 389.)

[2] Diseases of the Heart, Hebbs' Translation, p. 168.

[3] Edinburgh Medical and Surgical Journal, 1816.

[4] Dublin Hosp. Rep. vol. ii. 1818, p. 216.

[5] On Diseases of the Chest, Forbes' Translation, 1821, p. 229.

[6] It is difficult to believe that Laennec did not recognize the identity of "softening" and fatty degeneration, for he described the two conditions in identical terms.

[7] Traité des Maladies du Cœur, 1824, p. 431.

[8] Bertin noted (p. 400) as symptoms of "softening" many which are now ascribed

thus described also by Bouillaud.[1] The combination of fatty overgrowth with softening and degeneration of the remaining fibres was especially noted by Adams[2] in 1827, Elliotson[3] in 1830, Townsend[4] in 1832, and Latham in 1839. Simple fatty degeneration was described and distinguished from fatty growth by Hope[5] in 1839, and by Williams[6] in 1843, who compared it to the formation of adipocere.

Fresh interest was given to the subject in 1844 by the publication of Rokitansky's observation of the microscopical characters of the degenerated fibres.[7] In 1845 Peacock[8] published similar observations, made apparently in 1843, independently of Rokitansky's discovery. In 1847 a very clear description of the process in its wider associations was given by Paget,[9] and of its chemical pathology by Virchow.[10] In 1850 a series of cases illustrating the facts previously ascertained, were published by Ormerod[11] and by Kennedy,[12] and in 1805 Quain[13] contributed the very full account of the whole subject of fatty diseases of the heart, which has been already mentioned.

VARIETIES.—According to the appearance of the fibres, whether they contain granules or globules of fat, the two stages have been distinguished of *granular* and *fatty* degeneration, and it has been held that these two varieties are sometimes distinct forms of degeneration. There are, however, reasons for regarding them as stages of the same process, and both forms will be considered here. For convenience' sake the single term "molecular degeneration" may be used to denote them. "Primary" and "secondary" degenerations were distinguished by Quain, the former occurring without, the latter dependent on, preceding inflammation. Ponfick[14] would divide the degeneration into two forms, according as the muscular tissue of the heart was or was not in a preceding abnormal condition, and would further divide the cases in which the muscular tissue presents no other change than the degeneration, into "toxæmic," "senile," and "anæmic" varieties, according to their supposed causes.

ETIOLOGY.—(A) PREDISPOSING CAUSES. —*Hereditary Influence.*—A few facts are on record which suggest that fatty degeneration of the heart may own an inherited cause, and thus be transmitted. The cause may be a tendency to early decay of the muscular fibres, due to their defective vitality, or it may be a predisposition to one or other of the exciting causes of fatty degeneration, to be immediately described, especially to fatty overgrowth or arterial disease.

Sex has a marked influence. This is established by all the statistics which have been collected. The disease is at least twice as frequent in men as in women. Quain found the proportion 4 to 1, Ormerod about 3 to 1, Hayden more than 2 to 1. Ponfick states that the fatty degeneration which results from general anæmia is an exception to the rule, and is more common in women than in men.

Age.—Fatty degeneration of the heart may occur at any age. It has been found in the fœtus, and has been met with at every period of life from infancy to old age. But it is much more common in the second than in the first half of life. It is itself a degeneration, and it owns, as its frequent exciting causes, other degenerations, and is thus most frequent during the degenerative period. About three-quarters of the cases occur after forty years of age.

Habits of Life have probably less influence in causing fatty degeneration, than on the occurrence of fatty growth. The condition is more common among the lower classes than among the upper—the reverse of the proportion that obtains in cases of fatty growth. Sedentary habits predispose to imperfect nutrition of the muscular fibres, and some occupations act also by rendering the individuals liable to the exciting causes.

Depressing Emotions are believed by Quain to predispose, in some cases, to fatty degeneration. Moral emotion or long-continued physical pain is said to have such an influence.

Nutritive Influences.—The tendency to the formation of fat, which has so marked an influence on fatty growth, has apparently much less effect in causing fatty degeneration. Quain found that the disease occurred with almost equal frequency in fat and in thin persons.

(B) EXCITING CAUSES.—The exciting causes of fatty degeneration of the heart comprehend all those conditions which

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to fatty degeneration, such as weakened or inappreciable impulse, dulness of sound, and extreme frequency or slowness of the pulse.

[1] Traité Clinique des Maladies du Cœur, Ed. Quinzième, tome i. p. 615.

[2] Dublin Hosp. Rep. vol. vi. 398.

[3] Lumleian Lectures, p. 32.

[4] Dublin Journal of Medicine, 1832, p. 165.

[5] Diseases of the Heart, p. 348.

[6] Principles of Medicine, 1843, p. 304.

[7] Handbuch der Path. Anat. Bd. ii. 1844, p. 463.

[8] Monthly Journal of Medical Science, Jan. 1845, p. 20.

[9] London Med. Gazette, 1847 (lect. vi.).

[10] Virchow's Archiv, Bd. i. p. 152.

[11] London Med. Gazette, 1849.

[12] Dublin Med. Press, vol. xxi.

[13] Med.-Chir. Trans. vol. xxxiii.

[14] Berlin Klinische Wochenschrift, 1873, Nos. 1 and 2.

can interfere directly with the nutrition of its fibres. They are very diverse in character, but fall naturally into the two groups—general and local. They may act in conjunction with, or apart from, the predisposing causes.

I. *General Conditions* causing molecular degeneration of the heart are numerous, and various in their character. The tendency to degeneration may (1) be primary, or (2) it may be secondary to other morbid states, of which the most important are certain causes of general impairment of nutrition, certain states of poisoned blood, and certain poisons introduced from without.

(1) Fatty degeneration of the heart may own as its only cause the tendency to general degeneration which is natural to old age and which may occur at a much earlier date. This constitutional tendency is undoubtedly one of its chief causes. The degeneration is rarely confined to the heart; it is in most cases more widely spread and is seen in the inelastic skin, the rigid vessels, the white hair, the arcus senilis. But the tendency of such degeneration to unequal distribution is well known and may be manifested by disproportionate degeneration of the heart, especially when the conditions of life are such as to put an undue strain upon the organ. This degenerative tendency may or may not be associated with overgrowth of fatty tissue, and thus two types of degeneration are met with, the pathological tendencies of which Paget long ago pointed out.

(2) Fatty degeneration may be the result of some general condition of imperfect nutrition.

Anæmia both quantitative and qualitative may cause it. The influence of repeated losses of blood in causing this degeneration has long been observed as a clinical fact,[1] and it has been recently studied experimentally by Perl.[2] It can be readily produced in dogs by repeated bleedings, but much more readily by occasional large bleedings than by more frequent smaller bleedings. It was especially marked when the loss of blood amounted to three per cent. of the weight of the body. The papillary muscles of both ventricles are said to suffer first, then the walls of the left ventricle, and lastly the walls of the right ventricle. Stokes pointed out that depressing treatment may act in a similar manner. In idiopathic anæmia fatty degeneration of the heart also occurs. Biermer[3] has remarked that fatty degeneration of the bloodvessels often coexists. In pregnancy, intense anæmia sometimes occurs, and in such cases fatty degeneration of the heart has been found.[1]

Wasting Diseases were noticed first by Ormerod, to have as one of their consequences fatty degeneration of the heart. Those in which it is most frequently met with are phthisis, cancer, and chronic suppuration. In each condition the amount of degeneration may be considerable. In cancer it has seemed to the writer to be sometimes associated in degree with the degree of the fatty degeneration in the new growth. In Addison's disease it has also been met with.[2]

Toxæmic Influences constitute another group of causes. Fatty degeneration may result from many acute and some chronic blood changes. These include the various acute febrile conditions, specific and non-specific, and certain chronic diseases which alter the constitution of the blood.

In acute febrile diseases, molecular degeneration of the heart has been noticed by a large number of observers. Its naked-eye characters were distinguished by Laennec, and its conditions of origin and consequences were carefully studied by Louis and Stokes. Laennec pointed out that it occurred especially in those cases in which marks of "putridity" were present. By most writers the change has been regarded as inflammatory, as due to "myocarditis." It is certain that actual inflammation, as by extension from the pericardium, causes a similar degeneration. But in most of these cases, as Louis and Stokes pointed out, other evidence of inflammation is wanting; there is no purulent infiltration, no effusion of lymph on the pericardium; and Stokes pointed out that local inflammations are rare in the conditions in which this change occurs.

Moreover, identical changes may occur from other influences with equal rapidity, in which there is no suspicion of inflammation, but proof of a profound alteration in the state of the blood—as for example in phosphorus poisoning. Simple elevation of the temperature of the body has been shown capable of producing a similar degeneration.[3]

Many acute diseases are attended with this molecular degeneration. In acute rheumatism the condition is usually asso-

[1] Ormerod, loc. cit. p. 832.

[2] Virchow's Archiv, lix. 1. Similar experiments had also been made by Tschudnowsky. Botkin's Archiv, Bd. ii. 1866–67.

[3] Bericht über der 42en Versammlung deut. Naturforscher u. Aerzte. Dresden, 1868.

[1] Gusserow, Archiv für Gynækologie, 1871, ii. 2, p. 218.

[2] E. Wagner, Die fette Metamorphose des Herzfleisches. Leipsig, 1865.

[3] Iwaschkewitsch, Journal für Militärärzte, 1870, and Virchow's Jahresb. 1870, i. 179. Wickham Legg, Path. Trans. vol. xxiv. 1873, p. 226.

ciated with undoubted inflammation outside or inside the heart, and is confined to the adjacent fibres, and other evidence of inflammation is to be found where the change is most intense. But in other cases in which the blood-change is profound, a simple degeneration may extend through the whole thickness of the wall and be apparently related to the pyrexia or to the degree of the blood-change, as in other febrile diseases, rather than to the special form of the toxæmia.

The other diseases in which the change occurs are the various febrile affections, and especially those in which any pyogenic influence is at work. It is common, for instance, in erysipelas, puerperal fever, and smallpox. In the last it has been found to be very frequent.[1] It occurs also in yellow fever,[2] and malarial fevers.[3] In typhus and typhoid fevers,[4] it is also common, although other forms of degeneration are also found in the heart as well as in the voluntary muscles in these diseases. In typhus Stokes found that it marked some epidemics much more than others, and that it generally commenced about the sixth day. In typhoid, Wagner[5] found extensive fatty degeneration in nine cases out of fifty-nine. In diphtheria the change is also common. G. Homolle found it in six out of fourteen cases which he examined, and Parrot found it in almost as large a proportion.[6]

In measles also Parrot found it present in about one-fourth of fifty-four fatal cases. In one case it was extreme. Extreme degeneration of the heart has also been met with in acute atrophy of the liver.

Chronic alterations in the blood may cause fatty degeneration of the heart. It occurs in gout, as Charcot has pointed out.[7] At first it is slight, but as the disease progresses it may become very considerable and become a cause of sudden death. In the altered state of blood, which results from chronic renal diseases, it also occurs. It has also been found in purpura, scurvy, and the hemorrhagic diathesis;[1] in the latter perhaps as a result of the loss of blood. It has also been seen in trichinosis.

Certain poisons possess a remarkable power of inducing fatty degeneration of the heart in common with that of other parts. Foremost among these must be placed phosphorus, which has a very rapid action on the heart, liver, kidneys, and other organs, causing marked fatty degeneration in a few days. In a case recorded by Habershon, on the fifth day after a dose of five grains of phosphorus all the organs were the seat of fatty degeneration. The heart becomes yellowish or reddish-gray, soft, and friable, the fibres being filled with fat drops. According to Schraube[2] the affection of the heart is almost invariable in phosphorus poisoning. Arsenious acid, lead, and antimony[3] are said to cause a similar molecular degeneration. In poisoning by sulphuric and other acids it has also been found; and it occurs in greater degree the more readily the acid can get into the blood.[4]

Alcohol is, if not the most powerful, at any rate the most frequent toxic cause of fatty degeneration. In chronic alcoholism the blood is loaded with fat. The habitual use of ether and chloroform is said to have a similar effect.

II. *Local Causes.*—All local causes of atrophy of the heart (q. v.) may also cause the fatty degeneration of its fibres. This is, indeed, partly the mechanism by which the atrophy is produced.

External pressure may have this effect. Compression by fluid rarely causes molecular degeneration, but pressure by the contraction of lymph, limited in area, or by the pressure of calcified plates, may produce it. It is possible that the effect is in many cases produced, not by the direct pressure on the muscular fibres, but, as Walshe suggests, by the compression of the arteries and consequent defective supply of blood, a powerful cause of fatty degeneration.

Interstitial pressure on the muscular fibres may certainly, however, be an immediate cause of this fatty degeneration. It is seen in fibroid and fatty overgrowth. It is well seen in the effects of syphilitic and other growths in the heart. In each condition the fibres are compressed directly by the new tissue which is developed between them. In fatty overgrowth

[1] P. Sick quoted in Canstatt's Jahresbericht, 1866, ii. 39. Desnos and Huchard. Senac noted the frequency of syncopal death in this disease. Traité, &c. 1749, ii. 551.

[2] Bat. Smith, quoted in London Med. Record, 1874, p. 517.

[3] Ponfick, loc. cit.; Vallin, L'Union Méd. 1874, No. 23.

[4] Stokes, Diseases of the Heart, p. 366; Murchison, On Fever, pp. 256, 631.

[5] Loc. cit.

[6] Dict. Encyclopédique des Sciences Méd. vol. xviii. 1876, art. Cœur. It has also been observed in diphtheria by Bengelsdorf (Berl. Klin. Wochenschrift, 1871), and Bouchut (Gaz. des Hôp. 1872, p. 117).

[7] Maladies des Vieillards et Maladies Chroniques. Paris, 1868.

[1] Wagner, loc. cit.

[2] Schmidt's Jahrb. 1867, 209.

[3] Salkowsky, Virchow's Archiv, xxxiv. 1 and 2.

[4] Munk and Leyden, Berlin Klin. Wochenschrift, 1865, Nos. 49 and 50.

they may be little changed, but they frequently suffer, presenting narrowing and indistinctness of striation, sometimes simple atrophy, sometimes very distinct fatty degeneration. This latter occurs especially when any predisposing cause of fatty degeneration coincides in operation, such as congestion of the heart in fibroid overgrowth, sedentary habits, degenerative tendencies, or alcoholism in fatty overgrowths.

Local Anæmia from vascular obstruction is a frequent cause of extreme fatty degeneration. The obstruction is usually gradual, and due to atheromatous changes in the walls of the coronary arteries, calcification, &c.; sometimes it is sudden from thrombosis, or less frequently embolism. The left coronary artery is said to be affected more frequently than the right. The degeneration is limited to that part of the heart to which the diseased vessel is distributed. This connection was noticed by Quain, and his observations have since been abundantly confirmed. He found diseased coronary vessels present in thirteen out of thirty-three cases, and pointed out that the effect depends on the absence of anastomoses with other vessels, by which a collateral circulation could be established, the "terminal character" of the arteries, as it would now be termed, first demonstrated by Swan.[1]

Congestion of the walls of the heart is, as Jenner[2] pointed out, a cause of fatty degeneration of the muscular fibres. The degeneration is rarely simple, more or less fibroid growth is usually conjoined. The chief cause of such congestion is dilatation of the right side of the heart and obstruction, consequent on the distension of the auricle to the escape of the blood from the coronary sinus. Hence fatty degeneration of the heart is frequent in emphysema, long-continued pleural effusion, and diseases of the left side of the heart, which overload the right chambers.

Inflammation of the substance of the heart, "carditis," is also attended with molecular degeneration of the fibres. The effect is clearly seen in cases of pericarditis in which the inflammation invades the subjacent layer of muscular tissue. The depth to which the change extends varies according to the degree and duration of the inflammation. Sometimes only a sixteenth, sometimes a quarter, or more, of the whole thickness of the heart is thus damaged. Wagner found fatty degeneration of muscular fibres present in seventeen out of thirty-five cases of severe pericarditis which he examined. In other forms of carditis the muscular fibres suffer in the same way. In the rare cases of suppurative carditis the degeneration proceeds to the complete destruction of the fibres. It has been already stated that inflammation has been regarded as the mechanism by which the heart suffers in the acute febrile disease, and that these cases cannot justly be regarded as inflammatory.

Defective vitality of the muscular fibres of the heart has already been described as part of a general proneness to degeneration; it may also occur as a local condition. This influence is seen in the proneness of the fibres of certain individuals to undergo such degeneration, apart from any other exciting cause. It is seen also in the tendency of hypertrophied hearts to undergo this change. Other hypertrophied muscles have been said, after a certain period of use, to fail and undergo degeneration.[1] Fatty degeneration occurs with undue readiness in the newly-formed fibres, and in the majority of cases hypertrophied hearts present degeneration of some of the fibres. This is the case especially in conditions of valvular disease which entail venous congestion of the walls of the heart, but it is also found in other conditions of hypertrophy. In that which occurs in Bright's disease, for instance, E. Wagner found fatty degeneration in one-third of the cases (twelve out of thirty-five).

PATHOLOGICAL ANATOMY. — In considering the pathological anatomy of fatty degeneration it will be convenient to reverse the usual order and to describe first the microscopical changes in the fibres, and afterwards the alterations in the naked-eye characters which result from the minute changes.

The first indication of the degeneration is the appearance of minute black granules within the substance of the fibres. At first they may coexist with the normal transverse striation and seem to lie in rows between the primitive fibrillæ. As they increase they appear to replace the transverse striæ, which diminish in distinctness and finally cease to be recognizable. Often from the first no regularity can be observed in the disposition of the granules; they are scattered uniformly through the primitive bundle.

As the degeneration progresses the granules increase in size, and become translucent in the centre, being, in fact, globules of fat. These become larger, but rarely, as Quain observed, exceed the size of a blood-corpuscle. A linear arrangement of these fat globules is frequently to be observed: some are scattered throughout

[1] Med. Gazette, xlii. 751.
[2] Med.-Chir. Trans. vol. xliii.

[1] The hypertrophied biceps of the file-cutter is said by Clifford Allbutt, on the authority of Busk, to waste after a certain time.

the substance of the fibre, while others are arranged in rows. Ultimately they may occupy the whole area of the fibre: sometimes they are aggregated in one part of it, and the remaining space is clear, free from granules or striæ. The globules constantly appear to accumulate outside the primitive bundles; whether by the coalescence of granules formed there, or by migration from within the fibres, is not clear. The appearance is too constant to be accounted for by the accidental escape of globules when the section is being made. The muscular fibres are ultimately left clear; empty fibre sheaths appear to remain in their place. The existence of a sarcolemma to the muscular fibres of the heart has been denied: if absent, the appearance of the empty sarcolemma is simulated by the unchanged fibrous tissue between and separating the primitive bundles.

The affection of different fibres is rarely uniform. Some may contain many fatty globules, and others only minute granules, while others are still healthy. Similar degrees of affection may be observed in the course of the same fibre: one part may be healthy, in another part the granular stage may be present, and in another there are only globules of fat.

The globules and larger molecules of fat are soluble in ether and resist acetic acid. It is necessary to rupture the fibre in order to apply this test. There is some doubt whether the finer molecules at the earliest stage of the degeneration are all soluble in ether. It has been affirmed by some writers, but lately denied by Rindfleisch,[1] who maintains that at the commencement of a true fatty degeneration the granules are insoluble in ether. The point will be alluded to in its bearing on the pathology of the process. To the last the molecules and globules of fat maintain their appearance. They never blend into uniform masses such as occupy the cells in fatty overgrowth.

The effect of this molecular degeneration is to modify considerably the naked-eye characters of the affected part. It is changed in color. The granules and globules reflect light strongly, and render the tint paler. It becomes gray, ashy-gray or grayish-yellow. Laennec aptly compared the color often seen to that of a faded leaf. In the degeneration which occurs in acute diseases, the substance of the heart may be dark in color, from the rapid staining of the tissues consequent on the decomposition of the blood-corpuscles, and the escape and transudation of their coloring matter.

At the same time the consistence is changed. The affected part is soft and flabby. The fibres become brittle and break up easily into short pieces, so that a scraping from a cut section shows much shorter fragments of fibres under the microscope, than does that from a healthy heart. The effect of this brittleness of the fibres is to render the tissue friable and easily broken under the finger, and sometimes the change is so great that the tissue softens and breaks down in a limited portion, or the substance may be torn by a violent contraction of the heart. This diminished consistence may be the most conspicuous feature, and hence the change was described as "pale softening."

The part so changed may have a greasy aspect and feel, and may actually grease paper which is applied to it. The increase in the quantity of fat contained in the tissue is, however, smaller than might be expected. Hermann Weber, indeed, affirmed that there was no increase; and it has been established by other investigators that in slight fatty degeneration only the same amount of fat is to be obtained from a heart fattily degenerated as from a healthy heart. But it has been shown that in more considerable fatty degeneration, the amount of fat is increased from two or three per cent., to four or five per cent. over the quantity contained in a healthy heart.[1]

The distribution of the change varies. In the form which is secondary to acute diseases, the degeneration is often distributed uniformly through the whole heart. But frequently, as Louis and Stokes pointed out, the left ventricle is affected much more than the right. When secondary to pericarditis only the superficial layer is affected, adjacent to the inflamed pericardium. Occasionally, in fever, according to Stokes, the change may affect only the superficial layer. When arising from a chronic process it may be confined to the inner layer beneath the endocardium, or may be greater in that than in the superficial layer.[2] More commonly it is widely distributed through the heart, generally in the form of minute foci of degeneration, pale spots, lines, crescents, in apparent isolation, or connected, and forming, as has been said, a plexus of degenerated areas throughout its substance. The resulting mottling appears on section or may be visible through the endocardium. This form is often presented by the degeneration which succeeds hemorrhage. Lastly, a limited area of the heart's wall, generally near the apex, may be affected intensely and uniformly; the affected region is sometimes sharply

[1] Path. Gewebelehre, 1875, p. 16.

[1] Böttcher, Virchow's Archiv, xii.; Krylow, ibid. 1868, xliv. 4.' Stevenson, quoted in Wilks' and Moxon's Pathological Anatomy, p. 119.

[2] Ormerod, loc. cit. p. 832, case vii.

limited. It was this variety which first attracted the attention of Laennec. This form commonly results from vascular disease. Not rarely a diseased vessel may be traced passing directly into the area, as in cases which have been described by Quain and others.

When degeneration affects part of the heart the frequency varies with which different portions suffer. The ventricles are affected much more frequently than the auricles. Indeed, Ormerod doubted whether the auricles are ever affected: they are certainly occasionally the seat of this degeneration, and their wall may be affected in a limited area through its entire thickness. Quain found that in about half the cases both ventricles are affected, and that where one ventricle only is affected the left is diseased twice as frequently as the right.

The size of a heart the seat of fatty degeneration may seem to be increased, but this is due to the diminished firmness of the organ, in consequence of which it does not maintain its shape when placed on a table. In pure fatty degeneration the size of the heart is normal or only increased slightly by the occurrence of secondary dilatation, and not rarely it is diminished. A wall partially degenerated may be bulged out so as to cause a local dilatation of the cavity. Fatty degeneration, however, may and often does occur in hearts previously enlarged. Hypertrophied tissue, as already stated, undergoes degeneration more readily than healthy fibres.

Associated changes may be found in other organs, especially those which are causes of this condition or are other results of a common cause. Those most frequently met with are degenerated vessels, and fatty degeneration in other organs. Ormerod thought the fatty degeneration in other organs was more commonly associated with fatty degeneration of the right than of the left ventricle.

It has been already stated that hearts, the subject of fatty growth, frequently present fatty degeneration of the remaining fibres.

In all seats of fatty degeneration calcareous salts are apt to be deposited, but this seems to occur less frequently in the substance of the heart than in some other seats of degeneration. Most cases of "ossification of the heart" are cases of calcareous deposits in subpericardial inflammatory tissue. (See "Adherent Pericardium.") The papillary muscles are occasional seats of calcification of degenerated tissue. In rare cases calcareous deposits are found in the substance of the heart. In a case mentioned by Renauldin[1] the substance of the left ventricle of a man, aged 33, was infiltrated with grains of calcareous matter, larger towards the cavity of the ventricle. Some of them were as large as the tip of the finger. Two remarkable forms of calcification have been described by Coats[1]: in one the fibres were dotted with spherules of calcareous matter like globules of oil; in the other the process had resulted in a "petrifaction" of the fibres without change of form.

The blood has been said to contain fat in some cases of fatty degeneration. R. W. Smith stated that he had seen globules of fat visible to the naked eye in the blood after death, and Stokes noted the same thing. Some doubt attaches to these observations from the difficulty of avoiding the escape of fat from the divided tissues into the blood. Dumenil and Pouchet,[2] however, state that they found a considerable quantity of fat in the blood of a person, the subject of chronic alcoholism, who, on subsequent death, was found to have fatty degeneration of the heart and liver. Magnus Huss also affirmed that he had seen fat in the blood of drunkards.

PATHOLOGY.—The significance of this molecular degeneration is clear; it is a sign of lessened vitality, sometimes of actual death. But the nature of the process has been the subject of much discussion, and is still, to a considerable extent, obscure.

It seems probable that the first step in fatty degeneration is a molecular change in the muscular fibre, by which the fat which exists within it in an invisible form, combined with the protein constituent, is separated and precipitated in visible granules and globules. Invisible fat, to be detected by chemical analysis only, exists in the blood, the heart, and, in fact, all the animal tissues,[3] and is believed to be combined with the nitrogenous material, because it is found that the different nitrogenous substances have their own special forms of fat; that the fat of fibrin, for instance, is different from the fat of serum.[4] In the healthy heart the fat thus combined amounts to about two or three per cent. In moderate fatty degeneration, even when granules and some globules of fat are visible under the microscope, chemical analysis shows that there is no increase in the total quantity of fat.[5] It would thus appear that the first step of the degeneration is a separa-

[1] Journal de Méd., Jan. 1816. Quoted by Laennec, p. 231.

[1] Glasgow Med. Journal, August, 1872.

[2] Gaz. Hebd. de Méd. et Chir. 1862, p. 32.

[3] Virchow, in his Archiv für Path. Anat. i. 1847, p. 156.

[4] Lehmann, Physiological Chemistry; Virchow, loc. cit.

[5] Hermann Weber, Ormerod.

tion and precipitation of the combined fat. It is probable also that the granules, which constitute the first stage of degeneration, are not all of a fatty character; that some of them are of protein nature. Virchow suggested that the protein material may ultimately be changed to a soluble extractive and pass away, leaving the precipitated fat.

But in more advanced fatty degeneration the quantity of fat is greater than this explanation will account for. Fibres are seen to be filled with globules of fat, and analysis shows that the amount of fat in the tissue is actually considerably increased, often to double the normal quantity. What is the source of this additional quantity of fat? It must be either formed in the fibre or introduced from without. The former is the simpler explanation; by it the fat is supposed to arise by a chemical change, an imperfect oxidation, of the nitrogenous constituent of the fibre. This is the explanation which harmonizes well with the visible characters of the change, since the transverse striation disappears as the fat is formed. Rindfleisch[1] points out that the stage of "cloudy swelling" of cells, in which they are filled with minute granules soluble in acetic acid, if it does not resolve, passes into one in which the granules resist acetic acid and dissolve in ether. This view was suggested by Fick[2] and Rokitansky,[3] and has been adopted by Virchow, Paget, Quain, and others. It is certain that fat may be formed from nitrogenous material. The vegetable world affords many instances of this. Butyric acid, a fatty acid, may be formed by the decomposition of fibrin (Wurtz). Chemistry supplies other examples of the same class. If the fat, in molecular degeneration, is not formed by a change in the protein matter, it must be introduced from without. But the increase in the fat is sometimes found in situations in which it cannot have been introduced from without. Prolonged maceration in dilute nitric acid, for instance, will produce such a degeneration in healthy muscular fibres; a similar change is often seen in preparations preserved in dilute alcohol. Doubtless this is chiefly due to the separation of the combined fat, and it has accordingly been found that there may be no increase in the total quantity of the fat contained in the fibre before and after the occurrence of the degeneration. In some cases, however, a considerable increase in the amount of fat has been found; that is to say, there has been a considerable formation of fat. Handfield Jones[1] found the increase of fat to amount to nearly fifty per cent., and as any accession of fat from without is impossible it can have arisen only by the decomposition of the protein material. The formation of adipocere is another illustration of the same process, but this substance seems somewhat variable in its character and mode of formation. Ormerod, indeed, maintained that its composition always agrees with the composition of the fat of the animal, and that its fatty element is due to a change in, and infiltration of, the normal fat into the muscular and other tissue. But he found that one specimen consisted of at least half pure fat, and Quain found that adipocere from the muscle of a horse was almost entirely soluble in ether.

Other evidence, although less conclusive, of the origin of the fat from the protein matter, is drawn from the occurrence of extreme fatty degeneration in parts to which the blood cannot gain access. It is seen, for instance, in "infarctions." The area from which the blood-supply is cut off by embolism is the seat of intense fatty degeneration. It is seen also in the experiments (performed first by Wagner) in which portions of animal tissue have been inclosed in the peritoneal and other cavities of living animals, and have become changed to masses of fat. But these experiments are deprived of some of their significance by the fact that the inclosure of the fragments in an impermeable coating prevents any increase in the fat beyond that present in the healthy tissue (Burdach). Inorganic substances permeable to the animal fluids become charged with fat in just the same way as the organic tissue. These facts, indeed, negative all the significance of these experiments as proof of the transformation of protein substance. They do not, however, exclude the possibility that some of the fat may have arisen in this way, since an impermeable coating will prevent the access of oxygen, without which the oxidation of the protein material, imperfect though it be, cannot occur.

The fatty degeneration found in phosphorus poisoning, in poisoning by acids, and intense anæmia, has been regarded as further evidence of the formation of fat by imperfect oxidation of nitrogenous material, since it is believed that all these conditions act by a common mechanism, the diminished supply of oxygen to the tissues consequent on the diminished number of blood-corpuscles. The diminished oxidation is also indicated by the fall in the temperature of the body.

These facts prove that in fatty degen-

[1] Pathologische Gewebelehre, p. 16.
[2] Müller's Arch. 1842, p. 19.
[3] Path. Anat.

[1] British and Foreign Medico-Chirurgical Review, July, 1853, p. 59.

eration some of the excess of fat present in the fibres may be, and probably is, due to a chemical change in the protein constituent of the fibre. They do not however, exclude the entrance of some of the fat into the fibre from without. It has been argued, indeed, by Robin[1] and Ormerod,[2] and the view is supported by Walshe, that all the fat seen in the fibres in fatty degeneration enters them from without, that it is essentially an infiltration of the fibres with fat, derived directly from the blood, and replacing the protein constituent of the fibres, which has been removed by a process of atrophy. There are two ways in which such infiltration of fat is conceivable. Minute fatty globules may enter the fibre from the blood, passing through its wall as fat, just as the fatty molecules of the portal blood pass into the liver cells adjacent to the portal canals.[3] Or the fat may enter the fibre in invisible combination with the liquid protein material which must, in the normal course of nutrition, always permeate the fibres. Within the fibre this fat may be separated and precipitated by a process similar to that concerned in the separation and precipitation of the fat contained originally in the muscular tissue, the nitrogenous material passing out as "extractive." The constant repetition of such a process may fill the fibre with fat globules. There is some reason, as just stated, to believe that most of the fat which is found after a time in pieces of tissue inclosed in the peritoneal cavity of another animal passes in from without. If this is so with regard to substances separated from all structural continuity with the living tissues, it may be the same with the fat which is found in such excess in areas in which, in consequence of arrest of blood-supply, necrosis has occurred. The permeation of these areas with fat-containing plasma, from the adjacent healthy region, must constantly go on. The analogy of calcification, which often succeeds fatty degeneration, affords some support to this theory. Normally, the blood, heart, and other organs contain a small proportion of calcareous salts, probably combined with the protein substance. No transformation of the other elements can produce the lime salts which are found in the calcified tissue. They must enter the tissue from without, with the blood plasma, from which they are separated and deposited, while the nitrogenous element passes away. This process, continuing during a long time, ultimately effects a complete infiltration of the tissue with calcareous matter.

[1] Chimie Anatomique.

[2] Brit. Med. Journal, 1864, ii. p. 152; St. Barth. Hosp. Rep. vol. iv. 1868, p. 30.

[3] Fatty "degeneration" (infiltration) of the liver was produced artificially by Magendie and Gluge injecting fat into the portal vein.

A consideration of all the facts of fatty degeneration make it probable, then, that, as Handfield Jones suggested, each process may be concerned in the production of the excess of fat which is found in advanced fatty degeneration; that the fatty material at first seen in the fibre is merely separated and precipitated in visible form, and that the subsequent excess arises in part by a transformation of the protein material, and in part by the entrance of fat from without.

CONSEQUENCES.—The effect of fatty degeneration on the function of the heart is to lessen its propulsive power, and thus to lead to imperfect filling of the arterial system, and consequent visceral anæmia. This effect is much more marked than is the correlative venous distension, which is so prominent an effect of dilatation of the heart. The relative defective supply to the arterial system is recognizable in the symptoms which it causes during life, rather than by any pathological consequences which can be observed after death. These symptoms are described further on.

The fatty degeneration of the fibres may not only affect the function of the heart, it may lead to changes in its condition which have their own results. The weakened walls may yield unduly before the pressure of the blood, and the heart may become dilated. Such dilatation is rarely very great. Its mechanism and conditions have been already considered (Art. "Dilatation"). But the weakness which fatty degeneration produces may have a much graver result. The brittleness of the fibres may lead to their rupture, and when the degeneration extends through the whole thickness of the wall of the heart, the whole wall may give way. This accident, "rupture of the heart," is of such gravity and importance as to need detailed consideration, and it is therefore described at the end of this article.

SYMPTOMS.—The physical signs and the symptoms which attend fatty degeneration of the heart are usually indistinct and never distinctive. All are common to other morbid states. They depend on the diminished power of the heart, which modifies the signs of its action, and affects the function of other organs.

As the size of the heart in simple fatty degeneration is little changed, the area of dulness presents no alteration. The slight dilatation, which is the consequence of the fatty degeneration, rarely leads to the signs of enlargement of the heart. In a considerable number of cases the dulness is increased, but this increase depends rather on pre-existing hypertrophy or dilatation, or else it is due to concurrent fatty growth.

Diminished force of impulse is the most important physical sign of cardiac degeneration. The area of impulse, like the area of dulness, is only increased by coexisting conditions. As long as the impulse is perceptible, the apex-beat may, in most cases, still be felt. When dilatation has occurred in consequence of the weakening of the cardiac wall, the impulse may be diffused and peculiar in character, resembling, as Stokes remarked, rather the slight, general impulse of an aneurism than the normal impulse of the heart. When the patient is thin, and the lungs are small, so that the impulse of the heart can be well felt, a partial change in impulse may be observed to correspond to a partial degeneration. Stokes, for instance, observed that in fever, when the left ventricle was much more degenerated than the right, while an apex impulse might be lost, an impulse in the lower sternal region, due to the right ventricle, might be still perceptible.

The sounds of the heart are weakened in correspondence with the weakness of the impulse. The first sound, to which the contraction of the heart directly contributes, is that which presents the greatest change. It is usually toneless, shorter, and relatively high-pitched, and may become almost or even quite inaudible at the apex, only the second sound remaining. The first silence is longer than normal in consequence of the shortening of the first sound. The second sound is also weakened in consequence of the deficient distension, and therefore deficient recoil, of the aorta and pulmonary artery. When the first sound is shortened and raised in pitch it may resemble the second. The sounds of the fœtal heart are then very closely simulated, especially if the heart acts rapidly. When the degeneration is local, the sounds may be modified locally, just as the impulse. In the acute degeneration of fever, Stokes observed that the first sound might be lost over the left ventricle when it was still audible over the right, in cases in which the post-mortem examination showed the left ventricle to be the more affected. Walshe has observed a similar alteration of intensity in chronic disease under similar circumstances. According to Stokes, if, after ceasing to be heard for a time, the first sound reappeared, it was heard first over the right ventricle and afterwards over the left. In one case both sounds were inaudible for thirty-six hours before death.

Stokes believed that a systolic basic murmur might exist during the early stage of the degeneration. Other observers have noted the occurrence of an apex murmur due to regurgitation, and have ascribed it to fatty degeneration of the papillary muscles.

The rhythm of the heart's action varies much. It may be regular throughout, but is often irregular, chiefly, Walshe thinks, when dilatation coexists; sometimes it is frequent, even to an extreme. It may be slower than natural, and the diminution in frequency may proceed to a degree met with in no other affection. This was first pointed out by Adams. It may fall to forty, thirty, or twenty beats per minute. In rare cases it has sunk as low as eight or ten beats per minute for hours before death.[1] The pulse is weak and small, in proportion to the cardiac failure. Its rhythm, as a rule, corresponds with that of the heart; rarely it is less frequent than the heart's contractions, in consequence of the weaker beats of the heart failing to send a wave along the vessels sufficient to be felt.

Pain is not a common symptom, but now and then is complained of, and is sometimes very troublesome. It may be confined to the cardiac region, may be referred to the sternum, or may extend down the arm. It may be paroxysmal, and simulate angina pectoris in its characters. Occasionally true anginal seizures occur, but no direct relation is known between their occurrence and the fatty degeneration of the heart.

Syncopal attacks, as might be expected from the nature of the disease, are not rare; they are usually produced by some effort. They vary in intensity, sometimes amounting only to a sense of faintness, sometimes to loss of consciousness. They may be accompanied by a sense of great distress, as if death were impending. Death does not unfrequently occur in such an attack. Often in this condition cerebral symptoms are associated with those of cardiac failure. Convulsions may occur. Vertiginous sensations are not unfrequent. Walshe mentions a case in which loss of memory for recent events preceded each attack of syncope. Or the loss of consciousness, commencing as apparent syncope, may continue, and deepen, slowly or rapidly, to coma, with stertorous breathing. These "pseudo-apoplectic" seizures, as they have been termed, are usually brief, and leave no paralysis. They have, however, a great tendency to recur. They were referred by Adams and R. W. Smith to congestion of the brain, but Stokes pointed out that their association is with a deficient supply of arterial blood, and that they are probably due to cerebral anæmia, the immediate cause of the syncopal seizures. In confirmation Stokes mentioned a case in which they could be averted at their onset by hanging down

[1] Ormerod thought that infrequency was associated rather with fatty infiltration than with fatty degeneration. Lond. Med. Gaz. 1849, p. 917.

the head so that it nearly touched the floor. When death has occurred in these attacks, the brain has appeared free from organic disease. It is needless to remark that apoplexy from actual organic changes in the brain may occur in subjects of fatty degeneration of the heart. Other occasional symptoms on the part of the nervous system are numbness and formication, such as have been attributed to anæmia of the spinal cord.

Dyspnœa is a common symptom. It may be slight, felt only on exertion, especially on ascending an incline or on making some other effort. Or the dyspnœa may be severe, constant, amounting to a continuous sense of suffocation. Considerable dyspnœa is said to be present in one-half of the cases of pronounced fatty degeneration. Occasionally it has a special form. Sometimes frequent sighing is observed, as Stokes pointed out. Sometimes the dyspnœal breathing possesses a peculiar rhythm of striking character, which has attracted much attention since it was first described by Cheyne.[1] It was very carefully studied by Stokes,[2] and by him especially associated with fatty degeneration of the heart, although further observation has shown that it is by no means confined to that affection.

This form, which has been termed the "Cheyne-Stokes dyspnœa," or "ascending and descending respiration," is characterized by recurring series of respiratory acts, first increasing and then decreasing in intensity. In the intervals breathing seems to have almost or entirely ceased; then slight respiratory movements are noticeable, which gradually become deeper and deeper, until an acme of very deep and labored breathing is reached, after which the respirations gradually become shallower until they subside into the same apparent apnœa, which is again broken by the gradual onset of another series. In the classical case recorded by Cheyne, the cycle included about thirty respirations and lasted a minute. In most of the other cases recorded it has occupied a shorter time. Hayden has found the pulse unchanged during the paroxysms.

As just stated, this form of dyspnœa is by no means confined to fatty degeneration of the heart. It has been seen in other forms of heart disease, especially in valvular disease with dilatation[3] and in atheroma of the aorta.[4] It has been met with even more frequently and at all ages in affections of the nervous system, in cerebral hemorrhage,[5] in tumors of the brain, uræmia, and tubercular meningitis.[1] It has frequently been seen in cases in which affections of the heart and brain coexist. It has been produced artificially in animals by Filehne[2] by the injection of morphia and subsequent inhalation of ether and chloroform. It has also been observed in a case of fatty degeneration of the heart, during the narcosis which followed a fatal injection of morphia, and also in chloral narcosis. Its probable explanation lies in a lowered sensibility of the respiratory centre in the medulla oblongata, as was suggested first by Walshe,[3] and after him by Laycock and Traube.

A form of dyspnœa which has in several instances been described as that of Cheyne, is that in which the dyspnœa subsides slowly into dozing apnœa, to be broken after a few seconds by a sudden rouse to conscious, or half-conscious, dyspnœa, which, after a few seconds, slowly subsides. This occurs rather in dilatation than in fatty degeneration of the heart. It seems readily explicable on the theory of diminished sensitiveness of the respiratory centre which requires a voluntary or half voluntary reinforcement. The latter is only excited by a stronger degree of the physiological stimulus ("anoxæmia") than the former; the blood, when well aërated by the dyspnœal respirations, ceases to excite it; sleep gradually withdraws the reinforcement, and the respiratory centre ceases to act; the apnœal venosity of blood increasing, at last awakes the higher mechanism to renewed action. But the true Cheyne-Stokes breathing differs from this in the very gradual increase in the breathing, from shallow to deep, as the dyspnœa comes on.[4]

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[1] Dubl. Hosp. Rep. 1818, p. 216.

[2] Dubl. Journal of Med. Science and Diseases of the Heart, August, 1846, p. 324.

[3] Seaton Reid, Dub. Hosp. Gaz. 1860.

[4] Hayden, loc. cit. p. 632.

[5] Laycock, M. Fothergill, &c. The writer has also seen it in one case of cerebral hemorrhage, and has been informed of two other cases in which it was marked.

[1] Traube, Roth.

[2] Berlin. Klinische-Wochenschr. 1874, Nos. 13, 14, 32, 35.

[3] Diseases of the Heart and Aorta. Third Ed. 1862, p. 345.

[4] Several theories have been framed to explain the details of the phenomena. Traube accounted for the slow accession of the dyspnœa by supposing that the venosity of the blood first excites the terminal branches of the vagus in the lungs, which, it is known, can liberate only slight reflex respiratory movements, too slight to prevent accumulating venosity and general stimulation of the afferent nerves, producing the intenser dyspnœa. The gradual onset of the returning respiration is not, however, difficult to explain, for it is the natural form in which the physiological stimulus manifests its returning action after it has been withdrawn by the abundant aëration of the blood in the dyspnœal breathing. A state of apnœa may easily be produced in health by a series of very deep respirations. The highly oxyge-

In some cases there may be from first to last no embarrassment of the breathing. Walshe has pointed out that this freedom from dyspnœa may accompany even the syncopal and apoplectic seizures. Cough is sometimes present without bronchitic or other cause.

The other symptoms referable to the general system are in the main those of defective blood supply. The skin is pale, the muscular power deficient, the surface and extremities cold; the mind is weak, often depressed. The digestive organs suffer; anorexia is common. Symptoms of over-distension of the venous system are rare. Slight œdema may occur, but marked dropsy probably never occurs as a consequence of the fatty degeneration. It sometimes results from coexisting dilatation, especially when primary. It is only under such a condition that the urine contains albumen. In simple fatty degeneration of the heart the urine presents no deviation from the normal. Coexisting degeneration of other organs often modifies the general characters of the symptoms of fatty degeneration.

[*Absence* of symptoms of fatty degeneration of the heart must be rare, but it is sometimes met with. In the case of a lady well known to me, who died at about sixty years of age, nothing occurred to show failure of health until the last day of her life. Her *physique* was fine; she was accustomed to walk two or three miles a day, and to go up several flights of stairs without inconvenience. Autopsy[1] showed rupture of a decidedly fatty heart.—H.].

Course and Terminations.—The course of molecular degeneration of the heart varies according to the circumstances under which it arises, and especially as it occurs gradually as a slow degeneration, senile or premature, or acutely in consequence of blood-poisoning.

In senile degeneration the symptoms are gradual in onset, and may be marked, or may be very obscure. The duration of the affection may be long, sometimes twelve to fifteen years. In these cases other causes often increase the effect of age, and may be to some extent removable, and the extension of the degeneration may be arrested for a considerable time. Sooner or later the cardiac failure comes; late, if the conditions of a tranquil unemotional existence can be secured; soon, if the sufferer has still to face the storms of life, physical and moral. In an acute illness, preceding degeneration of the heart prejudices very much the patient's state. The pulse becomes weak and irregular, often, as Kennedy showed, extremely frequent; and, if the acute disease be at all severe, syncopal failure occurs. Under other conditions the end may come as slow failure, or sudden stoppage from loss of power, or from rupture. The latter occurs in a considerable proportion of the cases in which the disease is well marked. When the coronary vessels are diseased and the heart degenerated, the sudden complete obstruction of a large branch will stop the damaged heart.[2] The more acute degeneration commonly occurs in the course of some pyrexial affection. It is characterized by more or less sudden failure of the heart's action, out of proportion to the other evidences of intensification of the general disease. The condition usually corresponds with a high temperature, and often occurs before the primary disease has begun to subside. When the patient recovers, and the pyrexial stage is over, the action of the heart may become very infrequent or may remain unduly frequent.

The form of degeneration which succeeds a hemorrhage is marked by more

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nated blood no longer stimulates the respiratory centre; no *besoin de respirer* is felt, and, except by a voluntary effort, no respiratory movement is made, until, after a few seconds, the slowly increasing state of blood causes respiratory movements, slight at first, afterwards deeper, until the normal respiration is reached. To explain the degree and duration of the dyspnœa, as well as its gradations, Filehne assumed that vaso-motor spasm causes continued stimulation of the respiratory centre, until that spasm is slowly relaxed by a degree of aëration of the blood which ceases to stimulate the respiration, and thus the slow relaxation of the spasm causes a slow diminution, and finally cessation, of the respiratory movement. He found that by simple alternate compression and relaxation of the arteries in a guinea-pig (right innominate and left subclavian) he could produce perfect Cheyne-Stokes respiration. In further confirmation of his theory he states that he has arrested the characteristic breathing by inhalation of nitrite of amyl.

It is not difficult to understand the origin of this form of respiration in cerebral diseases, in which the lowered sensitiveness of the respiratory centre is likely, and the withdrawal of higher influence may leave its tendency to rhythmical action free to modify a series of its actions. Its connection with cardiac diseases is less easy to understand. Little's theory of unequal action of the ventricles is certainly unsupported. Hayden suggests that the etiological condition is atheroma of the aorta interfering with the supply of arterial blood to the peripheral vessels. This explains the occurrence of dyspnœa better than its rhythmical cessation. Long-continued over-stimulation of the respiratory centre may possibly lead to its diminished sensitiveness.

[1] This case occurred in the practice of Dr. Lodge, of Merion, Penna.—H.]

[2] Payne, Brit. Med. Journal, Feb. 5th, 1870.

gradual sinking, the patient becomes weaker and weaker, and dies asthenic at the end of a few days or a week or two, from the loss of blood.

Diagnosis.—It will be gathered from the preceding remarks that the diagnosis of fatty heart is never easy and is often difficult or impossible. The opinion of Latham that the existence of the disease does not admit of positive recognition, only of probable conjecture, is that of most later authorities. Many of the symptoms which are the most uniformly connected with fatty degeneration of the heart, are also due to so many other conditions, that they have not, even conjointly, much significance. The diagnosis, as far as it can be made, depends on the following points:—

(1) *On the Simple Loss of Power.*—A very similar loss of power may be due to dilatation, but dilatation diffuses the impulse and enlarges the heart; neither effect belongs to degeneration, unless dilatation is associated with it, and then the muscular degeneration can rarely be detected. Such simple loss of power may, however, occur in either a normal or a hypertrophied heart. In each it has the same significance, but in the latter the weakness is commonly relative only. It needs in all cases to be carefully distinguished from concealment of impulse in consequence of over-distension of the lung. A mistake may be avoided by attention to the other signs of emphysema, and especially to diminution or obliteration of the superficial cardiac dulness, which always occurs when a distended lung pushes the heart away from the chest wall. Weakness of the first sound of the heart is also most valuable, in the absence of emphysema, as concurrent evidence of the diminished force of its contraction. Other symptoms of fatty degeneration are of less significance, except perhaps slowness of pulse, which is, however, rare; the special forms of dyspnœa are also too rare, and they are also too equivocal, to be of much value. Some weight has been attached to the syncopal seizures which occur in this disease, and especially to the mixture of syncopal and cerebral symptoms. Mental depression has also received attention as adding weight to other symptoms. Pallor of the surface has a similar significance.

(2) *On the Presence of Similar Degeneration elsewhere.*—Senile degeneration is in some persons local, much more commonly it is general, and its wider manifestation may give significance to cardiac symptoms, which alone would be of little import. Of these degenerations the most important are those of the vascular system, of which the heart is part. The smaller vessels are accessible to direct examination, and their degeneration is manifested by hardness and tortuosity. Perhaps of next significance is the change in the cornea known as "arcus senilis," and which, since it was shown by Canton to depend on fatty degeneration, has attracted much attention as convenient indication of a diathetic tendency to such a change. That it has such significance in some cases is unquestionable,[1] and it has been thus accepted by Quain, Barlow, Paget, and others. But its value may easily be over-rated. Like every other local degeneration, it may be part of a similarly wide-spread change or it may be an isolated phenomenon. The latter is the case perhaps, more frequently than the former, and has led many observers to deny that any weight can be attached to it as evidence of fatty degeneration of the heart. Haskins[2] has recorded twelve cases with no affection of the heart. A wider observation has shown that the truth lies between the two extremes, and that the arcus senilis, as already stated, may give weight to other characters but alone is of little significance. Other evidences of degeneration are of still less value; but grayness of hair is probably a stronger evidence of degenerative tendencies than is its loss.

(3) *The existence of a recognized cause* of fatty degeneration is also of considerable value as an aid to diagnosis. Of the various causes, chronic alcoholism is that which is most frequently associated with the degeneration; and most frequently assists the diagnosis.

Prognosis.—Molecular degeneration of the heart is always serious, but its gravity varies much under different circumstances. The condition to which danger is especially related is the persistence of the cause of the degeneration. As long as the cause lasts, the degeneration continues and increases. Life depends on the maintenance of the functional power of the walls of the heart, and progressive degeneration must sooner or later produce death. If the cause of the degeneration ceases to act, the disease ceases to progress, and if moderate in degree, may, it is probable, even be recovered from. When a certain point of damage has been reached, the condition seems to preclude more than partial restoration of structure.

The forms of degeneration which occur in acute diseases are those in which the immediate danger is greatest, but at the same time the ultimate prognosis is usually favorable. It is immediately grave, because the heart is often unable to resist the prostrating influence of the general

[1] Luithlen, Virchow's Archiv, 1871, p. 91.
[2] Am. Jour. Med. Sciences. January, 1853.

disease. It is ultimately good, because the causal disease soon ceases, and often before the change in the heart has reached an irreparable degree. After the acute illness is over, the patient may die from the subsequent slow failure of the heart, but frequently he recovers, sometimes completely, sometimes with some permanent damage to the substance of the heart.

In chronic degeneration the immediate prognosis is less grave, but the ultimate prognosis is worse than in the acute cases. Those are the most hopeful in which there exists a removable cause of degeneration, such as the consumption of alcohol. Where the malady has arisen as a senile degeneration, or as a widely-spread idiopathic change, and the conditions of life are unalterable for good, the prognosis is very unfavorable. It is worse also the earlier in life the patient is attacked, since, as Quain pointed out, early age often entails an inability to obtain that rest which alone can ward off the consequences of the disease.

The fatty degeneration of the heart which coexists with a like degeneration in the vessels has been regarded as being not without advantages: adapted, in senile atrophy, to the lessened mass of blood,[1] and diminishing, by the lessened strength of contraction, the strain to which rotten vessels are exposed.[2]

TREATMENT.—Advanced fatty degeneration of the heart is generally an irremediable condition. Something may be done to ward off its effects, but little to restore the heart to its normal state. In slight degeneration, improvement, even perhaps recovery, may take place. The great end to be aimed at is the removal of the cause. In the acute degeneration of pyrexia there is, as stated, every reason to believe that a state of granular degeneration may be recovered from, when the cause has ceased to act. A chief object in treatment must therefore be to maintain the strength of the patient, to keep his heart going by judicious stimulation until the disease is over. General tonics will then aid recovery. Strychnia has been thought by many to be of great use.

In the chronic forms of degeneration there is frequently little room for therapeutics. The change is too often due to conditions beyond the influence of any means at our disposal. All removable causes, however, such as chronic alcoholism and gout, must be carefully searched for, and their influence, if possible, removed or neutralized. A fair amount of nitrogenous food is necessary. Restriction of fat is of more doubtful benefit. Tonics are useful—iron, quinine, or strychnine. Digitalis has been recommended to strengthen the fibres which are weakened but not destroyed. Walshe says it is most useful where the pulse is frequent and dilatation coexists. If the degeneration can be arrested, hypertrophy of the remaining fibres may occur, and help to restore the functional power of the heart.

In every form of degeneration care must be taken not to overtax the heart. Its weakened texture is easily damaged still further, and approximate health depends on the avoidance of exertion, &c. Effort with closed glottis must be especially avoided, such as pulling on boots, lifting weights, straining at stool; the latter has in several instances been the immediate cause of cessation of the heart's action. All causes of syncope must also be carefully avoided. In acute illness, the horizontal posture must be carefully maintained as long as the cardiac failure continues, and it must be left off with caution. The general health must be carefully attended to. A life in the open air is strongly praised for such cases by Stokes. The digestive organs must be put right, and the heart preserved from every cause of embarrassment to its action.

Stimulants are needful for the cardiac failure, and a diffusible stimulant may be kept at hand for the syncopal attacks. Coffee has been strongly praised by Desnos and Huchard in the degeneration of smallpox.

Hayden has found the nitrite of amyl of service in the paroxysms of rhythmical dyspnœa. It is equally useful in the attacks of suffocative oppression or anginal pain.

Pain may be relieved by sedatives such as have been recommended for the pain in dilatation of the heart. But equal caution is needful respecting the use of opiates, especially by hypodermic injection. I have known half a grain of morphia, injected hypodermically, followed by death, of which there was no premonition. Ormerod relates a case in which the same quantity was taken by the mouth, and death occurred during the ensuing sleep. Chloroform should not be inhaled; ether should be employed instead. In a large majority of cases of death while under the influence of chloroform, fatty degeneration of the heart has been found.

RUPTURE OF THE HEART.

This accident occurs in a considerable proportion of the cases of fatty degeneration, both simple and associated with overgrowth of fat. Conversely, fatty de-

[1] Crisp, Treatise on the Bloodvessels, 1851, p. 363.

[2] Sir W. Jenner, Address to the British Med Association, 1869.

generation is by far the most common cause of rupture. Spontaneous rupture never occurs in a healthy heart, and the number of cases due to any other cause, as abscess, or aneurism, or deep endocardial ulceration, is very small. Out of one hundred cases of rupture collected by Quain,[1] in seventy-seven fatty degeneration was detected by the microscope, and of the remaining cases, in all but two either softening was noticed, or the state of the heart was not mentioned. It is thus probable that in at least nine-tenths of the cases of rupture fatty degeneration is the condition of the cardiac wall to which the accident is due.

The degeneration which permits rupture is rarely uniform throughout the whole of the cardiac walls. Uniform degeneration causes uniform weakening; the force of contraction is lessened, and there is no spot specially incompetent to bear the lessened strain. It is when the degeneration, as is so commonly the case, is unequal, and especially when the degeneration in a limited area reaches a high degree, that rupture takes place. The contraction of the more healthy portions of the cardiac wall puts upon the more rotten portion a strain which the former can bear, but which the latter is quite unable to bear. This unequal change is the form which is associated, as its immediate cause, with local and degenerative, rather than with general or inflammatory causes. It rarely, for instance, results from the damage to the cardiac wall, from endo- and pericarditis, or from pyrexia; whereas it is common in the degeneration secondary to unequal fatty growth and infiltration, and still more frequent in that which results from vascular obstruction, chronic or acute. It is not uncommon to find a degenerated or thrombosed branch of the coronary artery going straight into a patch of intense fatty degeneration in which the rupture has occurred.[2] The sudden occlusion of a vessel by embolism may cause a similar patch of softening.

There is another way in which rupture has sometimes been produced by the association of diseased vessels and fatty change. The degenerated vessels may give way; the resulting extravasation readily tears its way in the softened tissue, and may reach the surface, being assisted, no doubt, by the contractions of the heart. The systole of the heart empties its vessels of blood, and when a hemorrhage has occurred into the substance of the wall, the contraction must compress the effused blood, and force it in the direction of least resistance. It is not uncommon to find more than one extravasation in the wall of the heart.[1] Such hemorrhages are said to be sometimes the result of embolism.

The chief other causes of rupture, aneurism of the heart, cysts in its walls, &c., are considered elsewhere.

The influence of the degenerative conditions is seen in the effect of age on the occurrence of rupture of the heart. The accident occurs chiefly in the old. Of the cases collected by Quain, two-thirds were over 60. The mean of forty-eight cases has been found to be 68 years.[2] Most collections of cases have shown the accident to be more frequent in the male sex, but Quain's statistics give an equal number of cases in each sex. Occasionally, hereditary predisposition has appeared to influence the occurrence, and even the seat of rupture, perhaps by similarity of vascular distribution. A classical instance is the death of George II. and his relation, the Princess of Brunswick, of rupture of the right ventricle.

In primary rupture of the heart the immediate cause of the tear is probably a contraction of undue strength, the strain upon the fibres being greater than the degenerated texture can resist. Thus the accident has commonly occurred during conditions of excited action of the heart, during unusual effort, such as running to catch a train, lifting a weight, cough, straining at stool,[3] or during emotional excitement. Of twenty-four cases collected by Barth, in five death occurred during the act of defecation. Sometimes no undue exertion can be traced, the symptoms come on suddenly while the patient is at rest, even during sleep.[4]

All parts of the muscular substance of the heart are liable to rupture. It has occurred in the walls of each ventricle, of each auricle, in the septum between the ventricles, and in the papillary muscles. It is, however, far more frequent in the left ventricle than elsewhere. All statistics agree in showing that the left ventricle is the seat of rupture in three-quarters of the cases, and that it is at least twice as frequent in the anterior as in the posterior wall. The usual seat is near and parallel to the septum, and not far from the apex. About twelve per cent. of the cases occurred in the right ventricle,

[1] Lumleian Lectures, Lancet, 1872.

[2] Quain, Path. Trans. iv. 80. Simon, Berl. Klin. Wochenschrift, 1872, No. 45.

[1] Colin, Gaz. des Hôpitaux, 1867, p. 104.

[2] In the few cases on record of rupture of the heart at earlier ages, most were due to other causes than fatty degeneration. In rupture of the left ventricle, for instance, in a woman aged 49, described by Gregorie (Virchow, Jahresb. 1870), the cause was the perforation of a circular ulcer, probably, since the woman was the subject of constitutional syphilis, due to a softened gumma.

[3] Arch. Gén. de Méd. 1871.

[4] Quain, Path. Trans. i. 62.

about six per cent. in the right auricle; while only two or three per cent. have occurred in the wall of the left auricle and in the septum.

The size of the rupture varies from a point scarcely recognizable to an inch in length. It may be larger on the inner surface than on the outer surface. Sometimes it is larger outside, and the inner opening may be small, and concealed among the columnæ carneæ. When the latter is the case, Blaud thought that the rupture had occurred from without inwards. The course of the rent is often oblique, so that inner and outer openings do not correspond. It is usually parallel to the muscular fibres, less frequently across them. The rupture is usually single; sometimes there are several partial ruptures, and one complete. A coagulum usually lies between the lips of the rent, and the cavity of the pericardium is usually filled with clot.

A morbid state of the heart, to which the rupture may be ascribed, is always present. In the rare cases in which fatty degeneration or softening, or other change, has not been detected, degeneration of the coronary arteries has been found when looked for, and the rupture has probably been produced by interstitial hemorrhage.

Symptoms.—Sudden, intense pain usually marks the occurrence of the rupture. The pulse becomes at once extremely weak and irregular, and soon imperceptible. There is pallor and coldness of the surface, consciousness is lost, the patient may vomit, respiration ceases, and death occurs in a few moments. Sometimes consciousness is lost before any manifestation of pain can be made. The person falls, pallid and unconscious, a few breaths are drawn, and he is dead. In seventy-one out of one hundred cases of rupture, collected by Quain, death was thus rapid. Occasionally the patient lives for several hours, even for a few days, with intense cardiac distress, and evidence of cardiac failure. The pain may extend down the left arm, sometimes down the right arm also. Vomiting is troublesome, and has been referred to the irritation of the fibres of the pneumogastric by the slowly progressing rent. It may be accompanied by diarrhœa so intense that an attack of cholera is simulated.[1] In a few cases the course of the symptoms is less regular. Some improvement takes place, and then the symptoms recur. Walshe believes these stages represent the rupture of successive layers of the cardiac wall. In a case recorded by Crisp[2] vomiting and pain for several hours were followed, before death, by the cessation of the pain. Five cases out of one hundred collected by Quain lived forty-eight hours. One lived a week. In some of the cases in which the patient lived for a few days the rupture was through the septum only.[1] A case is on record of rupture through the posterior wall in which the patient lived for six days.

When the patient lives sufficiently long to allow a physical examination of the chest to be made, the pulsations of the heart are found to be imperceptible, increase in the cardiac dulness may be recognized just before death, or for some hours previously.[2] Probably in such a case the rupture is at first small, blood escapes into the pericardium, slowly, and chiefly at the period of complete distension of the heart, and the accumulation of blood in the pericardium lessens the degree of diastolic distension of the heart, and of the escape of blood. A fall of temperature of 3½° C. was observed in Liouville's case.

Rupture of the papillary muscles and chordæ tendineæ give rise to sudden but less urgent symptoms, and are described in the article on Diseases of the Valves of the Heart.

Diagnosis.—From simple syncope, and from all forms of cerebral loss of consciousness, rupture of the heart is distinguished by the sudden, intense pain. Where pain is absent death is usually too rapid to permit diagnosis. The cessation of the pulse is peculiar to rupture, and distinguishes it from other less grave causes of cardiac pain, as angina pectoris. From rupture of an aneurism, that of the heart may be distinguished only by the rapidity of the symptoms, and by the absence of the physical signs of pericardial effusion. The pain and vomiting may cause a slow case to be mistaken for gastric disturbance;[2] the profound syncopal symptoms, and the seat of the pain should prevent such an error.

Prognosis.—Complete rupture of the heart is of necessity fatal. It is rarely, indeed, that the occasion for a prognosis presents itself. In cases of slower rupture, where the tear is at first incomplete, the prognosis is scarcely less certain. No case is on record in which a spontaneous rupture has been shown to have healed. The manner in which wounds of the heart sometimes heal suggests the possibility of a like result in rupture; but the diseased state of the walls which permitted the

[1] Land, Norsk Maga. für Laegevidsk, Bd. 23, p. 103. Virchow, Jahresb. 1870, ii. 96.
[2] Path. Trans. i. 62.

[1] De Barry, Arch. f. Klin. Med. vii. 152.
[2] Liouville, Gaz. de Paris, 1868, No. 50. Simon, Berl. Klin. Wochenschrift, 1872, No. 45.
[3] Thompson, Lancet, 1871, ii. 635.

rupture seems to preclude any attempt at cicatrization. It is just possible, however, that a partial rupture of a comparatively healthy heart, by interstitial hemorrhage, may, with careful treatment, heal. One or two cases are on record in which the symptoms of partial rupture passed away, and it is possible that they may have been of this character.

Treatment. — In complete rupture death is too speedy to permit treatment. Where the symptoms are of slower progress, and the rupture partial only, an attempt may be made to prevent its extension. The tearing force being, in all probability, due to the contractions of the heart, these must be reduced to a minimum. Absolute rest should be secured. Aconite may be given for the double purpose of thus lessening the force of the heart, and of relieving pain. No stimulants should be given. Ice should be sucked and stimulating applications to the epigastrium, such as chloroform, or spongo-piline, or camphor liniment, may lessen the vomiting and afford some relief to the pain. There is only too certain reason to fear a steady increase in the symptoms.

FIBROID DISEASE OF THE HEART.

By W. R. Gowers, M.D.

Synonyms.—Fibroid Infiltration; Fibroid Transformation (Ormerod); Connective Tissue Hypertrophy (Quain); Cirrhosis of the Heart; Induration of the Heart; Chronic Myocarditis; Schwielen des Herzfleisches.

Definition.—Fibroid disease of the heart, cardiac fibrosis, as it may be termed, consists in an increase in the interstitial connective tissue, without, or more commonly with, a secondary atrophy of the muscular fibres.

The change may affect the whole heart in slight degree, or a limited portion in a high degree. Increase in the interstitial fibrous tissue of the heart may result from chronic inflammation, and all forms of fibroid disease are, therefore, by some authorities, regarded as forms of myocarditis. But the condition may certainly arise by a slow chronic process, in which no characteristic of inflammation can be traced.

History.—Induration of the heart, when considerable, is so obvious a morbid state that it early attracted attention. It is said to have been described in 1529 by Benivenius, a Florentine physician. Senac and Morgagni noticed its occurrence. Fothergill recorded a case in which the heart was examined by John Hunter.[1] Corvisart described an example in which the ventricles sounded like horn when struck, and grated under the knife. Laennec, Bertin, Bouillaud, Hope, all described it, and the last three observers regarded it as an effect of chronic inflammation. Rokitansky described it in its relation to aneurism of the heart. Many cases have been brought before the Pathological Society, and the disease has been the subject of special study in this country by Quain[1] and Hilton Fagge,[2] in Germany by Skoda,[3] Dittrich,[4] and Skrzeczka,[5] and in France by Pelvet.[6]

Etiology.—The causes of fibroid degeneration are still little known. It is certainly more frequent in men than in women, and chiefly occurs during or after middle life. The right side of the heart is said to be affected occasionally in fœtal life. It is not commonly associated with fibroid degeneration of other organs, and it seems not specially related to the habits or conditions of life of the individual. Walshe believes, however, that it is sometimes due to chronic alcoholism. Its occurrence is chiefly influenced by local causes. Long-continued intermitting congestion of the heart causes, as Jenner[7] showed, toughening and induration of the organ, with increase in the interstitial tissue. The frequent existence of fibroid

[1] Med. Obs. and Inq. v. 1774, p. 252.

[1] Lumleian Lectures, 1872; Lancet, 1872, vol. i.

[2] Path. Trans. 1874. p. 64.

[3] Wiener Wochenschrift, 1856. Med. Zeitung, 1869.

[4] Prager Vierteljahreschrift, 1852.

[5] Virchow, Archiv, 1857, xi. 176.

[6] Des Anévrysmes du Cœur. Paris, 1867.

[7] Med.-Chir. Trans. xliii.

overgrowth in hypertrophy of the heart is probably due to the congestion which results from the cause of the hypertrophy. Local inflammation may result in fibroid change, as in the superficial layers of the heart after pericarditis. Where the fibrosis is limited in area, it has also been ascribed to an extension to the wall of adjacent endo- or pericarditis, but Hilton Fagge has suggested that the traces of inflammation which are found may be secondary to the fibroid change, and cannot be taken as proof of such an origin. Injuries, blows on the precordial region, have been assigned, in some cases, as the cause of the symptoms. Lastly, there is clear evidence that syphilis is capable of causing local indurations of the heart; most probably by the transformation into fibrous tissue, of gummatous growths.

Pathological Anatomy.—The slighter diffused form of fibrosis may affect the whole heart, or only one chamber. The intenser form is usually limited to a portion of one chamber. Occasionally a high degree of fibroid growth may extend around the heart, and has been described as a true "stenosis of the heart."[1] In the fibroid change secondary to pericarditis the outer layers of the heart are most affected, and sometimes one-half of the thickness of the wall may be transformed into fibrous tissue.

The local forms of fibrosis affect the papillary muscles more frequently than any other parts. These may be entirely transformed into fibrous tissue of tendinous aspect. More rarely the wall of the heart is the seat of circumscribed changes, especially the neighborhood of the apex. They are also found in the septum, and in the posterior wall at the base. In the right ventricle the degeneration is usually near the base. The local forms are commonly most marked towards the inner surface of the wall. If the whole thickness of the wall is affected, it is rendered thinner, even apart from aneurismal bulging. The endocardium over the degeneration is often thickened.

The diffuse fibrosis renders the wall of the whole heart tougher, more resistant to the fingers and knife. Sometimes, when the new tissue is soft, and the muscular fibres are degenerated, the consistence may not be increased, may even be diminished. The change may alter very little the naked-eye appearances, or the enlarged intermuscular septa may be visible in the cut section. The localized change usually presents a glistening fibrous appearance, gray or white, sometimes of a greenish or bluish tint. The section may have a spongy appearance. (Hilton Fagge.) Where less advanced, whitish bands and tracts of fibrous tissues may be seen in the muscular substance. In some cases several separate areas are affected. Occasionally calcareous deposits have been found in the changed tissue.

Under the microscope the localized forms present well-developed fibrous tissue with nuclei. In more recent cases a fusiform cell-growth has been found, developing into fibres. It is said to begin around the bloodvessels, in the intermuscular septa, with an infiltration of nuclei and leucocyte-like cells. Sometimes, it is said, the new substance appears very obscurely fibrillated or amorphous, and may undergo fatty degeneration. Pelvet has seen much elastic tissue in some specimens. Through the fibrous tissue the muscular fibres may be seen passing, lessened in number, sometimes narrowed by pressure, or the seat of fatty degeneration. It is rare for them to disappear entirely. Occasionally the degeneration is in excess of the development of fibrous tissue, and the affected area softens.

Consequences.—The effect of fibrosis on the form and size of the heart varies. Hypertrophy and dilatation usually coexist with the diffuse change, and sometimes the overgrowth of the muscular and fibrous tissues, advancing *pari passu*, may enlarge the heart to vast dimensions, as in the specimen preserved in St. George's Hospital and described by Quain.[1] The increase in the fibrous tissue which results from congestion is commonly greater in the right ventricle than the left. Localized fibrosis also often occurs in hypertrophied hearts, although it may be found in hearts which are normal in size. The cavity may present little change, or it may be generally dilated. More commonly the wall of the affected spot is bulged out into an aneurism. (See Aneurism of the Heart.)

Symptoms. — The necessary effect of fibrosis of the heart will be, as Corvisart clearly taught, to lessen its contractile power. The diffused form, therefore, promotes dilatation or lessens the effects of the hypertrophy which it accompanies. The symptoms of the localized form, in marked cases, have commonly been those of cardiac weakness. Dyspnœa and dropsy have been the chief troubles, and in their general character the symptoms resemble those of dilatation of the heart. The impulse is weakened.[2] The first sound is

[1] Dittrich, loc. cit.

[1] Lumleian Lectures, loc. cit.

[2] Laennec taught that induration increases the firmness of the heart's contraction; but this was probably a hasty conclusion from the firmness of hypertrophied and strongly-contracted hearts.

weak and toneless, and it has been noticed to be much weaker over the left than over the right ventricle, when the former was most affected. A systolic murmur has been present in many cases, due, in some, to regurgitation from fibrosis of papillary muscles. The pulse is weak, and has been, in some cases, very infrequent; only thirty beats per minute have been noted. Cardiac pain is present in a considerable number of the cases. In many instances, however, the symptoms have been entirely latent. These differences depend no doubt partly on the extent and position of the fibrosis, affecting the action of the rest of the muscular tissue more in some cases than in others. Death in many instances has been sudden, apart from the rupture, or even the existence of an aneurism.

Diagnosis. — Fibroid degeneration of the heart is at present hardly more than a pathological curiosity, for it is doubtful whether it has ever been recognized during life. Its detection, apart from the signs of aneurism of the heart, must depend on the symptoms of cardiac dilatation without its physical signs.

Treatment.—The treatment needed is that for cardiac weakness—for the dilatation which it resembles in its effects. Rest, the avoidance of all strain on the circulation, and the administration of digitalis, to strengthen the remaining fibres, are the chief measures. If there is any suspicion of syphilis, iodide of potassium should be given, although it is doubtful whether the stage of induration can be modified by that drug.

B. Associated Organic Changes.

Mediastinal Tumors.

ON MEDIASTINAL TUMORS.

By R. Douglas Powell, M.D., F.R.C.P.

The Mediastinum is that central space situated behind the sternum and between the pleuræ which is occupied by the heart and the great vessels connected with it, the trachea and its main divisions, the pneumogastric and phrenic nerves, and the thymus and bronchial glands.

The lungs with their pleural coverings are closely applied on either side of this region, their anterior margins overlapping it in front to an unequal extent on the two sides, so that in health the dulness on percussion that the solid contents of the mediastinum would naturally yield is in a great measure obscured, and permitted to become apparent only at the upper part of the sternum and over the triangular area of the heart's dulness. In emphysema of the lungs the mediastinum may be yet more completely covered up; in other pulmonary affections attended with diminution of bulk, it may become uncovered on one side or on both, giving rise to some singular, and at times perplexing, distortions of the mediastinal dulness without any corresponding disease within the space itself. The division of the interpleural space into anterior and posterior mediastinum is entirely arbitrary, the former term being loosely applied to that portion of the space in front of the trachea and the roots of the lungs, the latter to that portion situated behind this plane. The mediastinum is in health altogether obscured to us from behind by the spine and the thick posterior margins of the lungs closely in contact with it.

Our present concern being only with morbid growths affecting the mediastinum, we shall refer to such other diseases as aneurism, abscess, pericardial effusion, &c.,—only in so far as they affect diagnosis.

Varieties, Etiology.—Carcinoma,[1] sarcoma, and lymphoma are the three varieties of morbid growth which may give rise to tumor in the mediastinum. Since the time, only a few years ago, when attention in this country began to be more closely directed to the finer distinctions, based on anatomical structure, which separate true cancers from other tumors, and these from one another—an inquiry first entered upon by Professor Virchow, and largely stimulated and promoted by the labors of the Morbid Growths Committee of the Pathological Society of London—the rarity of primary carcinoma of the mediastinum has become more evident, and a large proportion of the cases that only a short time ago were designated scirrho-encephaloid, or scirrhous, or soft cancer, would now be classed amongst the sarcomata or the lymphomas. Still, cases of true cancer primarily affecting the deep-seated parts of the mediastinum do now and again occur; *e. g.*, one such case is recorded by Dr. C. T. Williams in the Pathological Transactions, vol. xxiv., its cancerous nature being confirmed, on minute examination, by Mr. Arnott. Even as a secondary growth, cancer rarely affects the mediastinum, save when, as, however, not uncommonly happens, it directly penetrates through the chest wall from a cancerous breast.

Sarcoma, too, as a primary disease, is most rare in this situation; when it occurs it usually arises secondarily to some similar or associated growth situated

[1] Throughout this article the terms "carcinoma" and "true cancer" are applied to that form of new growth whose typical structure is met with in the scirrhous breast.

elsewhere. Nor is it always clear how a secondary growth arises in the mediastinum; its path of transmission is not in all cases evident. We cannot always obtain evidence of a lymphatic connection between the primary and secondary growths, nor, on the other hand, is it easy to see how a disease germ could be conveyed from a distant part to the mediastinum through the circulation without first involving the lung; and in view of this difficulty, it is worthy of remark how frequently the disease, although mainly mediastinal, involves also one lung to a greater or less degree. It is very possible that, in some of these cases, the secondary growth may have really been conveyed to some portion of the lung or pleura first, and formed a small nodule there; but that, the bronchial glands becoming early infected, the disease in them proceeds with such great rapidity as soon to outstrip and obscure its pulmonary origin, and to give the case the clinical and even *post-mortem* features of primary mediastinal disease subsequently involving the lung. This explanation at least occurred to me as best accounting for a case of osteosarcoma of the mediastinum and lung, occurring subsequently to the removal of a shreddy sarcomatous growth from the knee-joint.[1] There will always remain, of course, the possibility of the disease of the mediastinum, though secondary in point of time, being due to a recurrence or a continuance of the same constitutional dyscrasia which led to the production of the first. Experience has, however, of late years, gone against the validity of such an hypothesis.

Of morbid growths affecting primarily the mediastinum, *lymphoma* or *lymphosarcoma*, or, as it is sometimes designated, *lymphadenoma*, is by far the most common. It was Dr. Murchison who first recognized lymphadenoma as a distinct variety of morbid growth, in a case of disease affecting the intestines, liver, mesentery and heart, &c., which he brought before the Pathological Society of London in November, 1868,[2] and of the minute character of which an ample report is appended to his description by the Morbid Growths Committee. In the same volume, p. 102, Dr. Church has also described a case of "Carcinoma of the pericardium, anterior mediastinum, and lymphatic glands, in the thorax and abdomen," which he recognized as different in minute structure from true cancer, and regarded as more correctly "ranked among the mediastinal sarcomatous tumors mentioned by Virchow[3] as approaching so closely to the structure of lymphatic glands as to be with difficulty separable from them." In the next volume (xxi. of the Transactions, p. 358), is recorded as such by myself the first case of lymphosarcoma or lymphadenoma of the mediastinum, although there are many cases in earlier volumes related as instances of cancer which would be undoubtedly more correctly included under the newer term. In subsequent volumes examples of the disease are given by Drs. Murchison, Bennett, Payne, Dickinson, and others.

In most cases, the growth originates in the lymphatic glands, either in the anterior mediastinum or at the root of the lung, the connective tissue surrounding the glands becoming quickly implicated. In a case, however, reported by Dr. Church, and in another by myself, the thymus gland appeared to have been the original seat of the disease. The growth invades other tissues, the neighboring glands, the lungs, the heart, and even the vessels. It does not, however, incorporate to itself with the same avidity as cancer does all the tissues with which it comes in contact, but prefers to creep along the bronchial or vascular sheaths, and to involve organs more slowly, guided by the lymphatic paths into their interior. The calibre of large bronchi, veins, or even the auricles of the heart, may, however, be invaded by flattened projections of this growth, which by the unaided eye could not be distinguished from cancer. As a local disease, then, lymphoma in this situation is decidedly malignant, but in an intensity rarely as great, and sometimes much less, than that of cancer. It is sometimes, however, a part of a more general disease, affecting more or less the whole glandular system, and in one remarkable case, which was for several months under my observation both as an out-patient and in the wards of the Brompton Hospital, and which subsequently terminated fatally in the Middlesex Hospital under the care of Dr. Murchison,[1] the disease, mainly affecting the glands of the neck, mediastinum, and axilla, and the spleen, was marked by periodical attacks of fever, accompanied by intumescence of all the affected glands and of the spleen, which gave to it an altogether peculiar character. After death, almost every organ was found disseminated with lymphatic growths identical in structure

[1] Path. Trans. vol. xxiv. p. 28.

[2] Vide vol. xx. p. 192.

[3] Die Krankhaften Geschwülste, Band ii. p. 376.

[1] A description of the clinical characters presented by this case while in the Middlesex Hospital, with an account of the autopsy and an admirably summary of the literature of the subject, is given by Dr. Murchison in the Path. Trans. vol. xxi. p. 372, and to it is appended an account of the microscopical examination of the diseased structure by Dr. Sanderson.

with those already described. In this case then was seen exemplified the highest degree of malignancy conceivable, only comparable to that of disseminated cancer or miliary tuberculosis.

Age.—Growths in the mediastinum may be met with at almost any period of life, but they are more prevalent before the middle period; and it is useful to remember this fact, since, if we meet with a case of mediastinal tumor of doubtful nature in a patient under the age of 25, it is more likely to be malignant than aneurismal, this probability being increased as the age is earlier. Of six cases which have fallen under my personal observation, all but one were under 30—viz., one at the age of 6, two at 20, one at 27, and one at 29; the sixth case being aged 49. Of seven cases specially referred to by Dr. Bennett as mediastinal, two occurred at the age of 11, one at 17, one at 20, one at 23, one at 40, and one at 60. So that if we might fairly strike an average from such limited numbers we should get 24·8 as the mean age for the occurrence of this disease.

Sex.—As regards *Sex*, five out of Dr. Bennett's seven cases quoted were females, and five of my six cases were also females. On the other hand, however, of six cases specially referred to by Dr. Symes Thompson in a pamphlet on Mediastinal Tumors, published in 1865, all were of the male sex. We must, then, for the present, say that these growths may occur in either sex, with perhaps a slight preponderance in favor of the female sex. When a mediastinal growth invades the lung secondarily, it appears to have a special but not exclusive preference for the left lung, the other lung as a rule wholly escaping. In four of Dr. Bennett's cases one lung was invaded, and in each instance the left. Of the three of my cases in which one lung was invaded, it was in two instances on the left side.

Symptoms.—The *Symptoms* of mediastinal tumors are little influenced by the kind of growth (and the same remark applies to physical signs), they are due to compression or obliteration of vessels and nerves, of the air-tubes or œsophagus, or to the invasion of the heart or the lungs or other structures on the confines of the space. They therefore closely resemble those presented by aneurismal tumors, and the diagnosis between the two is often perplexing.

In cases of tumor, deep-seated *pain in the chest* or back is not so prominent a symptom as in aneurism; there is often no pain experienced until the growth approaches the surface, when it assumes the pleuritic character. The characteristic stabbing pain of cancer is occasionally complained of. If it is borne in mind that malignant growths, cancer, lymphoma, sarcoma, differ from aneurismal tumors in two important respects—viz. (*a*) they tend to incorporate to themselves the structures they encroach upon, thus invading and replacing more, and compressing and displacing less than aneurism; and (*b*) a change in the *direction* of expansion, so common in aneurism, is much less so in them, and is not attended with that relief to symptoms dependent on local removal of pressure—it will be more readily understood why the pressure symptoms should *as a rule* be more insidious, yet when present more constant and persistent with them than with aneurism.

The *Dyspnœa* depends upon the size and seat of the tumor, and increases day by day with its growth; but severe paroxysmal attacks of dyspnœa are occasionally witnessed in tumor as in aneurism. These paroxysms are usually due to direct pressure upon the trachea or a main bronchus, and are more frequently observed among the later symptoms of the disease. *Cough*, dry, ineffectual, or attended with only scanty mucous or frothy expectoration, is an early and very constant symptom, and, together with a certain sense of constriction or pain about the sternum, constitutes the complaint for which the patient usually first seeks advice. The cough may have a clanging laryngeal character, and may be attended with *huskiness of voice* or *aphonia*. These symptoms are, however, less frequent than in aneurism. *Sanguineous expectoration* may be present, and is a sign that the tumor has invaded the lung. Profuse *hæmoptysis* is rare, but has been met with as an early symptom. In the later stages of the disease profuse hæmoptysis sometimes occurs, and is followed by marked relief to pressure symptoms; it then becomes an important sign of tumor. In a large proportion of cases, however, no hæmoptysis occurs throughout the disease. *Dysphagia* is in mediastinal growth a far more common and prominent symptom than in aneurism. It is more constant when present, although, as in the case of dyspnœa, it too may be increased by paroxysms, more especially in the earlier stages of the disease.

Physical Signs.—At the time of seeking advice patients with mediastinal tumor are not as a rule emaciated; they often indeed appear to be well nourished, although on inquiry it will be invariably found that they have of late lost flesh; nor do they ever evince as the disease progresses that degree of emaciation so commonly seen in chronic phthisis, save in those cases in which the œsophagus is involved or pressed upon. The face is

usually pale, with often some lividity of lips, and in most, perhaps in all instances, there is a certain anxiety of expression, a slight contraction of the brow, giving an aspect of distress which is often sufficient to mark off the case as one not of ordinary chest disease. A slight staring of the eyes, with noticeable puffiness of face, commonly present, may, as the disease advances, be intensified into the aspect of semi-strangulation, characteristic of a tumor pressing upon the great veins. The *temperature*, unless there should be some inflammatory complication, is not raised. In the exceptional case already referred to the periodical attacks of fever were but phases in the progress of a general disease affecting the whole glandular system.

In further considering the physical signs of mediastinal tumor it must be remembered that, as has already been incidentally remarked, these growths very commonly involve sooner or later one of the lungs. The lung thus secondarily affected is most frequently, but not always, the left.

On inspecting the chest, some alteration in shape is frequently to be observed. The upper sternal region may be unduly prominent, or one side of the chest may be both to eye and measurement larger than the other, the enlargement being perhaps more decided above than below the nipple level. The side, however, which yields most evidence of disease is not always the larger, it may be smaller, to measurement; and this negative sign, taken with other positive ones, *e. g.*, displacement of heart, would be very significant of tumor. Cases have been observed by Dr. Pollock, Mr. Holmes, and others, in which the tumor has projected through the sternum and cartilages. Some enlarged glands at the root of the neck, or in the axilla, mobile in adenoma, fixed in cancer, may give us a clue to the nature of the disease. The superficial veins of the chest are frequently found distended, more frequently and more decidedly so than in aneurism. They may be more distended on one side of the median line than on the other, and one upper extremity may show venous obstruction and œdema.

Displacement of heart is one of the most important signs of intra-thoracic tumor; it is a result of direct pressure, and its direction is, generally speaking, determined by, and is an important index of, the seat of the tumor. The growth may occupy the upper part of the anterior mediastinum, and extend downward in front of the pericardium, covering it with a thick, solid apron, or, growing from behind, it may push forward the heart against the sternum; or again, encroaching forwards from the root of the lung (a common site) between it and the pericardium, it may press aside the heart. We should endeavor, therefore, in all cases, by palpation and auscultation, to ascertain the exact and relative position of both the apex and base of the heart. In certain rare cases—one such came under my notice in Dr. Cotton's wards, at the Brompton Hospital, in 1866—the heart is fixed *in situ* by the growth extending on both sides of it. The mediastinal growth may extend downwards between the lung and heart to the diaphragm, forming a large mass between it and the base of the lung. Some downward displacement of the *liver* or *stomach*, with hardness and bulging of the hypochondrium, are then to be observed.

Increased *dulness* on percussion is an essential sign of mediastinal tumor. It may amount to only a patch of lessened resonance at one sterno-clavicular angle or in one interscapular space, or there may be three or four square inches of dulness over the upper sternum continuous below with that of the heart area. Again, the toneless percussion may and often does extend beyond the confines of the mediastinum, so as to include the whole or a greater part of one side; in consequence of the growths involving by direct invasion, or indirectly, by the destruction of its main bronchus, the lung on one side. Several questions in diagnosis arise from this fact, and it is curious how invariably the disease in such cases is mainly one-sided, and how frequently the other lung altogether escapes. Supposing the dulness to be thus extended, its *quality* and *distribution* become matters of great importance in diagnosis. Its quality is essentially hard and resisting—often unequally so at different parts, so as to have a lumpy character, being more toneless and resisting over small scattered areas than in the intervening parts. Above the clavicle, in the outer scapular region, and at the acromial and axillary regions of the chest, a resonant note may still be obtained—in fact, a mediastinal growth, when it invades a lung, almost invariably does so from its hilus, extending outwards so as to occupy the whole middle part of the lung, coming to the surface in rounded prominences and leaving the remoter "corners" of the thorax, so to speak—the summit, humeral, and scapular regions, and the base—to be last involved. It will be remembered that in pleural effusion the dulness advances steadily from below upwards, the circumferential parts of the chest first becoming toneless, the central—sternal and interscapular regions—being last affected. Displacement of heart away from the affected side is common to both conditions. If the base of the lung, however, becomes the seat of secondary pneumonia,

or is collapsed by some attendant effusion, the validity of the contrast just drawn is somewhat obscured.

On auscultation the heart's sounds are found to be unduly conducted over the dull region in front and too audible in the interscapular region behind. When the tumor is mainly seated in the anterior mediastinum, the conduction of the heart's sounds may be intense in this region, and may be attended with an impulse distinctly appreciable to the ear. The *impulse* is, however, in such cases knocking, not expansile; but it sometimes closely resembles that yielded by an aneurismal sac thickly lined by coagulum. A systolic *murmur* is sometimes audible over some portion of the dull region; it has, however, the simple, short, blowing character distinct from the rasping or expansive bruit which would most likely accompany an aneurism of similar superficial dimensions. In cases of tumor growing from the posterior mediastinum, the heart pressed against the sternum may yield *to the ear* a very peculiar sensation, analogous to that experienced by the hand when laid upon a struggling bird. In an obscure case which came under my notice three years ago, the presence of this sign, together with displacement of the apex towards the ensiform cartilage, enabled me correctly to surmise the nature of the case before any other positive sign could be detected. The pericardium and the heart, more particularly one of the auricles, very often become involved in the growth, and a *cardiac murmur* or *friction sound* may thus be given rise to.

The *respiration* is commonly bronchial over the tumor, or it may have a stridulous or sibilant character. Stridor is less common than in aneurism, and for the reason before named, that growths tend to occlude rather than compress the tubes with which they come in contact. The observation of very well marked stridor has so often led me carefully to examine for a tumor which has proved not to exist, that the sign has, for me at least, lost much of the value often ascribed to it. Still it would not be safe to disregard it, especially when localized at one part of the chest. When the growth, however, occupies the anterior mediastinum, and is of considerable thickness, no respiratory sound may be audible over it, more or less bronchial respiration and râles being heard in the outer subclavicular regions. It is indeed in parts of the chest distant from the tumor in the mediastinum that we often get auscultatory signs most suggestive of its presence, *e. g.* there may be observed at one base marked feebleness of respiration, amounting to a mere muscular struggle without any accompanying respiratory sound, yet the percussion dulness is perhaps little if at all impaired, quite insufficiently so for effusion; there is no ægophony, the vocal resonance is diminished, or altogether annulled. On the opposite side the respiratory murmur is normal or exaggerated. Here is a grouping of signs very confirmatory of a tumor compressing or obliterating a main bronchus.

When the dulness extends from the mediastinum over one side of the chest, it is accompanied by enfeebled or even annulled respiration, and we may have many of the signs most significant of fluid effusion into the pleura—displacement of heart dulness, absence of respiration, and even of vocal fremitus, with enlargement of the side. The diagnosis between the two is only made with extreme difficulty, and without puncture of the chest, is often indeed impossible. In this dilemma, however, attentive auscultation will often discover here and there over the affected side a slight grating *friction sound*, a sign which becomes of the greatest importance, showing the pleural surfaces still to be in apposition.

Moist rhonchi—mucous or gurgling râles—are never to be heard in any abundance over the dull region, as would be almost inevitably the case in any similar extent of consolidation from scrofulous disease.

This remark is not the less true although after death we do occasionally find softened patches in a lung which has become invaded by a growth, the softening having as a rule been preceded by obliteration of the bronchi leading to them, so that they yield no sign during life. Indeed, on making a section of a lung whose root has been invaded by a tumor, we frequently find a striking appearance as of multiple abscesses dispersed through the organ, and the condition has been repeatedly so described. In truth, however, these "abscesses" are generally nothing more than bronchial tubes which have become enormously distended with secretion in consequence of obstruction at the main bronchus, collapse and slow inflammation of the surrounding pulmonary tissue being also present. The distended tubes, with their opaque contents, shine through the pleural surface of the lung as yellow spots, giving to it a remarkable appearance. No doubt some of the alveoli become filled and yield before the accumulating pressure of the bronchial secretion. Sir George Burrows many years ago related, to the Medico-Chirurgical Society, a case of carcinoma of the lungs in which the right bronchus was obstructed, and some bronchial tubes in different parts of the lower lobe "when cut across were found distended with thick, yellow, tenacious pus, giving the appearance of small abscesses." A typical case of the kind, too, is described by my col-

league, Dr. C. T. Williams, in the twenty-fourth volume of the Pathological Transactions, and I have seen other instances, the most remarkable one being from a case of aneurism compressing the left bronchus, of which I made the autopsy in July, 1870. An extract from my note-book states as follows :—"Left lung very large, consolidated throughout. Bronchial tubes much congested and dilated, many presenting terminal dilatations, filled with muco-purulent secretion. Lung studded throughout with nodules of yellow pneumonia, having for their centres bronchial tubes which exude their secretion on pressure. Surrounding these nodules the tissue is in a condition of gelatinous pneumonia, so that the total result is a solid lung. Some of the broncho-pneumonic centres have broken down into small cavities filled with muco-purulent fluid. Right lung healthy, but in a state of active congestion.

Dr. Budd[1] regards the secondary inflammatory changes—thickening and adhesion of pleura, and inflammatory destruction of lung—that occur in a lung whose root is invaded by cancer as due, not to irritation of the invading new growth, for cancer *per se* has little tendency directly to cause inflammation of the surrounding parts, nor for the most part to obstruction of veins and arteries (pulmonary and bronchial) which might cause gangrene or atrophy of the lung, but he thinks that these changes result "from the tumor involving and destroying all or a great part of the nerves with which the several tissues are furnished." In one of his cases Dr. Budd regards the pericarditis present as due to the same cause. I have seen one case to which this explanation might very well apply. It was one that occurred at the Brompton Hospital under the care of Dr. Pollock in 1868, in which there was found after death a tumor invading the left lung from its root to about one-third of its extent, the rest of the lung being shrunken from inflammatory softening; encroaching upon the summit and also upon the base were found two pleural cavities occupied by purulent fluid: the left main bronchus was almost, but not quite, obliterated by the growth. It is readily conceivable that partial destruction of the pulmonary nerves may, by lowering the vitality of the lung, render it more liable to the occurrence of inflammatory changes, and that complete destruction of these nerves at their origin might directly induce inflammatory destruction of the organ. In the main, however, I should feel disposed to regard the mechanical obstruction at the main bronchus as being, in most cases, sufficient to account for such secondary lesions as we find. In some of the cases which I have quoted the secretion from the bronchial membranes went on with a vigor undiminished by any impairment of nervous influence, and the pneumonic changes present were, apparently, directly due to obstruction to the escape of this secretion. It is very possible that pressure upon the nerves may give rise to muscular paralysis of the bronchial tubes, as suggested by Dr. Bennett, without affecting their secreting power.

[1] "On some of the effects of Primary Cancerous Tumors within the Chest." Med.-Chir. Trans. vol. xlii. 1859.

DIAGNOSIS.—In relating the symptoms and signs of mediastinal tumors in the present chapter and in the succeeding section on aneurism, much has been said incidentally respecting the diagnosis of these tumors from other diseases. We have still, however, to summarize and discuss the most important difficulties that may arise in the way of diagnosis. These difficulties vary somewhat, according as the disease is (*a*) purely mediastinal, or (*b*) involves the lungs secondarily.

(*a*) Growths which are purely or mainly mediastinal may closely simulate *aneurism*. The following are the chief considerations which would tend to decide the question in favor of the tumor being a morbid growth :—

The age of the patient being under 25.

The presence or history of tumors elsewhere.

The absence of marked disease of the vessels.

The absence of characteristic pulsation or bruit, especially when combined with

The presence of local venous engorgement, and

Extensive area of superficial dulness.

"Extensive area of dulness must in aneurism mean a large sac, and with such a large tumor we should almost inevitably get marked expansile pulsation. Again, aneurismal sacs, before they produce extensive dulness of any portion of the parietes of the chest, point, as it were, in some particular direction, becoming distinctly prominent and producing an eccentric motion around them in consequence of the thoracic parietes being absorbed, or yielding at the point of greatest pressure."[1]

In the early stages, however, of the disease, it may be extremely difficult, nay, impossible, to make the diagnosis with certainty. We must duly consider and weigh the probabilities in each case, there being no further rules of sufficient general value to be worthy of mention here.

[1] Graves' Clinical Lectures, 1848.

Mediastinal abscess is very rare, at least of such dimensions as to simulate tumor. Such an affection would usually be accompanied with scrofulous abscesses elsewhere, and probably with hectic symptoms.

Sub-sternal thickening may cause many of the signs of tumor, particularly when associated, as in one case which has been for some time under my notice, with œsophageal spasm. The effect of treatment (antisyphilitic) upon such cases soon discovers their real nature.

A variety of chronic pericarditis has been described by Prof. Kussmaul[1] under the name of *callous mediastino-pericarditis*, in which the pericardium becomes greatly thickened and its cavity completely obliterated by the tough products of a chronic inflammation, which, moreover, extends to the cellular tissue of the mediastinum, indurating it and surrounding the great vessels with a contractile tissue which constricts and distorts them. The increased mediastinal dulness and other signs attendant upon this condition closely simulate those of tumor, but Kussmaul states that there are two signs which are characteristic of this lesion,—viz., a complete, or almost complete, failure of the radial pulse during inspiration, and, simultaneously, visible swelling of the great veins of the neck instead of the collapse that usually takes place during this portion of the respiratory act. Adhesion of the great vessels to the sternum, either directly or through the medium of the pericardium, is supposed to account for these phenomena.

Certain cases of *phthisis*, in which chronic disease at one apex has led to exposure of the mediastinum from retraction of the margin of the lung on one side, may be mistaken for mediastinal disease. Such cases, however, especially some cases of so-called senile phthisis, are more apt to be confounded with cancer of the lung or with aneurism. They have already been referred to in the previous chapter.

(*b*) A mediastinal growth secondarily invading the lung on one side may present more difficulties in the way of diagnosis than one more strictly confined to its original site. Deeply seated, invading the lung at its hilus, and, as it were, functionally choking it, such a tumor may simulate *chronic pneumonia*, local or general *empyema*, or *aneurism* of the descending aorta.

The close resemblance between certain cases of mediastinal growth spreading through a lung and effusion into the pleura has already been referred to. The signs in favor of tumor may be thus summarized :—

1. An unconformity of increased measurements to those which would be occasioned by fluid accumulation.

2. The presence of large tortuous veins and œdema of upper extremities or head.

3. Dulness marked at the mediastinal region becoming less uniform in tone and firmness at the circumferential parts of the chest, where patches of resonance may be found which could hardly coexist with fluid.

4. The loud transmission of the heart's sounds (Walshe).

5. The detection of pleuritic friction-sound over parts dull on percussion. (It must be remembered that there may be some effusion which has supervened upon the mediastinal disease, and that this effusion may be general or limited.)

6. Hæmoptysis or "currant-jelly" expectoration would negative the disease being one simply of effusion.

7. The presence of the signs of pressure upon central parts (Walshe).

This last-named consideration is certainly of value, but it may mislead ; *e. g.*, I have myself seen two instances in which considerable effusion into the pleura has been attended with that peculiar laryngeal cough and husky voice which I regarded as significant of tumor in addition to the effusion, and which in one case led others of great experience also into the same error. These signs both, however, disappeared after the removal of a large quantity of purulent fluid. Another pressure sign I have seen exemplified in a case of simple effusion, which might suggest some associated mediastinal tumor, though more probably of aneurismal than malignant kind, viz., increased size, tortuosity, and throbbing of the radial and brachial arteries on the *affected* side. The case was under the care of my colleague, Dr. Tatham, and the best conclusion we could come to was that the phenomenon was due to hardening and hypertrophy of the vessels from increased resistance to circulation through them, in consequence of impediment to venous return from the limb. There was no œdema of the limb, however, and after the removal of the fluid the thickening remained ; no sign or symptom of tumor has since occurred.

8. In all cases of doubt, and when the dyspnœa is at all urgent, an exploratory trocar should be inserted.

Cases of mediastinal growth invading the lung from its root have often been mistaken for *chronic pneumonia*.

Dr. Walshe lays stress upon the following signs as distinguishing tumors from chronic exudative pneumonia :—[1]

[1] Berliner klinische Wochenschrift, 1873, Nos. 37, 38, and 39. An abstract of these papers is given by Dr. M. Bruce in the Medical Record for December 17th, 1873.

[1] Diseases of the Lungs, 4th Edition.

1. A tendency to increase instead of diminution of bulk of the affected side.

2. Implication of the mediastinum.

3. The more serious change in the results of percussion.

4. Emaciation is of earlier appearance and more marked in chronic pneumonia than in tumor.

5. Dyspnœa out of proportion to extent of consolidation favors the diagnosis of tumor.

These five signs would be of equal value in distinguishing between chronic pneumonia in its less restricted sense of chronic inflammatory consolidation of the lung affecting the lower lobe, and mediastinal growths secondarily affecting the lung. Dr. Walshe gives three further distinctions, viz. :—

6. The failure or disappearance of vocal fremitus, which remains in chronic pneumonia ;

7. The different characters of respiration in the two diseases ;

8. The presence of hæmoptysis and red jelly-like expectoration which never occur in chronic pneumonia. These do not, however, help us in eliminating those chronic basic consolidations which do not clear up, and which are therefore most apt to come before us for diagnosis from tumor. I have for instance so repeatedly seen cases of chronic pneumonia in which from great thickening of the adherent pleura the vocal fremitus has been much deadened or almost annulled, that I cannot but regard the distinction No. 6 as apt to mislead. The respiration, too, in such cases is remarkably feeble. The diagnosis would, under such circumstances, be cleared up in favor of tumor by finding with these signs tolerably limited to the lower lobe the *heart displaced towards the opposite side.*

I have had also under my observation for some years a patient with consolidation of the base of the right lung who has had decided hæmoptysis on several occasions, and two years ago he expectorated a currant-jelly-like sputa more or less continuously for nearly three months, which induced me several times to seek carefully for signs of malignant disease, but without result ; and the patient is now greatly better, all symptoms being in abeyance. My own experience would indeed lead me to say that hæmoptysis is not very uncommon in chronic basic pneumonia.

In the diagnosis of mediastinal growth from *aneurism* compressing the root of the lung, and setting up in it secondary inflammatory disease, we must have regard to the distinctions to be laid down between tumor and aneurism. Hæmoptysis would by no means necessarily decide the question in favor of tumor.

In endeavoring to come to a conclusion as to the *nature of the morbid growth* which we have ascertained to be present in the mediastinum, an inquiry so far as we at present know of no great practical importance, we may bear a few general facts in mind.

1. If the disease be primary in the mediastinum, it will be almost certainly lymphoma.

2. The younger the patient the more likely is it to be of this nature.

3. The presence of enlarged movable glands in the neck, or in other parts of the body (which may suppurate, but do not ulcerate) are also favorable to the diagnosis of lymphoma.

4. If the disease be secondary to a growth elsewhere, it will be of the same nature as the primary disease, or allied to it within the range of pathological variation, *e. g.* If the disease makes its appearance subsequently to a limb having been removed, or a joint resected for a malignant growth, we may feel confident that it is one of the sarcomata, either soft oval or spindle cell, or osteo-sarcoma, or enchondroma. If a fixed, nodulated, hard tumor were present in the neck or had been removed from the breast, we might expect the disease in the mediastinum to be cancerous.

5. So far as invasion of the lung goes, this feature is common to all these growths.

PROGNOSIS.—The prognosis is unfortunately in all these cases at present equally fatal. But we cannot say whether in the case of lymphoma some remedy may not hereafter be found to exercise some control over the growth. The duration varies according to the parts involved by the tumor, it is rarely greater than a few months.

TREATMENT.—There is no treatment to be adopted in these diseases, save to combat so far as is possible such symptoms as pain, restlessness, anæmia, &c. By attending to these points and to the digestion, by local depletions when indicated, or in the case of complication with hydrothorax, by tapping, the lives of the unfortunate sufferers from these dire maladies may be certainly prolonged and rendered more endurable.

C. DISEASES OF THE VESSELS.

AORTA. PULMONARY ARTERY. CORONARY ARTERIES. SYSTEMIC ARTERIES. VEINS. CARDIAC CONCRETIONS. LYMPHATICS.

THE DISEASES OF THE AORTA.

BY R. DOUGLAS POWELL, M.D., F.R.C.P.

IN the ensuing articles, the term "thoracic aorta" will be used inclusively as applying to that portion of the main arterial trunk which is contained within the thorax, having its origin at the left ventricle of the heart, and escaping through the diaphragm to become continuous with the abdominal aorta at the level of the last dorsal vertebra. In its passage from the ventricle to the left side of the third dorsal vertebra, the aorta describes a somewhat twisted curve, and this *arch* of the aorta is divided for convenience of anatomical description into an ascending, a transverse, and a descending portion. That portion of the vessel extending between the third dorsal vertebra and the diaphragm, to which the term thoracic vertebra is sometimes exclusively applied, is better described as the descending thoracic aorta.

The function of the aorta—that of receiving and distributing, in more equable currents, and with the least possible conversion of motive force, the blood impelled into it with each systole of the left ventricle—is mainly performed by the arch, and for the most part mechanically, by virtue of its being a curved elastic tube furnished with appropriate valves. But the anatomical structure of its walls, the organic muscular fibre they contain, and the phenomena occasionally witnessed in disease, forbid our regarding the whole function of the aorta as being quite so simply discharged. We have organic muscular fibre nowhere else in the body save where the property of muscle is appreciably exercised, at least to that ill-defined extent which we characterize by the term *tonicity*. It is by the local deprivation of this unobtrusive form of muscular activity through nervous agency that we may most reasonably explain those violent aortic pulsations which are occasionally met with in nervous people; and no doubt mental depression and anxiety exert largely through this means what influence they have in predisposing to aortic aneurism.

AORTITIS.

Acute inflammation of the aorta, in the sense of an acute exudative inflammation, is a disease of very doubtful, if not impossible, occurrence. Professor Lebert,[1] after detailing the symptoms that have been ascribed to the disease by Frank, Bizot, and others, confessed that, in the course of twenty years' experience, he has not seen one case, either clinically or anatomically, corresponding to it. Professor Rindfleisch[2] observes that, "apart from thrombotic arteritis and phlebitis, there is hardly such a thing as acute inflammation of the walls of the vessels," save, as he proceeds to explain, in so far as their external coats, which must be regarded as part of the general connective tissue, may partake in any contiguous inflammation. But even in this partial

[1] Virchow's Handbuch der spec. Path., Krankheiten der Blut- und Lymph-gefässe, 1855.

[2] Pathological Histology, vol. i. p. 249, Dr. Baxter's translation for Sydenham Society, 1872.

manner the aorta is very slow to share in such processes, and when it does so the inflammation is very chronic and limited, giving rise to no special symptoms.

On the other hand, it must not be denied that such a disease as acute inflammation may affect those portions of the walls of the aorta which are vascular in the usual way, and involve the non-vascular inner coat in an irritative and disorderly cell proliferation, after the manner in which acute inflammation affects such tissues. Such cases have not, however, yet been distinguished by definite clinical symptoms. The very striking cases of aortitis related by Dr. (now Sir Dominic) Corrigan, in the *Dublin Medical Journal* for 1838, would, in the light of a newer pathology, be regarded as examples of atheroma of the vessel running a rather rapid course.[1] The case which most nearly perhaps of any on record presented the symptoms which have been ascribed to acute aortitis—viz., fever, rigors, tumultuous action of heart, with intense and painful throbbing of the aorta, and embolic infarction of distant organs—is that related by Mr. Moore in the "Medico-Chirurgical Transactions." vol. xlvii. p. 129, in which he endeavored to promote consolidation of an aortic aneurism that was rapidly making its way through the thoracic parietes, by the insertion into it of numerous coils of fine iron wire, but after death the inflammation was found to be confined to a secondary sac of the aneurism which had perforated the chest-wall. In a remarkable case recorded by Dr. Parkes in the *Medical Times and Gazette*, February, 1850, there was *post-mortem* evidence of recent inflammatory disease, affecting a large portion of the descending aorta, which, however, appeared to have supervened upon disease of old standing which had doubtless much modified the anatomy of the walls of the artery and the ultimate distribution of its nutritive vessels. The disease in this case was not attended with any characteristic symptoms during life, the absence of which might, however, have been due to the almost insensible condition of the patient while under observation.

It would be unprofitable to dwell further upon a disease of the existence of which, as a primary affection, there is, we think it may be said, as yet no sufficient clinical or *post-mortem* evidence.

Aortic Endarteritis, Atheroma.

The inflammatory process by which the aorta is commonly affected is necessarily of a slower kind from its attacking primarily a non-vascular tissue—the internal coat of the vessel. It consists essentially of proliferation of the cell elements of this coat, commencing in its deeper layers and

[Fig. 124.

Atheroma of the Aorta.—Showing the cellular infiltration of the deeper layers of the inner coat, and the consequent internal bulging of the vessel. The new tissue has undergone more or less fatty degeneration. There is also some cellular infiltration of the middle coat. *i.* internal, *m.* middle, *e.* external coat of vessel. × 50, reduced ½. (Green.)]

extending sooner or later to the middle and external tissues.

The pathology of this process is minutely described in another article; its effects upon the walls of the aorta—which are spoken of collectively as atheroma—may be referred to in three stages or degrees—(1) patchy thickening, with some softening, mainly affecting the inner coat, unevenness of the inner surface, and diminished elasticity of the vessel; (2) fatty

[1] The cases described by Norman Chevers, Guy's Hosp. Rep. 1841, are so difficult to recognize in accordance with the pathology of the present day, and so complicated with other diseases, as to be useless for clinical illustration.

degeneration of the affected tissues, fibroid thickening of the whole vessel wall; (3) crumbling down, or infiltration with calcareous salts, of the degenerated internal or middle coat; great consequent roughening of the inner surface of the aorta, and increased brittleness of the vessel.

In the earlier stages of aortic endarteritis there is sometimes narrowing of the calibre of the vessel from the intrusion of its thickened walls; but dilatation almost always takes place later on in the disease. The later stages of atheroma, softening, and calcification, are little more than the effects of imbibition and chemical change acting upon a part which has lost its vitality.

Atheroma may then be defined as degeneration of the coats of the aorta, the result most commonly of preceding inflammatory change (endarteritis), but, it must be added, sometimes occurring primarily as fatty transformation from senile decay.

The *seat* of the disease is most commonly at the commencement of the aorta, and this is the portion most affected even in those cases in which other parts of the vessel are involved. Moreover, we can rarely obtain clinical evidence of atheroma affecting the aorta beyond its ascending or transverse portion.

Etiology.—It is of some importance clinically to bear in mind respecting the pathology of atheroma, with which we are now concerned, that the slow inflammatory changes which lead to its production are, in the majority of instances, of a degenerative kind from the first—that is to say, they are associated with some constitutional cachexia or with senility. Even in those cases in which the disease occurs in earlier life, and apparently as the result simply of undue arterial strain, there may usually be strongly suspected some antecedent impairment of nutrition to account for that sensitiveness or want of resilience to strain which leads to the setting up of atheroma.

It has, however, been shown very clearly by the clinical observations of Dr. Clifford Allbutt and Mr. Myers, and the pathological inquiries of Dr. Moxon and others, that aortic atheroma is particularly common among those who are engaged in occupations of a constantly laborious kind—strikers, bargemen, those who work heavy pumps, &c.; and Dr. Allbutt regards daily continued heavy labor as much more efficacious in producing this result than intermittent toil of even a more severe kind, such as the athletic sports of the higher classes; but he also lays stress upon his opinion that depressing circumstances of life, bad air and food, greatly favor atheroma arising from strain.[1]

Rheumatism, gout, syphilis, and kidney diseases are the maladies most predisposing to atheroma. Intemperance and hereditary tendency hasten its appearance. Of senile changes, atheroma is one of the most constant, but it only rarely comes under our clinical observation, from the diminishing activity and vigor of the circulation with advancing age rendering the results of atheromatous change in the aorta less likely to manifest themselves.

Mr. Francis H. Welch of Netley has in a recent paper read before the Medico-Chirurgical Society of London (1876) contended that nodular disease of the aorta (endarteritis) is one of the most frequent internal lesions of the syphilitic virus and also one of the earliest produced—a view supported by some high military authorities. Mr. Welch found that out of 56 cases of syphilis terminating fatally from specific lesions 60·7 per cent. showed aortic nodulations. Again of 34 cases dead of aortic aneurism 50 per cent. at least were strongly infected with syphilis. Mr. Welch's important paper did not pass without criticism, and it will no doubt do much to hasten the solution of a very complex question as to the real potency of syphilis in developing atheroma. The main issue rests upon what is regarded as evidence of syphilis both during life and after death.

Symptoms and Physical Signs.—The symptoms of atheromatous change in the aorta are always obscure; and extensive disease may exist without any symptoms being complained of; nor on the most minutely careful physical examination can we in all cases assert that there is or is not atheromatous disease of the main vessel present. The secondary results of atheroma are, in the first place, mechanical—viz., dilatation, aneurism or rupture of the vessel, or embolism from the conveyance of masses of fibrine which have been entangled by the roughened surfaces to distant parts; and it is only when such secondary phenomena begin to arise that symptoms or signs of disease present themselves. Our object must then be to discover, at the earliest possible moment, the commencing secondary consequences of atheroma, so as to be on our guard, so far as it is possible, against their further extension.

Attacks of angina or of palpitation, occurring independently of effort, but readily brought on by exertion, are suggestive of this form of the disease; but it as often happens that some casual symptom which, so far as symptoms are yet classified, might mean anything or "dyspepsia," in the man before us directs our attention to the heart. The patient is of an age at or beyond that of middle life,

[1] On Overwork and Strain of the Heart, and Aorta.

which is in favor of the probability that such cardiac attacks may be dependent upon dyspepsia (or, in the case of women, climacteric hysteria), and the presence or absence of these conditions must of course be carefully ascertained; but their existence must not be regarded as sufficient to exclude the graver malady, for they frequently coexist with and complicate aortic disease. There is nothing characteristic in the appearance of the patient; he may be thin and cachectic looking, or the reverse; but whether his appearance suggests such questions or not, rheumatism, gout, syphilis, and intemperance should be inquired for, and the urine repeatedly examined for albumen or morbid deposits. On examination, the radial and brachial arteries will commonly be found more rigid and inelastic than natural.

Although during an attack of dyspnœa the heart's action is tumultuous and the pulse alarmingly irregular, yet at the time the patient comes under observation the cardiac movements may be quite steady, with perhaps an occasional intermission, there is some evidence of hypertrophy of the left ventricle (increased impulse with muffled sound), and perhaps nothing else can be discovered; from these patients being big-chested and more or less emphysematous, it is also sometimes difficult to judge of the cardiac hypertrophy. In more marked cases, however, there is with indistinctness of the first sound, accentuation of the second and a *short systolic, or rather post systolic, murmur* over the aorta beyond the valves. It may be that this murmur is only discoverable when the heart is acting strongly as from excitement, or after taking a few turns up and down the room—and in suspicious cases this exercise prior to a second auscultation should never be omitted; the murmur does not displace the first sound, but is superadded to and immediately follows it. In other cases there is a *partial* replacement of the second sound by a fine diastolic murmur. These latter physical signs are often not to be found for the first few months, during which the patient has presented suspicious symptoms, and *their supervention in this way is the most significant feature in the history of such a case for diagnosis.* In the upper sternal region both the cardiac sounds are accentuated, and may be attended even with slight shock *to the ear;* in order correctly to value this sign, however, a reverse precaution to that mentioned above should be adopted—we should note that the patient be quite calm, and if possible, make a second examination after he has been lying down for a short time.

At a later period, signs of decided dilatation may become apparent, there may be some dulness over the aortic region, and some pulsation—rather flapping than thrusting—may be felt by the finger at the second interspace close to the sternum. The bruit becomes more distinct, sometimes very rough and accompanied by fremitus, when calcareous degeneration may be presumed to be present. The dyspnœa increases, and the anginal attacks may become more frequent and severe; or, on the other hand, they may disappear; but palpitation is always more persistent. The symptoms of embolism may now come on, among which hemiplegia, rigors and hæmaturia, superficial hemorrhages, and gangrene may be enumerated; or sudden pain, dyspnœa, and faintness may announce the commencement of a sacculated aneurism, or death may suddenly take place from cardiac syncope or rupture of the aorta. In other cases sacculated aneurism may imperceptibly arise.

The acute symptoms signalizing the formation of a dissecting aneurism may be the first to announce to us the existence of long preceding atheroma, and it is unnecessary to say that the disease, the phenomena of which are thus related in chronological order, may first present itself to our notice at any of the stages referred to. In all cases of suspicion the general state of the circulation should be carefully examined, the aid of the sphygmograph being sought.

DURATION.—The duration of aortic endarteritis or atheroma cannot be precisely stated, from the insidious manner in which the disease commences. It may be considerably, perhaps indefinitely, prolonged by careful management, hence the importance of its early recognition. The disease proceeds to its fatal termination either by simple progress leading to rupture of the vessel, or by embolism of the brain or other organs from conveyance of debris or fibrinous plugs; or by dilatation of the aorta, or direct involvement of its valves, giving rise to incompetency with its attendant cardiac results, or, finally, by the formation of a sacculated or dissecting aneurism. In calculating the *prognosis*, the present duration of the disease and the progress it has already made in one or other of these directions has to be considered.

TREATMENT.—The treatment of aortic degeneration is purely palliative. A careful regulation of the diet so as to avoid both overloading of stomach and too long fasting, is of the first importance; stimulants should be reduced to a minimum, or dispensed with altogether, and the hepatic function should be carefully attended to. A mild but bracing climate with level

walks and carriage exercise are desirable. Of drugs the aromatic stimulants and antispasmodics are most useful during the attacks of dyspnœa. The subcutaneous injection of morphia is very useful in warding off the attacks, when, as is not infrequently the case, they show any tendency to recur at definite times.

ANEURISM OF THE THORACIC AORTA.

By R. Douglas Powell, M.D., F.R.C.P.

Aneurism at the Sinuses of the Aorta.

Aneurism affecting the very commencement of the aorta, either at or immediately above one of the sinuses of Valsalva, rarely attains a sufficient size to assume special characters of its own before death takes place, either from its rupture into the heart or pericardium, or indirectly from the grave derangement of the valves and orifices at the base of the heart it has occasioned. It may therefore be more conveniently considered here than among the larger aneurisms affecting the rest of the aorta. The aneurism occurs usually above the right coronary valve, next most frequently above the intercoronary valve or between these two (Sibson). It is also always of the sacculated variety. The pouch necessarily projects in most cases into the right side of the heart at or near the commencement of the pulmonary artery.

Symptoms and Physical Signs.—In a certain number of cases there are no symptoms to attract our notice to the heart, until the patient suddenly dies from rupture of the sac. In other cases the symptoms and signs are those of atheroma, with some dilatation of the first portion of the aorta. The expansion of the portion of the aorta forming the base of the aneurism, tends to displace downwards the attachment of the aortic valves, and if situated above the junction of two of these valves necessarily occasions great incompetency, with all the signs and symptoms of aortic regurgitation. The pouch by projecting, as it most commonly does, towards the base of the pulmonary artery, tends also to displace its valves or narrow its orifice, and hence there may be a systolic or a diastolic murmur situated in the pulmonary region. This latter sign in particular might lead us to suspect the disease. Hypertrophy of the left ventricle, with or without a similar affection of the right, is usually present. In two cases which have come under my own observation, the hypertrophy and dilatation of the right side of the heart were very marked.

Diagnosis.—The *diagnosis* can rarely be made with certainty. It is almost impossible to single out from among the symptoms which may be accounted for by the many attendant lesions, those peculiar to an aneurism rarely exceeding the size of a filbert, and buried in the base of the heart. Yet, where we have evidence of these lesions—of aortic atheroma with some dilatation, and of regurgitation through the valves—the existence of aneurism should always be reckoned upon as possible; and if there be any murmur detected over the pulmonary artery, accompanied by marked hypertrophy and dilatation of the right side of the heart, the presence of aneurism may be fairly assumed.

The termination of the disease is usually by rupture into the pericardium or right side of the heart. Death may ensue, however, from the valvular derangement occasioned by it, or in some other way from the extensive disease of the vessel by which it is most commonly accompanied.

The general treatment of this disease is identical with that of atheroma.

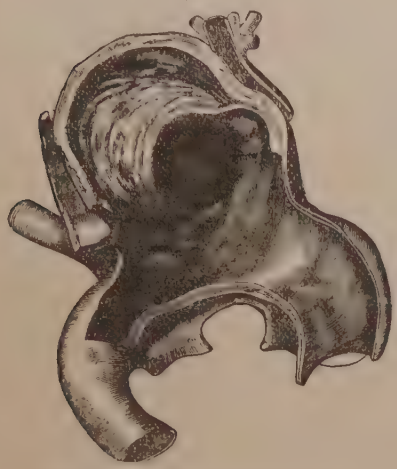
Aneurism of the Thoracic Aorta beyond the Valves.

Aneurism of the aorta is a preternatural dilatation of that vessel at some portion of its course. The dilatation may be *general*, involving the whole circumference of the vessel for a certain length, and such an aneurism may assume the *fusiform*, *cylindrical*, or *globular* shape. The aneurismal expansion of the vessel is more commonly *partial*, from the yielding before the blood-pressure of some circumscribed portion of the arterial wall previously weakened by disease, which bulges outwards from the

vessel as a bud, or *cul de sac*, of gradually increasing dimensions. This variety is spoken of as the *sacculated* aneurism; it is always associated, however, with some general enlargement of the arterial channel.

These varieties of aneurism are more easily classified in the museum than distinguished clinically. Among the *circumscribed* aneurisms of which the sacculated variety is the type and form of most frequent occurrence, we must also include,

[Fig. 125.



Section of Arch of Aorta, with Aneurism.]

for purposes of description, the *globular*[1] aneurism, and those aneurisms strictly speaking perhaps of the dissecting kind, *i. e.*, commencing suddenly with rupture of the internal coat, but in which the lesion is limited, and the further progress of the disease is by gradual expansion. No doubt more aneurisms, regarded even *post-mortem* as sacculated, originate in this way than is generally supposed. The ordinary *fusiform aneurism* is in its slighter degrees more commonly spoken of under the simpler name dilatation of the aorta, and has already been referred to in speaking of atheroma. It usually affects the first portion of the aorta, and, when extensive, yields many of the signs of aneurism presently to be described.

ETIOLOGY.—Speaking generally, whatever increases the pressure of the blood within the aorta or impairs the resisting power of that vessel, favors the production of aneurism. Increased propelling power of heart and increased resistance to the escape of blood from the aorta, into the vessels beyond, are the two conditions which, separately or combined, augment the pressure of blood within the vessel: disease of the walls of the vessel, of whatever kind, diminishes its power of resisting the normal or enhanced blood-pressure.

In discussing more minutely the etiology of aneurisms of the aorta, we must refer separately to the disease as occurring as a result of senile changes, and as being prematurely produced by artificial or accidental circumstances. Senile decay of the arterial wall is one of the natural causes of aneurism. In the normal progress of age, degeneration commences in the large vessels, and probably too in the smaller ones, before the tissue of the heart suffers in nutrition;[1] indeed, the first effect of this arterial decay is, as is well known, a certain increase in the muscular power of the left ventricle to compensate for the increased resistance to the circulation through more rigid vessels; and it is at this period of advanced middle life that aneurism is most frequently met with—but sometimes age in these special tissues is hurried on by favoring disease. Beyond the period of advanced middle life the tendency to sacculated aneurism is lost, the bulk of the blood is diminished, the nutrition of the heart begins to suffer and the vigor of the circulation becomes correspondingly lessened; a lower activity of life is thus necessitated which is quite normal to advancing age and in harmony with the changes taking place in the tissues. In old age, however, it sometimes happens that the senile atheromatous change—no doubt aided by some attendant (secondary ?) inflammation—proceeds to actual softening and erosion of the inner tunic of the aorta, and the blood then insinuates between the coats of the vessel, and a dissecting aneurism arises.

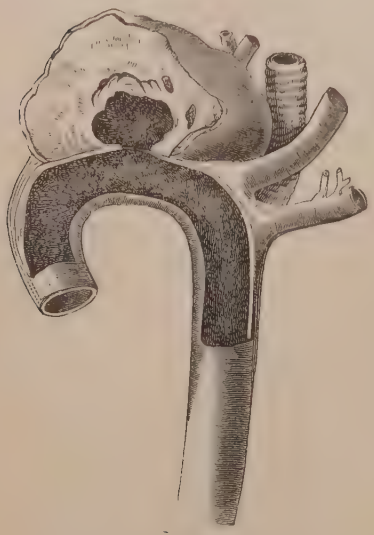
Subacute and chronic arteritis, the varying stages and modifications of which

[1] This is perhaps the best term to apply to those cases in which the dilatation affecting the whole circumference involves only a comparatively limited portion of the length of the aorta. It is not a new one, being used by Dr. Walshe in the same sense, though the cases referred to he regards as of extreme rarity. An aneurism of this globular form presents all the physical signs and clinical phenomena of a sacculated aneurism, even to perforation of the thoracic parietes, as in Dr. Murchison's case related at page 849. One or two instances of this variety, which seems to be more frequent in the descending thoracic aorta, have lately appeared before the Pathological Society.

[1] This statement, it must be admitted, is based rather upon general clinical experience than upon any exact inquiry into the natural relative progress of age in the different tissues, which is as yet wanting in medical literature. It is, I believe, however, in accordance with the impression of most physicians, and is in all probability exactly true.

have already been referred to as accounting for the production of most of the appearances recognized under the term atheroma—thickenings, scars, erosions, calcifications, &c.—is the diseased condition most commonly preceding aneurism; indeed, it is through the intervention of arteritis that all the known causes of aneurism of the aorta, and, perhaps, of other vessels, become effective. The constitutional states, then, which tend to produce atheroma, also favor the occurrence of aneurism—hereditary predisposition, rheumatism, gout, syphilis, kidney disease, alcoholism. Even strain, with but rare and violent exceptions, leads to the production of aneurism, not simply by rupturing any of the coats of the aorta, previously healthy, but by overstretching, or too violently exercising them, and thus setting up local atheromatous disease of the slow inflammatory type. Dr. Moxon[1] has brought together so many arguments to prove the direct effect of strain in producing atheroma as to have, I think, settled the question. He shows how the disease affects first the aorta in the region of the valves and at its ascending portion, and that the inflammatory degeneration occurs in points arranged longitudinally in the course of the vessel. Thus arising, it is not difficult to understand how any of these little disease "rifts" may slowly widen to the production of a large aneurism, or may, under some severe effort, suddenly give way with those acute symptoms which occasionally usher in the obvious disease. Hereditary predisposition has not been found to exist, save in exceptional cases, in aneurism. One remarkable case came under my own observation seven or eight years ago, in which there was a tolerably trustworthy history of a mother and four sons dying of internal aneurism. Dr. Fuller[1] relates the case of a gentleman whose paternal grandfather, uncle, and father, had all died from aneurism, and whose sister was laboring under that disease.

[Fig. 126.



Aneurism of arch of the aorta.]

Although rheumatism is usually enumerated among the diseases predisposing to aneurism, it has hitherto been included among such general causes as syphilis and renal disease, on the tacit understanding that the "rheumatic diathesis" is favorable to the occurrence of premature arterial decay. Whether this be the case is at least, I think, a question for further inquiry; but rheumatism must in a more definite sense be regarded as a possible cause of aneurism, if I may judge from the history of some cases which have come under my own observation.[2] It has been found in certain cases of rheumatic fever with aortic valve disease, that the aorta beyond the valves has presented patches of arteritis corresponding with the impingement against it of vegetations fringing the margins of the valves. Does the rheumatic endocarditis ever extend beyond the valves to produce endarteritis affecting the aorta at its commencement? I have only seen *clinical* evidence in one case which would lead me to answer this question in the affirmative. The case was that of a lad aged 17, in whom aortic aneurism occurred traceable to two attacks of rheumatism occurring at the ages of eleven and twelve. No other cause for the aneurism could be made out, and the patient was too young for degeneration of the vessel, save of an acute kind, such as might possibly be occasioned by rheumatic fever.

The operation of aortic regurgitant disease of the heart, left behind by rheumatic endocarditis, as a cause of aneurism, may be readily conceived in persons at or beyond middle life. I have met with three examples of aneurism, the histories of which will, I think, admit of no other interpretation; and in two other cases I have had reason to suspect the disease to have arisen in this way. Greatly increased power of cardiac systole is required in aortic regurgitant disease to carry on the circulation with an aorta whose action is crippled through imperfection of its valves; the whole shock of

[1] Guy's Hospital Reports, series iii. vol. xvi. p. 448; also recent work on Pathological Anatomy by Wilks and Moxon, chap. "Aneurism."

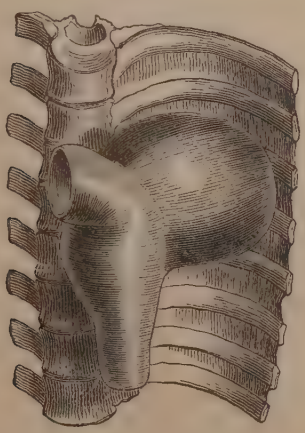
[1] Diseases of the Chest, p. 656.

[2] *Vide* Clinical Transactions for 1874.

this extra power is received of course by the ascending portion of the aorta. Moreover, instead of the unduly forcible impulse of blood being received by an aorta already containing a certain residuum of blood sufficient to diminish the shock, the great vessel is, on the contrary, more empty and flaccid than natural at the moment of ventricular systole, the blood in regurgitant disease escaping back into the ventricle. The effect of this increased impulse, or shock, upon the aorta must be to predispose it to the occurrence of atheromatous disease and of subsequent expansion.

Intemperance, mental emotion, and violent exercise operate as causes of aneurism by increasing the blood pressure within the aorta. Dr. Rendle's statistics of cases of internal aneurism occurring among the female convicts in the Queen's Prison, Brixton, conclusively show the effect of mental depression and excitement in predisposing to aneurism. As already hinted, loss of muscular tone of the vessel, through nervous influence,[1] may take a more important part in such cases in the origin of aneurism than is at present allowed. Mechanical impediment to the circulation through the great vessels beyond the aorta is, however, perhaps the most important cause of aneu-

[Fig. 127.



Aneurism of Aorta.]

rism, with special reference to which the malady will be found to prevail more at certain ages, among those following certain occupations, and, with some reservations, in the male sex.

In an exhaustive essay on the etiology of diseases of the heart among soldiers, Mr. Myers[1] gives the statistics of Inspector-General Lawson ("Army Medical Report," 1866), showing that the *deaths* from aortic aneurism are in the army eleven times greater than among the civil population; and he accounts for this enormous disproportion by the tightness of the dress and accoutrements of the soldier occasioning greatly increased blood-pressure within the aorta during any violent exercise, by compressing the great vessels of the neck and upper extremities. In proof of this Mr. Myers gives one set of 703 cases of aortic aneurism from Dr. Sibson, in 420 of which (59·7 per cent.) some portion of the ascending aorta was involved. He also gives—in support of Dr. Sibson's view that that portion of the aorta on which there is most strain is most often affected with aneurism—109 other cases, culled from the Netley Hospital records, and from those of his own regiment, in 75 (about 70 per cent.) of which the disease affected the ascending portion or arch of the vessel. He thus successfully endeavors to locate the cause of the disease in soldiers from its point of manifestation being disproportionately more frequent in them at that portion of the aorta which is at or above the origin of the great vessels of the neck and upper extremities.[2] As further evidence of the effect of strain in producing heart disease among soldiers, Mr. Myers mentions the significant fact that among them aortic valve disease is twice as frequent as mitral, whereas in civil life mitral disease is slightly the more frequent. He also refers to the very large proportion of valvular heart disease in the army, which are not traceable to either rheumatism or albuminuria, the two diseases answerable for most such cases occurring in civil life. It must, of course, be remembered that wherever the obstruction may be, the aortic at its origin must be most importantly affected; for it is here next to the ventricle, that the supplementary force is gathered to overcome it, and perhaps the great and almost sudden change in the mode of life on entering the military ranks—from that of the slow-moving, slouching, ill-fed farmer's lad, or the

[1] Niemeyer refers to palsy of the vasomoter nerves as a questionable cause of aneurism, though he gives Rokitansky's opinion in favor of it.

[1] On the Ætiology and Prevalence of Diseases of the Heart among Soldiers. London, 1870.

[2] Taking the whole of Dr. Sibson's 880 cases, of which in 460, or 52·3 per cent., the disease was situate at either the ascending or transverse aorta, or between the two, and further making allowance for the fact that these cases were of all ages, whereas those quoted by Mr Myers were within the age of effective service, the preponderance of the affection in ascending aorta among soldiers appears still more considerable.

loafer from among the unhealthy recesses of large towns, to that of the smart, straight, large-eating and more or less plethoric soldier—should be also taken into account, as well as his disadvantages in dress, in considering the effects of the soldier's occupation in predisposing to aneurism.[1] However this may be, it will be readily perceived that arterial strain tending through the medium of atheroma to produce aneurism among other diseases of the heart and aorta, is the common result of many of the conditions of the soldier's present life and training. Sailors, with an equal liability to great physical effort, and affected in about equal proportion with the taint of syphilis, although more subject to aneurism than civilians, are less so than soldiers, their greater amenity in this respect appearing due mainly to their looser dress and more gradual training. The occupations of hammermen, lightermen, smiths, and others, necessitating long-continued muscular effort, predispose to aneurism.

Age.—Aneurism of the aorta may occur at any age; it is, however, extremely rare before twenty, and is most prevalent between the ages of forty and fifty. A small number of cases, 5 or 6 per cent., occur before thirty. Professor Lebert[2] finds the disease most prevalent between the ages of forty-five and sixty. In Dr. Crisp's[3] tables, 132 out of 175 cases occur between the ages of thirty and sixty, this wide margin being, as before explained, accounted for by certain habits and constitutional states anticipating by disease the effects of age upon the vessels. The great majority of aneurisms occurring between these periods of life are of the circumscribed sacculated or fusiform kind; in advanced life, on the other hand, those cases which do occur are most commonly of the dissecting kind (Peacock[4]).

Sex.—Males are much more liable to true aneurism of the aorta than females, though the latter are by no means exempt from the disease. Of circumscribed aneurism of the aorta from two-thirds to four-fifths of the cases occur in males (Crisp, Peacock, Blakiston); on the other hand, dissecting aneurism appears to be of as frequent occurrence in women as in men. That this disproportion between the sexes is due entirely to the difference in their habits of life is apparent from the statistics of Dr. Rendle, already referred to. Mr. Holmes[1] remarks that "internal aneurisms seem equally if not more common among women when their way of life exposes them to the vascular excitement consequent on intemperance, vice, and mental emotions."

Though aneurism appears to prevail more in Great Britain than in other countries, this excess is attributable to our rougher mode of life rather than to any climatic influence.

Symptomatic History and Symptomatology.—As a rule the commencement of aneurism of the aorta is unmarked by symptoms, and the disease may continue latent up to the time of death. The so-called *exciting causes* of aneurism, those acts or accidents which seem immediately to determine the commencement of the disease, cannot well be classified; when recognizable they consist either of some temporary exaggeration of usual daily toil, or of some shock, or fall, or blow, violently affecting the circulation. Such causes are, however, but rarely sufficient to account for *internal* aneurism if we attempt to isolate them from the predisposing, *i. e.* the true causes of the disease. In the sacculated or circumscribed forms the history, when any can be discovered, may be referred to one of the following types. In one case there may long have been signs of failing health and nutrition, and positive signs of degenerative disease of the aorta may have previously been detected. In another the patient has perhaps enjoyed robust health and indulged in active pursuits and free living until, after a fall from a horse, or a blow, or a sudden violent effort, he feels a momentary pain in the chest and faintness, soon passing out of memory but recalled by the consciousness, on subsequently assuming his wonted exercises, of a gradually increasing shortness of breath and palpitation, with deep-seated pain in the chest; the dyspnœa becomes habitual, and he is ever conscious of too great a pulsation within the chest which disturbs his rest. He then seeks advice. Sometimes the symptoms come on more acutely—*e.g.*, a tailor on lifting while in the "squatting" posture the heavy seam press he has been accustomed to wield for years in the same way, is seized with agonizing pain in the left breast, prolonged faintness and violent palpitation, and soon a pulsating tumor appears in the aortic region, where he had felt "something

[1] It is a fact worthy of note that severe gymnastic exercises have only been in vogue in the army within comparatively recent years. I have, however, met with aneurism in several soldiers who have never been subjected to them.

[2] Virchow's Handb. der spec. Path., Bd. v. abth. ii.; Krankheiten der Blut- und Lymphgefässe, p. 25.

[3] Diseases of the Bloodvessels, p. 135.

[4] On Dissecting Aneurism. Edin. Surg. and Med. Journal, vol. lx. p. 291, 1843.

[1] System of Surgery, vol. iii. p. 419

give way." The symptoms in other cases come on almost imperceptibly with cough, dyspnœa, and partial aphonia, attributed to cold, yet the patient will, on being questioned, perhaps date them back to some protracted labor or "heavy job," as in the case of a blacksmith or lighterman.

At the time of applying for advice the symptoms vary in their nature and intensity with the seat of the tumor and the direction in which it is growing. They are all due to pressure upon the neighboring parts causing their displacement or erosion, interference with the patency of air and food tubes, irritation or destruction of nerves, and they may be thus enumerated nearly in the order of their frequency — *Pain*, *dyspnœa*, cardiac or pulmonary, or both, persistent in greater or less degree, but often associated with paroxysmal attacks; *voice altered*, husky, uncertain, or whispering; *cough*, dry, hoarse, or ringing, laryngeal, *stridulous breathing*, *headache*, *disordered vision*, loss of power or positive *paralysis* of lower extremities.

Few if any of the symptoms as thus enumerated can be regarded as specially distinguishing aneurism from other tumors in the chest, their significance rests rather upon their grouping and upon certain characters about them which require further consideration. The existence of an aneurism may sometimes be inferred from the presence of certain symptoms whilst the physical signs are as yet most obscure, and otherwise insignificant.

The *pain* in aneurism is of a wearing, aching, or burning character, corresponding in position with the seat of the tumor. It is a fixed pain, but associated with paroxysmal pains of an evidently neuralgic character, radiating in the course of contiguous nerves. The first onset of the disease is occasionally ushered in, as before said, by very acute suffering.

[Fig. 128.

Aneurism of Aorta, which burst into the Trachea.]

The *dyspnœa* present in cases of thoracic aneurism is of various kinds and grades, and has many causes. (1) The mechanism of the circulation being disordered, any effort giving rise to an extra demand upon the heart occasions dyspnœa. (2) The simple fact of there being a tumor within the chest compressing and displacing its other contents necessitates some degree of dyspnœa. The fact of the tumor being pulsatile and of dimensions varying slightly from time to time with the fulness of the circulation may serve to give a distinguishing feature to the dyspnœa, viz., its more marked sensitiveness to conditions tending to disorder circulation than in the case of other tumors. A certain deep-seated throbbing is sometimes complained of after effort or excitement. But (3) the dyspnœa most characteristic of aneurism occurs in paroxysmal attacks, in one of which the patient not unfrequently dies. Much dispute has arisen as to the mechanism of this form of dyspnœa, and upon the views we hold respecting it will depend our treatment of cases of impending suffocation from this cause.

The pneumogastric nerve is sometimes affected in the disease; it may be either compressed and flattened by the tumor, or destroyed and incorporated with the sac by inflammatory change. More commonly one or both of the recurrent branches are affected, the left most frequently so by direct compression, or the right indirectly by dragging of the tumor upon the origin of the subclavian. Either paralysis or irregular spasm of the laryngeal muscles on one or both sides may thus be occasioned. Among those who attribute these attacks of dyspnœa to disturbance of the innervation of the glottis, some regard them as due to *spasmodic* closure of the glottis, others to *paralytic* closure. Spasm of the glottis is the cause to which the dyspnœa is most commonly attributed.

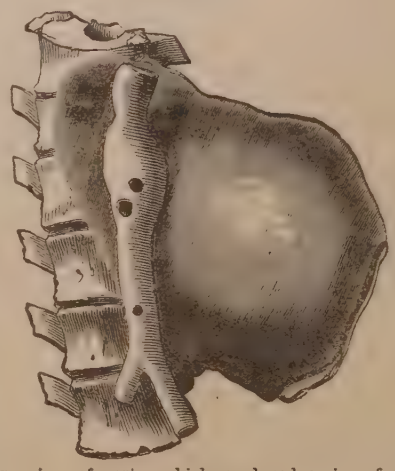
It is generally held that, to quote the words of Dr. John Reid,[1] "all the muscles which move the arytenoid cartilages receive their motor filaments from the inferior or recurrent laryngeal nerves. And as the force of the muscles which shut the larynx preponderates over that of those which dilate it, so the arytenoid cartilages are carried inwards, when all the filaments of one or both nerves are irritated." This is the view upon which those who hold the spasm theory mainly

[1] Physiological Researches, No. iv. Experimental investigations into the functions of the eighth pair of nerves, 1848. Dr. Sanderson (Handbook of Practical Physiology, p. 298) regards all the intrinsic muscles of the larynx as expiratory, the widening during the inspiration being a condition of general relaxation.

rely. M. Krishaber[1] observes, as the result of some experimental inquiry, that in paralysis of the chords either from destruction of the recurrent nerves or division with the knife of one or both of them, there is no dyspnœa, the glottis being actually more patent under such circumstances than natural. This view, however, is entirely opposed to the conclusions drawn from similar experiments by others. The experiments of Legallois and John Reid distinctly show that on section of the recurrent nerves the paralyzed chords are sucked together with each inspiratory effort, giving rise to suffocation or great dyspnœa. Some experiments I have myself made on the cat entirely confirm in this respect those of Legallois[2] and Reid, while in another particular, the effect of irritation of these nerves upon the larynx, they, so far as they have gone, lead me to hesitate in accepting the conclusions of these and other experimenters. For while the cat was in a state of urgent distress from the sucking together of the paralyzed chords with each inspiration, first one inferior laryngeal nerve, then the other, and then both, were galvanized with a weak current, and in each case the corresponding arytenoid cartilage was powerfully rotated outwards, so as to widely open the glottis and instantly relieve the dyspnœa.[3] While, then, I think that further experimental inquiry will tend to show that spasmodic closure of the larynx may not in man be caused by irritation of the recurrent nerves, clinical observation is also in favor of the dyspnœa of aneurism, so far as it is laryngeal, being due to paralysis. When the larynx is at all affected, paralysis—denoted by the altered voice, and observable by the laryngoscope—is the lesion usually present. Dr. Habershon[4] has found actual atrophy of the laryngeal muscles on the affected side. Dr. George Johnson and Dr. Baümler in the 23d and 24th volumes of the Pathological Transactions respectively, have each recorded a case in which bilateral paralysis of the chords was found during life, and *post mortem* the laryngeal muscles on both sides were found to have undergone atrophic changes. In these two cases the trunk of the vagus, as well as the recurrent nerve on one side, was found compressed by the tumor, the nerves on the other side being free. Dr. Johnson considers that the bilateral paralysis in these cases may have been due to the compression directly paralyzing the muscles on the same side through the recurrent, and causing reflex paralysis of the muscles on the opposite side through the trunk of the pneumogastric and the efferent nerves in relation with its centre. Dr. Johnson holds that bilateral spasm may be occasioned in a similar way.[1]

But how does paralysis of the chords give rise to occasional paroxysms of dyspnœa? The mechanism of these attacks of dyspnœa seems to be either (*a*) that owing to sudden enlargement of the aneurism from excitement of heart, after a full meal, or from mental emotion or physical exertion, the increased pressure on the nerve renders a partial paralysis complete, and a paroxysm of dyspnœa occurs from sucking together of the flaccid chords; or, (*b*) as one may observe in some cases in

[Fig. 129.



Aneurism of aorta, which produced caries of vertebræ.]

which the chords have been destroyed by ulcerative disease, so when they are paralyzed, effectual cough being impossible, mucus collects at the glottis, and gives rise to dyspnœa. And moreover we must remember that—as may be well seen in experimenting with animals—during any excitement of breathing, the dyspnœa already present from paralysis of the chords necessarily becomes more urgent; the more powerful the inspirations the more completely is the glottis closed by atmospheric pressure. But there are at least two other causes of paroxysmal dyspnœa in aneurism, affecting the trachea or bronchi. Dr. Bristowe, in a valuable communication to the St. Thomas's Hospital

[1] Comptes Rendus des Séances de la Société de Biologie, October, 1866, d' l'Opportunité de la Trachiotomie dans les Anévrismes de la Crosse de l'Aorte.

[2] Sur le Principe de la Vie, 1812.

[3] These experiments were made in the summer of 1874 with the kind help of Dr. M. Bruce and Mr. Schäfer.

[4] Medico-Chirurgical Transactions, vol. xlvii.

[1] *Vide* Medico-Chirurgical Transactions, for 1875.

Reports for 1872, expresses his belief that the dyspnœa of intrathoracic tumor is only purely laryngeal, and contends that it is most commonly due to direct narrowing of the trachea by the pressure of the aneurismal or other tumor, and to accumulation of mucus at the point of stricture, acting merely mechanically by plugging the narrowed opening, and perhaps causing in addition some spasmodic contraction of the tube. He illustrates his view by several cases; and there are many others, one of which has lately come under my own observation, which bear no other interpretation; and I think the explanation will be found applicable to those cases in particular in which there is marked stridor on one or both sides of the chest, according as the trachea or one main bronchus is pressed upon. In such cases of course all idea of relief by tracheotomy must be abandoned. Again in some cases of aneurism, particularly when affecting the third portion of the arch, paroxysms of dyspnœa, closely resembling those of asthma, may be occasioned by compression of the small branches of the pneumogastric forming one of the pulmonary plexuses. (Gairdner.) Direct pressure upon the trachea may also, however, as I have seen in one remarkable instance, give rise to dyspnœa having very closely the characters of asthma.

Palpitation, or cardiac dyspnœa, is usually an intermitting rather than a constant symptom in aneurism; it is generally complained of on slight exertion. Attacks of true angina pectoris are sometimes witnessed in cases of aneurism affecting the first portion of the aorta, and probably arise from pressure on the cardiac plexuses (Gairdner). There is usually more functional disturbance of the heart when the disease is thus situated. Acceleration of pulse is sometimes, however, a persistent symptom: in the case of a woman for many months under my notice with aneurism of the first portion of the arch, the pulse was constantly beating at a rate of between 130 and 140 per minute, the patient complained of palpitation, and assured me that the heart's action was not quickened from excitement at the times of my repeated observations.

Dysphagia, although often present, is a less constant symptom in aneurism than in other tumors in the same situation.[1] I have seen two or three instances in which an aneurismal tumor has caused a circular perforation of the wall of the œsophagus, without any distress having been complained of from difficulty of swallowing.[1] There is sometimes a spasmodic character in the dysphagia, which is regarded as of reflex origin.

Headache and *disordered vision* are occasional symptoms, the former referable to obstructed return of blood from the head, the latter to pressure upon the sympathetic affecting the size of the pupil.

Hæmoptysis, though it may occasionally, and to a slight extent, occur in the course of aneurism, from bronchial congestion or lung irritation, as a rule only presents itself as the final symptom in those cases in which rupture of the aneurism takes place into the trachea, œsophagus, or one of the great bronchi. Sometimes this final gush is preceded for a few days by a sanguineous tinging of the scanty expectoration, and Dr. Gairdner mentions a case in which this expectoration preceded death by a considerable interval. Dr. Blakiston[2] has recorded a case in which fragments of discolored coagula were expectorated with blood two or three weeks before death, which materially aided the diagnosis of an aneurism of the descending arch communicating with the left bronchus.

PHYSICAL SIGNS.—It is by the physical examination of the patient before us—the observation of all the signs discoverable by the eye, the hand, the ear, aided by the stethoscope, the laryngoscope, and the sphygmograph—that we clinch the diagnosis as to the existence and probable seat of the aneurism which the symptoms present have led us to look for.

The physical signs of aortic aneurism vary greatly in the distinctness with which they are manifested according to the position and size of the dilatation. It is, practically speaking, true that an aneurism may be present and give rise to death by rupture without having ever presented any distinctive signs. Such signs, however, even in obscure cases, are more often overlooked than absent, and may usually be discovered on diligent examination. We will first enumerate all those that may be found in tolerably obvious and typical cases.

Summary of the Physical Signs that may be Observed in Aneurism of the Ascending or Transverse Aorta.[3]—It is in a consider-

[1] M. Leudet observes that dysphagia in compression of the œsophagus by aneurism of the aorta is often absent. "Recherche sur les Lésions de l'Œsophagie causés par les Anévrismes de l'Aorte." Compte Rendus de la Soc. de Biol., 3me sér. 1863, p. 180.

[1] For a good example of this, *vide* case of aneurism by Dr. Quain, Path. Trans. vol. xvii. p. 110. Dr. Fuller also refers to the occasional occurrence of severe lesion of the œsophagus from aneurismal pressure without any corresponding dysphagia. Diseases of Chest, 1862.

[2] Diseases of the Heart, p. 56, case 24.

[3] These aneurisms are specially referred to here, not only because they are the most

able proportion of cases at once evident on *inspection* of the patient stripped to the waist, that he is suffering from aneurism of the aorta. We observe the veins large, full, and tortuous in the humeral region and neck on one side, more rarely on both, a certain fulness, and deepening of the antero-posterior diameter of the upper chest near the sternum on that side, or the superior portion of the sternum itself is rounded and prominent, marbled with blue veins. At the most prominent portion of the costal or sternal bulging, there is distinctly visible pulsation, or there may be a more confused but rhythmic shock apparent with each beat of the heart. Sometimes the pupil on one side corresponding with the pulsating tumor is notably smaller than the other.

On now employing *palpation*, one hand being applied to the seat of the apex beat, which is usually shifted a little downwards and to the left, and the other placed on the tumor, we feel two centres of pulsation, synchronous or nearly so, within the chest—a very significant sign of aneurism. More carefully noting the character of the morbid impulse, we may observe it to be distinctly heaving, expansile, spreading out from some central point. Thrill, either systolic or diastolic, or both, may be perceptible over the tumor; it is only very rarely, however, to be observed in sacculated aneurism. It may very often be found on comparison that the pulse is more feeble at one wrist than the other, or it may be obliterated on one side altogether.[1] Some hardening from degeneration of the vessels may perhaps be observed at the same time.

On *percussion*, which must always be performed with great gentleness, the note is found to be dull, over the unusual prominence; the dulness includes the sternum, extending laterally on one or both sides to gradually fade into lung resonance. It may be continuous with the cardiac dulness which is lowered or separated from it by a band of resonance, or it may encroach upon the sterno-clavicular or episternal regions. Together with the dulness there may usually be noticed increased resistance or hardness over the seat of the tumor, and this is especially marked in cases of large aneurism containing much coagulum. Some dulness may also be detected on percussion in the upper interscapular region on one or both sides.

On *applying the stethoscope* to the suspected region, the first thing which attracts attention is the impulse or systole' shock, which may be intense to the ear when it is not perceptible to the eye, and only barely so to the most attentive palpation. This systolic shock may be accompanied by a bruit usually grave, rough, expansive, more distinct over the centre of the tumor than over the aortic valves. The second sound, clear and ringing at the base of the heart, may over the tumor be accompanied by a peculiarly abrupt shock or second impulse to the ear, which impulse may be even apparent also to the hand. Sometimes a diastolic bruit is audible, in which case the second shock sound is usually obscured or lost. All the auscultatory sounds of aneurism are most audible directly over the tumor; they may be conducted along the course of the aorta, and become very audible at one or both interscapular regions. Over the tumor the respiratory murmur is absent, but on passing the stethoscope aside to the acromial region, the breath-sound is found to be more or less bronchial, and the voice-sound to be more bronchial, though true bronchophony is rare. In the interscapular or supra-spinous region of the corresponding side, the respiration may also have a tubular quality. Over one lung, more rarely over both, the breath-sound has often communicated to it a peculiar sonorous vibrating quality, probably by conduction from the laryngeal stridor present.[2] The respiratory murmur is often weakened, and it may be completely annulled at one base, though this is rare with the obvious aneurismal tumors we have now principally in mind. With the laryngoscope no alteration may be found in the condition of the cords, or they may be lax and act feebly with respiration, or one (usually the left) may be completely paralyzed and motionless. In rare cases both vocal cords have been found paralyzed. The employment of the laryngoscope and sphygmograph would be quite superfluous in the presence of half the signs above enumerated. They be-

common, but because of them alone can any general description including all the essential phenomena of the disease be given. Aneurisms in the other situations present one or more of the same signs obscured by their greater depth from the surface; such could only be treated of as individual cases, and will be referred to more particularly in discussing the diagnosis of thoracic aneurism.

[1] It is usual, but perhaps scarcely necessary, here to warn the too eager observer against mistaking abnormal distribution of radial or contraction of pupil from old iritis for signs of aneurism.

[1] In speaking of the systolic and diastolic phenomena of aneurism, I refer to those signs presented synchronously (or nearly so) with the systole and diastole of the heart respectively.

[2] Professor Stokes attaches great importance to this sign. "Diseases of the Heart and Aorta," 1854, p. 556.

come useful aids in certain obscure cases, however, to which we shall presently refer.

Such are the signs of aneurism which may be present in cases in which the dilatation is situated near the surface, at the ascending or transverse aorta — its favorite seats. We must, before considering the less certain signs presented by aneurism more obscurely placed, discuss the mechanism and diagnostic value of the more important of those above enumerated.

The unequal pupils, venous obstruction, local bulging of the chest wall, displaced heart, percussion dulness, and the auscultatory phenomena of tubular, enfeebled, or annulled respiration, are mere pressure signs common to aneurismal or other tumors within the chest. The unequal pulses, the rhythmic pulsation, bruits, and shock signs, are specially characteristic of aneurism, though some of them may be produced or simulated by solid growths.

Inequality of pupils is not a very common sign of aneurism, although when present it is a very striking one. The affected pupil is usually contracted and immovable, and corresponds with the side on which the aneurism is situated. Of thirty-six cases of aneurism of the arch, of which I have notes, the pupils were unequal in four only. Dr. Walshe has observed the affected pupil vary within a few days, being "now equal to, now notably, now slightly smaller, now larger, than the other in size," and in one of the four cases above mentioned the pupil varied from day to day in a similar manner. The cause of the affection of the pupil is admitted to be pressure upon the sympathetic, paralyzing it and permitting the unopposed action of the third nerve upon the pupil, or irritating it and producing the rarer phenomenon of dilatation of the pupil by excited sympathetic action (Walshe). The degree of displacement of heart depends upon the position and size of the aneurism. The apex beat when the first or second portion of the arch is affected is lower and more or less displaced to the left. This displacement may be extreme in aneurism of the first portion, the tumor taking up the whole of the normal position of the heart, and being readily mistaken for it. The base of the heart is also lowered, so that the organ lies more transversely in the chest than natural, and there is commonly pulsation at the epigastrium, which must not be hastily received as evidence of dilatation of the right ventricle. The cardiac impulse may be increased in force; it is often, however, not stronger than natural, and may be enfeebled. It is remarkable how much less common hypertrophy of the left ventricle is than one would expect.[1] The cause of this is not clear, unless it be deficiency in the coronary circulation, for one would suppose there must be increased resistance to systemic circulation in all cases of aortic aneurism.

The heaving expansile impulse, distinct from that of the heart, is diagnostic of aneurismal tumor, which, however, must be near the surface to give this sign. It is very frequently present in aneurism of the first and second portions of the arch, and it may also be present in large aneurism of the descending arch, but only in the later stage when erosion of the ribs and vertebral processes has enabled the tumor to present as a pulsatory swelling in the left interscapular region. In cases in which the wall of the sac is greatly thickened by fibrinous laminæ, the expansile thrust may be entirely lost, and a knocking impulse alone felt which it is impossible to distinguish from that communicated to a solid tumor by the aorta underlying it. The position of the tumor and the nature of the diastolic sounds will greatly assist the interpretation of this sign. In all cases where the impulse is obscure, the plan suggested by Dr. Stokes will be found of great value, viz., to "make pressure with the flat of the hand on the anterior part of the chest, while the other hand is placed between the shoulders during expiration." By this means an obscurely and deeply expanding character of the impulse may be detected which will favor the probability of its aneurismal origin. There is sometimes to be felt a distinct thrill with the beat of the tumor. This "frémissement cataire," stated by some authors to be almost always present, is in reality not of frequent occurrence. Of eight cases in which I have myself observed marked thrill, in one accompanying also the diastole, in four the aneurisms were secondary to regurgitant disease of rheumatic origin, and they were all probably of the fusiform kind.[2]

The systolic bruit, often absent,[3] though occasionally to the experienced ear very characteristic of aneurism, is by no means, as a rule, a reliable sign, except in those cases in which it is localized at some portion of the aorta distant from the heart,

[1] Hypertrophy of the Heart was present in 8 only of 22 cases carefully recorded by Dr. Blakiston, in his work on diseases of the heart.

[2] Dr. Hope had never seen a case of aneurism with thrill. Dr. Walshe states it to be more common in "peripheral dilatation" than true aneurism.

[3] Lebert states it to have been present among the earlier signs in half his cases. Loc. cit. It existed in 22 out of my observed 36 cases, in 12 instance the bruit being double.

as in the right or left interscapular region or along the left side of the spine. A diastolic bruit most audible at the site of the suspected aneurism, while the second sound is clear at the base of the heart, is an important sign; a murmur replacing the second sound at the base of the heart is of value in diagnosis when there is also evidence of a thoracic tumor, the nature of which is otherwise obscure; its value consisting in such a case in its indicating disease within the aorta, and so rendering the aneurismal nature of the tumor very probable. The importance of ascertaining whether a bruit replaces one of the sounds of the heart, or is superadded to it, being, as it were, heard through it, has been well pointed out by Dr. Parkes:[1] in at least a third of the cases, however, of obvious aneurism of the thoracic aorta there is no bruit at all audible, and when we take into account obscure cases the proportion becomes much larger.

The peculiar diastolic shock sound when once heard, or rather *felt* by the ear, is not easily forgotten. It is, when present, most significant of aneurism, and when succeeding to a more or less distinct systolic impulse, I believe absolutely so. It is only to be heard in aneurism affecting the first and second portions of the aorta, and when the tumor is very near the surface the shock is not infrequently so great as to communicate a second impulse to the hand. Of the thirty-six cases I have already referred to this sign was present in fourteen; and in ten of these there was no murmur present, although in the majority of them other signs of percussion, impulse, and pressure rendered the diagnosis clear. In a few instances, however, and notably in three, the diagnosis (speedily verified by death) was very difficult, and depended mainly upon the importance attached to this sign. This phenomenon has been variously explained, and has given rise to much ingenious discussion.[2] It is no doubt of complex mechanism, and is made up partly of the conducted second sound which is accentuated in these cases, but is chiefly caused by the transmission of a wave to the surface with the closure of the aortic valves. The sac of the aneurism becomes fully distended a little later than the aorta itself, so that the systole of the vessel commences a trifle sooner than that of the aneurism. The aortic valves close at the moment of aortic systole, and at the instant of their closure the shock wave is transmitted through the aneurism. If there be any imperfection of the aortic valves so as to give rise to appreciable regurgitation, the shock-sound or impulse is either not developed or very imperfectly so.

Diagnosis.—Having given at some length the general symptoms and signs of aneurism, keeping in view more particularly those cases in which the disease affects that portion of the aorta occupying the anterior mediastinum, we have to take also into account in the diagnosis the question as to the part of the aorta affected by the aneurism, and, if possible, how to distinguish between aortic aneurism and dilatation of the innominate, subclavian, carotid, and pulmonary arteries respectively.

In aneurism of the *ascending aorta*, but beyond the heart, the signs are grouped about the second right space close to the sternum as their centre. Greater displacement of heart, and interference with its function, with cyanosis and dropsy of the upper half of the body from pressure upon the innominate vein, are more often met with when the disease is in this situation. The rule is nevertheless for the aneurism to extend towards the surface rather than deeply, so that the vein often escapes serious compression. As the disease advances, the downward displacement of the base of the heart becomes more decided, and the area of pulsation enlarges downwards and to the right (Sibson). When the *transverse portion* of the arch is affected, the *manubrium sterni* is the central region of disease signs, which have a tendency to extend, however, more to the left than the right of the sternum. There is frequently no external tumor from the aneurism projecting backwards from the arch. The signs of pressure upon the air and food tubes, and their functional disturbance through involvement of the pneumogastric nerve, are most common in this variety, inequality of pulses also shows extension of the disease to this portion of the arch. In aneurism of the *descending portion* of the arch the signs usually present themselves most distinctly on the left side of the spine in the upper interscapular region, although the tumor may present at the second left space near the sternum. The pain is severe in the back and shoulder, the dyspnœa is usually pulmonary, either paroxysmal, assuming the character of asthma, or constant from pressure on the left bronchus, or partaking of both these characters. Diminished respiration with dulness at the base of the left lung is commonly to be observed. There may be partial or complete paraplegia from erosion of the vertebræ and pressure upon the cord. Aneurisms affecting the *descending thoracic aorta* are comparatively uncommon, and unless very large, difficult to detect. If very large,

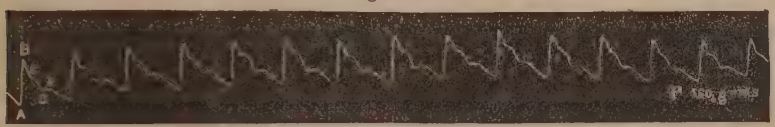
[1] Clinical Lectures. Med. Times, Feb. 1850.

[2] See a criticism of the opinions of Drs. Bellingham and Lyons and M. Guerin, in Dr. Stokes's book above quoted, p. 546.

there may be curvature of the spine, displacement of the heart forwards and to the right, local dulness and possibly pulsation. The whole side may be dull with absence of respiration, from pressure upon the bronchus and subsequent blocking of the lung by retained secretions. A bruit localized in the back is almost diagnostic of aneurism in this situation. Persistent pain is always present, and is often the only sign of the disease. Laryngeal pressure signs are not present.

It would be a matter of great practical importance to be able to say whether an aneurism affecting the innominate, subclavian, or carotid artery involved the arch of the aorta, since, if the aorta be distinctly included, the slender hope of permanent relief which might otherwise be entertained from operative procedure is still further diminished. It may also come to be of considerable importance to know whether in a given case the disease principally affecting the aorta involves one of these main vessels at its origin, as affording the chance of temporary arrest by checking circulation through that vessel. Unfortunately, the diagnosis in both these respects is in many cases beset with the greatest difficulties. There is no absolutely diagnostic sign separating aneurism of the innominate (the vessel with regard to which the question most often arises) from that of the aorta. If, however, the aneurism has its centre of pulsation or other signs below the second rib, or if it encroach upon the sternum without also presenting behind the sterno-clavicular articulation and the episternal notch, the presumption is, that the arch of the aorta is decidedly involved. A well-marked shock-sound would be also very significant of the disease being mainly aortic. We may also get some information from a comparison of the state of the pulse at the two radials; and this naturally leads us to consider the value of the sphygmograph in the diagnosis of aortic aneurism. For the purpose of showing the kind of information yielded by the use of the sphygmograph, we will take one or two tolerably well-marked cases.

Fig. 130.



Pulse in Aortic Aneurism.

In Case I., that of Eliza B——, from which the above pulse-tracing (Fig. 130) was kindly taken for me by Dr. Burdon Sanderson, there was a circumscribed aneurism of the ascending aorta, presenting its distinguishing signs—local bulging, dulness, systolic impulse, and faint diastolic bruit—at the second right intercostal space near the sternum. Here it will be seen that the systolic wave, A C D, is prolonged, occupying a greater proportion of the tracing than it should do—*i. e.*, there is a prolonged effort on the part of the left ventricle to overcome increased resistance. The impaired elasticity of the arteries is also shown by the blunted, elbow-like point at A, at the commencement of the systolic upstroke. The shock or percussion wave, A B C, of the pulse, is at the same time somewhat diminished; but as this phenomenon is general, not limited to one radial, it has no significance, since it may be produced by many other conditions; it is, however, very commonly, but by no means constantly, met with in aneurism of the main vessel. The only evidence we gain in this case, therefore, is that of the presence of arterial disease.

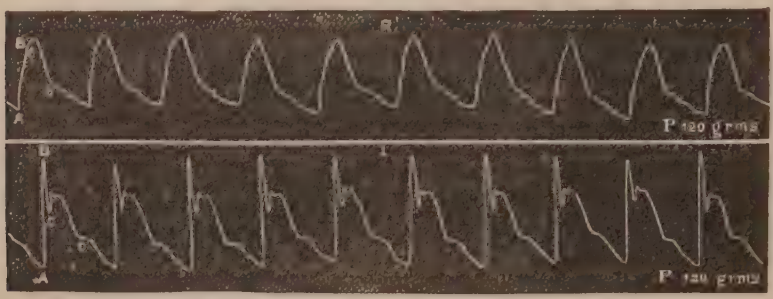
Case II., in which the tracings were also taken by Dr. Sanderson, Dr. Murchison has kindly allowed me to make use of to illustrate some further points respecting the use of the sphygmograph. (Fig. 131.) In this case the pulsations were different in the two radials.

Thomas J——, a coachman, was admitted into the Middlesex Hospital, under Dr. Murchison, in November, 1868, with a pulsating tumor in the chest, most prominent at the third right interspace, where a double bellows murmur was audible. There was scarcely any difference to be detected by the finger in the two pulses, but the right was thought to be slightly the smaller. It was for this reason only, there being no doubt about the diagnosis, that Dr. Sanderson was requested to take tracings of the two pulses.

In the left radial tracing, the lengthened systole, A C D, is the only noticeable feature; in the right, on the other hand, we have superadded to this sign of general increased resistance to circulation, others indicative of local interference with the arterial movements. The systolic vibration, or percussion wave, A B C, very marked (rather exaggerated) in the left pulse-tracing, is almost lost in the right. The dicrotism is also more distinct in the left than the right. At the same time the right pulse is not notably smaller than the left. Hence from the pulse alone we find evidence (1) of arterial dis-

ease, in the prolongation of the systole: (2) of a tumor pressing upon or dilatation of the artery, in the local effacement of vibratile impulse (percussion wave) in the right radial, without diminution in the size of the pulse.

Fig. 131.



Right and Left Radial Pulse in Aneurism of Aorta.

Had this latter phenomenon been due to extension of a coagulum, so as to partially occlude the innominate, the pulse would have been much smaller on the right side. But a saccular dilatation of the vessel, or a solid mass in contact with it, might act as an inefficient damper in destroying the vibratile shock, without affecting the size of the pulse.

In this case the aneurismal tumor invaded the thoracic parietes, and presented to the right of the sternum, but the patient died of an intercurrent attack of pneumonia. After death, the aneurism was found to be globular, as large as the two fists, and involving the whole circumference of the aorta, from its commencement to the origin of the right subclavian, which came off as a separate trunk. The aortic orifice was incompetent.

It has been observed that evidence of arterial disease coexisting with that of a tumor within the chest strongly favors the presumption that the tumor is aneurismal. Thus the intelligent use of the sphygmograph may prove in certain cases of considerable value in helping the diagnosis of aneurism, although it should be clearly understoood that alone it is nearly useless for that purpose.[1]

Another sign of aneurism, much insisted upon by some authors, is *postponement* or delay of pulse in one wrist. Strictly speaking, it is not correct to say that the pulse is postponed, for it begins and ends at the same time in the two wrists; but the sense of delay may be conveyed by the finger through the initial shock or percussion wave being obliterated on one side, while it is well marked on the other. Thus, in Case II., we can readily understand that the shock wave, A B C, constituted the pulse wave, as appreciated by the finger of the observer when applied to the left radial, while on the right side this wave was annulled, and the true systolic wave, A C D, alone appreciable. Hence the pulse on the right side might have appeared to the observer postponed (this point is, however, not entered in the notes of the case). This point of delay in the pulse, then, is, when present, of some practical value in diagnosis; it means the appreciation by the finger of the deficiency of shock wave on one side.

The distinction of aneurism of the thoracic aorta from other tumors in the same situation, from mediastinal abscess, local empyema, uncovered aorta from contracted lung disease, dilated heart, pericardial effusion, aortic valve disease, laryngitis, asthma, and angina pectoris, requires a few further remarks.

The leading features which separate aneurism from other mediastinal tumors have already been incidentally referred to in speaking of the value of the individual signs present in aneurism, and have been more specially considered in the diagnosis of mediastinal tumors. We may, however, briefly mention a few other considerations of some importance to bear in mind in cases of difficulty, as affording, when taken in association with the signs of tumor, additional evidence for or against aneurism.

1. If the age of the patient be under twenty-five, in the absence of any history of direct injury, the chances are against aneurism.

2. Great emaciation in the absence of intense prostrating pain, is against aneurism.

3. Great displacement of heart, in the absence of marked signs of a large pulsating tumor, is against aneurism.

4. Female sex of the patient is against aneurism.

5. On the other hand, severe pain, con-

[1] In the Med. Times and Gazette for 1873, pp. 141 and 122, will be found valuable original papers, by Dr. F. A. Mahomed, on the use of the sphygmograph in the diagnosis of aneurism.

stant, with occasional exacerbation, is in favor of aneurism.

6. The more inconstant the distal signs of pressure—unequal pulse, irregular pupils, laryngeal and bronchial dyspnœa, dysphagia—the greater the probability of the disease being aneurismal.

7. Dr. Walshe observes that "the absence of symptoms and signs, indicative of ordinary affections of the heart and lungs, in an individual suffering from persistent anomalous disturbances within the chest, even though he does not, or rather because he does not, exhibit any failure of general health, affords strong motive for suspecting aneurism." In the diagnosis of pulsating empyema from aneurism, very rarely required, the presence of fluid impulse without bruit or shock sound, the extension probably of the effusion beyond the limits of an aneurism, the pointing of the abscess, and the presence of irritative fever, would be the distinguishing signs.

Cases not infrequently occur of diseases affecting the apex of one lung, causing the retraction of its margin away from the base of the heart and aorta. Such cases present many of the signs of aneurism—dulness, forcible local pulsation, sometimes a bruit, with palpitation and dyspnœa.[1] The history of such a case, the evidence of distinct disease of the lung, and of enlargement of the opposite lung, with elevation rather than depression of the heart's apex, and the absence of other pressure signs, will usually prevent error in diagnosis.

In some cases, aneurism of the descending aorta, compressing the left bronchus, and leading to collapse of the lung, or to its blocking by catarrhal products, becomes so masked by the secondary affection, and from the depth of the tumor in the mediastinum, as to make the diagnosis from chronic pneumonia or pleurisy most difficult. In such cases, however, the urgency of the dyspnœa, especially on excitement, and the deep-seated pain and palpitation, will usually awaken at least grave suspicion in the right direction.

Dr. Hope lays some emphasis on the possibility of aortic regurgitant disease being mistaken for aneurism. The symmetrical pulsation of all the vessels and the seat of the murmur will usually prevent such an error. Dilatation of the commencement of the aorta is not unfrequently occasioned, however, by regurgitant disease, and the locomotion of the vessel being always increased, the signs of the dilatation are thereby much exaggerated.

Laryngeal symptoms are apt to be present in the most obscure forms of aneurism, projecting deeply from the back of the arch. Such cases may be mistaken for laryngitis; but the inconstancy and paroxysmal character of the symptoms, so rare in adult laryngeal affections, should at once arouse suspicions, and laryngoscopic examination with a minute exploration of the chest will then usually solve the difficulty.

PROGNOSIS.—In aneurism of the aorta, the prognosis is within a brief and uncertain space of time fatal. It is the uncertainty as to the time of death, and the suddenness with which it may at any time occur, that gives to this disease its peculiar terror. It is this peculiarity, moreover, which should make us so extremely careful not to make a wrong diagnosis; and when there is any doubt, as there often must be, we should give the patient or his friends only a sufficient insight as to his critical condition to insure the due settlement of his affairs.

Duration.—From six months to four years from the time of detection of the aneurism (Lebert).[1] This limit has been extended in certain cases. The main characteristic of the duration of aneurism, however, is its uncertainty. The aneurismal wall may be obviously thickening by fibrinous deposit at one point while fatal erosion is taking place at another.

Mode of Death.—Rupture is by far the most common termination of aneurism, and the more latent the aneurism, the more uniformly does death occur in this way. Dr. Sibson[2] remarks, "It will be observed throughout, that the greater the proportion of ruptures in any group of aneurisms of the aorta, the smaller is the proportion of instances in which the disease is indicated by symptoms during life, and *vice versâ*." The rupture may take place into the pericardium, left or right pleura, bronchi or lungs, trachea or œsophagus, or externally or more rarely into the vena cava, right auricle, &c. Dissecting aneurism, most commonly affecting the first portion of the aorta, almost invariably bursts into the pericardium. Circumscribed aneurism of the ascending aorta most commonly also gives way into the pericardium, of the transverse aorta into the trachea or œsophagus, of the descending into the left pleura, left bronchus, or œsophagus, or into the abdomen. A certain small number of patients die of exhaustion, fewer still of syncope or as-

[1] I have met with several cases of the kind which might readily have been mistaken for aneurism; and in one case the error had been actually made, and the diagnosis of aneurism communicated to the patient by an observer of some experience.

[1] Loc. cit. Analysis of 30 cases.

[2] The Aorta and the Aneurisms of the Aorta.

phyxia (apart from hemorrhage), or from acute intercurrent disease.[1]

Treatment.—The objects we have in view in the treatment of thoracic aneurism are, firstly, to diminish the strain upon the injured vessel as much as possible; secondly, to encourage the deposition of fibrine within the aneurism. We have further, by the administration of anodyne remedies internally or locally, to lessen suffering. In carrying out these objects, rest as far as is possible for the circulation is of the first importance, and the conditions most necessary to secure this rest to the circulation, are strict muscular repose, mental quietude, and regulated diet. It requires much intelligence on the part of the patient, and tact on that of the physician, to maintain these conditions for a sufficient length of time to be of service, even when surrounding circumstances are favorable.

The recumbent posture, or that most nearly approaching it and yet comfortable to the patient, should be preserved, all excitement avoided, and the nutrition maintained by a diet restricted in quantity, but enriched in quality. Blood rich in nutritive elements more readily deposits fibrine, and favors those efforts at repair which result in a welding together of the fibrinous layers nearest the arterial wall, while, mechanically speaking, it has no more pressure effect upon the weakened vessel than blood deficient in these elements. On the contrary, we daily find that whenever an anæmic condition is present the circulation is hurried and more easily disturbed. Evidence of a rapid softening down of the laminæ which had almost blocked an aneurism may sometimes be found *post mortem* in those who have subsequently to careful treatment again been subjected to the debilitating circumstances of a life of poverty. Fluids must be only very moderately partaken of; milk, soft puddings, eggs, and a moderate quantity of meat may be allowed. Mr. Tufnell,[2] whose treatment of large internal aneurisms has been remarkably successful, restricts the food taken to 2 oz. of bread-and-butter and 2 oz. of new milk for breakfast; 2 or 3 oz. of bread and 2 or 3 oz. of meat for dinner with 2 oz. to 4 oz. of milk or claret; and 2 oz. of bread-and-butter with 2 oz. of milk for supper. In his first recorded case he maintained this treatment, combined with absolute rest, for nearly two months with complete success. Mr. Tufnell would of course allow a certain slight variation from this diet measure to suit individual cases: his treatment is moreover only applicable to cases of sacculated aneurism. Dr. Sibson[1] also advocates the regulation of the diet in conjunction with rest, but is content to limit the quantity of fluid taken *per diem* to within one pint. Let me repeat, rest in the recumbent posture is of the utmost importance to the success of this treatment; Mr. Tufnell reckons that in some of his cases this alone lessened the number of distensions of the aneurismal sac by more than 20,000 a day![2] Stimulants, or I should rather say stimulation, must be absolutely interdicted. During this treatment too frequent examinations are to be avoided, but the circulation and the aneurismal impulse, when within our reach, should be carefully watched. Certain drugs—chloral, opium, digitalis, veratria, and aconite—are useful to allay excitement of circulation. They are none of them efficacious without rest, but they, and particularly chloral and opium, may help in diminishing the restlessness and impatience of persons naturally irritable who have great difficulty in submitting to the treatment by diet and recumbency. Belladonna applications may be employed for the purpose of relieving pain, but when this is acute the subcutaneous injection of morphia is the best remedy. The continuous application of an ice bag, suspended from above the patient, to the tumor when prominent externally will often greatly relieve pain, reduce inflammation, and perhaps even help to promote consolidation within the sac. Gentle laxatives or saline purgatives must be given to prevent any effort in relieving the bowels. When, as sometimes happens on the patient first coming under notice, there is any undue fulness of vessels present, free purgation with salines will be attended with marked relief up to a certain point. In cases of urgent dyspnœa with engorgement of vessels, vene-

[1] See on this point the statistics of Dr. Crisp, Dr. Sibson, and Professor Lebert. The above general remarks are more especially founded upon the very careful and complete Tables of Dr. Sibson—584 cases. Lebert's statistics are on this head less trustworthy, the numbers being much smaller; thus of 41 cases he finds 4 rupture externally, Drs. Crisp and Sibson finding this termination in about 4 per cent. Lebert also gives no cases of rupture into œsophagus, whereas this is rather a frequent termination of cases of aneurism—of transverse and descending aorta—about 5 per cent. (Sibson).

[2] Thoracic Aneurism successfully treated by restricted Diet and the application of Ice. Dublin Hospital Gazette, January, 1858; also on the Treatment of Thoracic and Abdominal Aneurisms, by J. Tufnell, F.R.C.S.I., Army Med. Rep. for 1862, and Medico-Chir. Transactions for 1874.

[1] Croonian Lectures, Lancet, 1870.

[2] See an able review of the modern treatment of aneurism in the Medical Times and Gazette for December 20, 1873.

section should be promptly employed; repeated blood-letting after the manner recommended by Valsalva[1] in the last century, combined with the lowest possible diet, is not likely to find favor in the present day.

Various internal remedies have been administered with the view of favoring the formation of coagulum within the aneurism, either by rendering the blood less watery—*e. g.*, saline purgatives, diuretics; or by affecting it or the aneurismal wall in some specific way—*e. g.*, acetate of lead, iodide of potassium, ergot. The free administration of iodide of potassium in aneurism has been pretty extensively tried. It seems to have been first introduced by Nelaton[2] in 1859, and subsequently tried by Drs. Bouillaud,[3] Chuckerbutty[4] of Calcutta, Roberts[5] of Manchester, and Balfour[6] of Edinburgh; and the concurrent testimony of these several observers is very favorable to the drug as a valuable agent in the treatment of this disease. Dr. Balfour holds that "no treatment for aneurism, and especially for internal aneurism, holds out anything like an equal prospect of relief, if not of cure, with that by the iodide of potassium." The drug must be given in large doses (gr. xx) and continued for many months. Its mode of action is unknown; Dr. Balfour[7] thinks that it is not by increasing the coagulability of the blood, but by its sedative action upon the heart and "by some peculiar action on the fibrous tissue, by which the contraction of the sac is aided and its walls are strengthened and condensed."

Langenbeck[8] having observed the great value of ergot, particularly when used subcutaneously as ergotin in arresting hemorrhage, tried the drug in the same way in aneurism, and with considerable success in a case of the disease affecting the innominate and subclavian trunks. He injected ½ gr. of the watery extract, increasing to 3 grs., every three days. It is difficult to say how far in this, as in other instances of the reputed efficacy of drugs in the treatment of circulatory diseases, the attendant rest may have aided in producing the amelioration of symptoms.

Rest and recumbency, with the aid of drugs, failing or being impracticable for the obliteration of aortic aneurism, the question arises, whether any operative measure can be adopted to control the circulation through the sac so as to favor its gradual obliteration by laminated clot. M. Velpeau,[1] in 1839, seems to have been the first to entertain the question of ligature of one or more of the main branches of the aorta for the cure of aneurism, and this treatment has been most recently advocated by Dr. Cockle[2] and practised by Mr. Christopher Heath.[3] The exact value of this method of treatment, and the rules which should guide us in adopting it, cannot be regarded as yet thoroughly ascertained. In cases in which one of the great vessels—most commonly the innominate—proceeding from the arch is largely involved, or in which the aneurism is at least extending in the direction of one of these main branches while there is no evidence of extensive arterial degeneration, the distal ligature may be most appropriately tried, the principle of the ligature being to greatly enfeeble the current through the sac by arresting the circulation through the branch principally involved or most nearly proceeding from the aneurism. Ligature of the right carotid or subclavian, or both, or of the left carotid, must be decided upon according to the direction of the growth of the aneurism. Distal pressure upon the great vessels of the neck has been employed, and though most difficult to effectually carry out, is yet, perhaps, worthy of a further trial.

The common object of the different kinds of treatment we have hitherto considered, viz., rest, diet, compression, and ligature, has been that of encouraging spontaneous coagulation within the aneurismal sac, whether by lowering the circulation generally, or by locally arresting it entirely, or lessening its force or altering its direction.

Mr. Moore[4] in 1864 proposed a new

[1] *Vide* Observations on Aneurism, by Mr. Erichsen, Syd. Soc. 1844, pp. 239 and 261.

[2] Clinique Européenne, July, 1859.

[3] Idem, August, 1859.

[4] Iodine of Potassium in the Treatment of Aneurism. Brit. Med. Journal, July, 1862.

[5] Clinical Lecture on the successful use of Iodide of Potassium in the Treatment of Aneurism. Idem, January, 1863.

[6] On the Treatment of Aneurism by Iodide of Potassium. Edinb. Med. Journal, July, 1868.

[7] Idem, January, 1874, p. 645.

[8] Idem, November, 1869. Abstract from the Berliner klinische Wochenschrift, March, 1869.

[1] For the most modern authoritative information on the surgical treatment of aneurism, *vide* Lectures delivered by Mr. Holmes, at the College of Surgeons, 1872. Lancet, July, 1872, lect. ii.

[2] Further Contributions to the Pathology and Treatment of Aneurismal Tumors of the Neck and Chest, by John Cockle, M.D. Lancet, 1869, pp. 422 and 489. See also Clinical Transactions for 1872.

[3] On the Treatment of Intra-thoracic Aneurism by the Distal Ligature, by Christopher Heath, F.R.C.S., 1871.

[4] On a New Method of procuring the Consolidation of Fibrine in certain Incurable Aneurisms. Med.-Chir. Trans. vol. lvii. p. 129 et seq.

treatment of aneurism, by the insertion into the sac of a foreign body (fine iron wire) to act as a nucleus for rapid and firm coagulation. The result in the case to which he applied this treatment—a case of aneurism of the thoracic aorta rapidly perforating the chest-wall—was discouraging, inflammation of the sac, embolism of distant vessels, and great increase in the sufferings of the patient being occasioned. The same idea has since, however, found favor among a few bold operators, who have severally tried needle punctures, the introduction of carbolized catgut, watch-spring (14 inches!), and iron wire, with uniformly deplorable results. The latest and most rational method of the kind adopted is that of Dr. Levis of Philadelphia,[1] who has introduced through a capillary trocar a great length of fine horsehair into the sac of a subclavian aneurism. I think it must be allowed, however, that this mode of treatment is wrong in principle, and must, therefore, in the majority of instances fail in practice. If direct coagulation of blood within the sac be aimed at, it is only thus accomplished in a dangerous and imperfect manner, the clot formed being subdivided and soft, and entangled round a foreign body in the centre of the sac instead of being laminated and firm, and deposited around its circumference; hence danger of inflammation of the sac, of capillary or larger embolisms, and of blood-poisoning from rapid disintegration of the clot. If the object be to determine coagulation within the sac by setting up a certain degree of inflammatory action, or if the fear of inflammation resulting from these procedures be disregarded on this ground, the practice must equally be condemned as dangerous, if not unjustifiable, for we have no means of controlling the inflammation when induced in the main blood-channel. It must further be borne in mind that by producing suddenly coagulation within an aneurism without that local or general lowering of the circulation which would favor its natural occurrence, we necessarily run great risk of causing rapid extension of the disease in some other, perhaps more fatal, direction. These objections, it must be confessed, have, too, a certain force when applied to the somewhat less modern practice of galvano-puncture, which has recently, in common with all other methods of treating this deadly malady, been very keenly discussed and tested. There is this striking advantage in galvano-puncture, however, that no foreign body is allowed to remain within the aneurism. It has, moreover, been tried with success now in a few instances.[1]

The idea of employing electricity in the treatment of aneurism seems to have sprung from the physiological experiments of Scudamore 1824, Müller 1832, and Ansell 1839, and to have been first applied in practice by M. Pétrequin in 1841.[2] It was Ciniselli, however, who, in 1846, first employed electro-puncture in the treatment of aortic aneurism,[3] and the first definite principles on which to proceed in the employment of this agent were laid down still later by MM. Baumgarten and Wertheimer in 1852,[4] as the result of extended experimental inquiry. The experiments of these gentlemen showed (*a*) that coagulation might be with certainty induced at any point within a large vessel of an animal during life by electrolysis, (*b*) that this coagulation was most firmly[5] affected around the positive needle when both were introduced: (*c*) that the best way, however, of producing coagulation was to insert the positive needle only, applying the negative element by means of a moistened sponge or metallic conductor placed on the external surface.

Dr. Fraser, in 1867, confirmed and added to these conclusions, observing that, whereas at the positive needle a firm and comparatively small clot was formed, at the negative needle, on the other hand, the coagulum was large and frothy. Dr. John Duncan[6] is, however, of opinion that the needles connected with both poles should be introduced, and in this view he agrees with Ciniselli.[7] Dr.

[1] Referred to by Mr. Holmes, in London Medical Record, December 17, 1873.

[1] Of 13 cases treated by Ciniselli's method, between July, 1868, and July, 1870, 6 were "cured." Of them 3 relapsed after 17, 3, and 4 months respectively; the latter case, however, after a second operation, being again "cured" and remaining so, after 8 months. For details of 23 cases, and for a description of Ciniselli's method of employing electro-puncture, see his paper in the Annali Universali di Medicina for November, 1870, p. 292 et seq., Table, p. 625. An abstract of this paper is given in the Jahresbericht for 1870, Bd. ii. s. 109.

[2] *Vide* Essay by Dr. Fraser, of Edinb., on the Action of Galvanism on Blood and on Albuminous Fluids, 1867.

[3] Case related in the Annali Universali di Medicina, 1870, p. 294, and referred to with 22 others in a Table at p. 625.

[4] Gazette des Hôpitaux, June 19, 1852.

[5] Confirming a previous observation by Prof. Schuh, Vierteljahrschrift für die praktische Heilkunde, Bd. i. 1851.

[6] On the Surgical Applications of Electricity, Edinb. Med. Journal, 1872, 506 et seq.

[7] In this view also Mr. Marcus Beck concurs: and the comparative effect upon an albuminous fluid of inserting only one or both

Althaus,[1] on the other hand, regards the negative pole as the one to the use of which "we have to look for the cure of aneurism."

As to the mode in which electricity effects coagulation within an aneurismal sac, the question whether it may be by the mere passage of the electrical current through the blood, is set at rest by the observation of Dr. Fraser, that although the two needles be separated by an interval of blood through which the current must pass, yet coagulation takes place only around each needle separately. A certain amount of inflammation is often set up in and about the sac by this agent, which no doubt, if it do not proceed to a dangerous extent, may help to promote consolidation; this inflammation is, however, shown by Drs. Duncan and Fraser to be not essential to coagulation, but, on the contrary, to be guarded against as one of the chief sources of danger. The coagulation is produced in truth, as pointed out by Steinlein[2] and confirmed by others, by (*a*) electrolytic decomposition of the salts of the blood which are mainly instrumental in maintaining its fluid state; (*b*) by the acid elements set free at the positive pole directly causing coagulation there, or combining with the oxidized metal to form salts which precipitate the albumen. This latter action may be increased by coating the positive needle with some more oxidizable metal, such as zinc.

In the writings of Dr. John Duncan, Dr. Fraser, Mr. Holmes,[3] and Prof. Ciniselli already quoted, will be found recorded the clinical and physiological experiences which furnish us with all the knowledge we at present possess on the subject. The valuable records of individual cases of galvano-puncture in aneurism which are gradually collecting are as yet too few to admit of classification. So far as they have gone, however, they do not encourage us to anticipate a very favorable opinion of the operation, for of nine cases of aortic aneurism thus treated of which accounts have appeared in the English journals within the last two years, in only one, that of Dr. McCall Anderson's, has a decidedly good result been obtained, although in one or two other instances, in Drs. Ralfe and Johnson's case, and in a case of Dr. Bastian's, perforation of the chest wall has been for a time averted. In other instances, however, this perforation has undoubtedly been hastened by the needle punctures. Appropriate cases in which the operation has been undertaken with the view of curing the aneurism must be classified apart from those in which the puncture is made with the view of temporarily stanching an aneurism already diffused and threatening to burst through the surface, before we can hope to obtain reliable statistics upon which to decide as to the true value of this treatment: moreover we certainly have not yet arrived at the method of operating agreed on all hands as the best. Meanwhile the following appear to be the most important points respecting the operation which the physician should bear in mind:—

1. (*a*) The cases of thoracic aneurism which seem most suitable for treatment by galvano-puncture *with a view to permanent relief* are those in which the dilatation is presumably sacculated, near to the surface, and advancing outwards, the sac being as yet entire; (*b*) the operation may sometimes be performed as a palliative measure to retard rupture through the surface, or to relieve suffering.

2. The treatment by rest, with the aid of restricted diet and appropriate drugs, must have first been fairly tried and proved useless or impracticable before any operative procedure is justifiable, and the same absolute rest and careful diet must be maintained throughout the operation and the subsequent treatment.

3. It is best not to freeze the surface before puncture, although its sensibility may be deadened by cold. Sometimes the operation may be usefully preceded by the administration of a dose of morphia subcutaneously to enable the patient to remain without suffering in one position during its performance.

4. The battery used should be of many (10 to 30) cells with plates of small surface. By diminishing the size of the plates and increasing the number of the cells used, a given electrolytic power is obtained with a less intensity of heat than with fewer cells and larger plates.[1]

needles is well illustrated by a simple experiment made by Mr. Beck and Dr. Poore. Into some white of egg held in the hollow of the hand the positive needle connected with twenty cells of Weiss's battery was introduced, a sponge connected with the negative pole being applied to the back of the hand. After five minutes no action upon the albumen had been effected. Both needles were then introduced together into the albumen, and in two minutes a firm coagulum of the size of an oat surrounded the positive needle-point, and a frothy mass as large as a pea was found at the negative needle. Lancet, 1873, p. 550.

[1] Medical Electricity, 2d edit. 1870, p. 607.

[2] Galvanopunctur bei Varicositäten und Aneurysmen—Zeitschrift der k. k. Gesellschaft der Aerzte zu Wien, 1853. Quoted at p. 471 of Dr. Hammond's translation of "Meyer's Electricity in its Relations to Practical Medicine."

[3] Lancet, 1872, vol. ii. pp. 336 and 663.

[1] Foveau's improvement of Weiss's instrument is now considered the best. Dr. Poore has, however, electrolyzed white of egg by

5. The needles connected with both the positive and negative poles and of the dimensions of medium-sized harelip pins, insulated with vulcanite to within a certain distance of their points (according to the size of the aneurism), should be thrust vertically into the aneurism so as to avoid scratching or puncturing its inner surface at any other point.

6. The electrolysis should be continued for twenty minutes or half an hour, or until some decided alteration in the pulsation or bruit of the aneurism has been produced. The withdrawal of the needle must be effected with the utmost caution, to avoid the loosening of the clot should it happily have become adherent to the wall of the aneurism. A second and a third employment of the agent may be made at intervals of several days.

The question of *tracheotomy* occasionally comes before us in cases of thoracic aneurism, in which death is threatened from paroxysmal dyspnœa. If we accept the views lately advocated by Dr. Bristowe, that not only the constant, but even the paroxysmal dyspnœa is in the majority of cases due to direct pressure upon the trachea, we can expect no relief from this operation. If, however, the view that the paroxysms of dyspnœa may be occasioned either by irritation or destruction of the recurrent nerves be still tenable, we may expect to prolong life and to render death less terrible by its performance. There have been cases in which the operation has distinctly though only temporarily saved life.

Spontaneous Rupture of the Aorta.

Spontaneous rupture of the aorta is most commonly the result of antecedent disease of the coats of the vessel, more particularly of the senile atheromatous kind. It may occur, however, in consequence of stenosis of the vessel (Rokitansky), though such cases are necessarily very rare. Even in such cases, too, the proximate cause of the rupture is disease of the vessel wall, either abnormal delicacy and thinness (Rokitansky) or atheroma the result of heightened blood pressure. Rupture of the aorta most commonly takes place at or near its commencement (Broca, Peacock). It may at once extend through all the coats of the vessel, causing immediate death from hemorrhage, or, as is more commonly the case, it extends through the internal to the middle coat, and the effused blood tearing apart the layers of this coat, or separating it from the external for a greater or less distance, a *dissecting aneurism* arises. The coats of the vessel may be thus dissected or separated apart throughout the whole length of the aorta, and even along some of its main branches. Dr. Todd[1] relates a case where the separation extended from half an inch beyond the valves to the abdominal aorta and along the innominate and right carotid arteries, causing drowsiness and partial left hemiplegia by compressing the canal of the last-named vessel.

There is frequently no discoverable exciting cause of the rupture; in a few cases it has been traceable to great mental emotion, or to cardiac excitement from overdistension of the stomach; external violence, as the shock of a blow or fall, may also give rise to it. Rupture of the aorta may occur at any age after thirty. Dissecting aneurism occurs mostly in advanced age; it is about equally common in both sexes.[2] There are no special symptoms attributable to rupture of the aorta save those of fatal syncope. In cases, however, in which the rupture is incomplete and a dissecting aneurism occurs, the symptoms are, acute rending pain in the præcordial region extending to the left shoulder and spine, severe cardiac dyspnœa, orthopnœa, with profound shock to the system, pallor of countenance, great anxiety, and feeble, irregular pulse. The rupture of the external coat may quickly follow that of the inner, and death immediately ensue, or the patient may rally for a few hours, or even days, before a second attack proves fatal. In some rare cases, in which the aneurism becomes circumscribed, its future course may closely resemble that of other circumscribed aneurisms. The treatment of aortic rupture or dissecting aneurism is, of course, merely palliative.

Narrowing of the Aorta.

In certain rare instances there is a congenital deficiency in the calibre of the whole aortic system. This general nar-

employing a Pulvermacher's chain of fifty-eight elements moistened with dilute sulphuric acid (one part to thirty); and he succeeded in producing in an hour a firm clot at the positive electrode three-quarters of an inch long, of the diameter of a goose-quill and weighing nine grains. Dr. Poore suggests, as well worth a trial, the employment of a similar battery in the treatment of aneurism, and thinks it possible that patients might be able to bear the application of a current from such a battery for far longer periods than when elements of larger surface are made use of. This is, of course, a question only to be decided by direct experiment. *Vide* the book now published.

[1] Med.-Chir. Trans. vol. xxvii. 1844.

[2] Rokitansksy's cases show a slight preponderance on the male side; in Dr. Peacock's it was slightly more frequent among females.

rowing usually affects mainly the descending aorta, particularly the descending portions of the arch. It is most common in females, and is often overlooked, being attended with no marked symptoms until the period of puberty, when the insufficient general development, with marked deficiency in the sexual system, become apparent. The only physical signs present are those of hypertrophy and dilatation of the left ventricle, and smallness of pulsation in the abdominal and iliac arteries.

A more common form of congenital narrowing of the aorta, is that in which the constriction is limited to that portion of the arch beyond the subclavian artery, either at or a little above or below the *ductus Botalli*. The term "coarctation of the arch of the aorta" is often applied to this condition. The constriction rarely occupies more than half an inch of the length of the vessel. It may be ring-like, as though the vessel had been tied by a moderately thick piece of string, or it may be caused by a fold, more or less deeply projecting into the vessel, or by a scar-like contraction corresponding with the position of the duct; or again, the vessel at this point may be converted into a thickened, impermeable cord. The degree of constriction varies from complete closure to a diameter of three or four lines, or a mere narrowing. Only in five cases out of forty collected by Dr. Peacock was the obliteration complete. The walls of the aorta at the seat of the contraction may be natural or thickened or thinned. The *ductus arteriosus* is usually closed, but in some cases it is open for part of its extent, in others it is more or less pervious throughout; it is affected in one of these ways in about one-sixth of the cases. Above the constriction the aorta is as a rule widened, sometimes greatly dilated; it may, however, be of natural dimensions; below the constriction, it is either of normal size, or more frequently somewhat diminished: occasionally, however, it is even widened, and in one case quoted by Dr. Peacock, an aneurism was found immediately below the stricture. A deficiency in the number of aortic valves has been found in one-eighth of the cases, and other cardiac malformations have been mentioned. The great branches of the arch are always enlarged, and by the communication of some of the branches derived from them (the transversalis colli, superior intercostal, internal mammary), with corresponding branches from the descending aorta (intercostal, epigastric), a tolerably free collateral circulation is maintained. Hypertrophy of the left ventricle is a necessary consequence, and is often attended with dilatation and with, no doubt, secondary dilatation and hypertrophy of the right cavities of the heart.

This affection is three times more frequent in the male than the female sex. In almost every case the deformity is either congenital, or acquired in the first few days of infant life: death however may occur at any age, usually but not always preceded by symptoms of heart disease. There are several theories to account for the occurrence of this deformity of the aorta, of which three are admitted by different authors as applicable to certain cases:—

1. Although the normal process of closure of the *ductus arteriosus*, which occurs within the first week or ten days of life, is simply one of gradual withering and contraction, yet in some instances it is delayed by the formation within the duct of a fibrinous coagulum, which may extend into the aorta and completely occlude it at the point corresponding with the entry of the duct. As the clot subsequently becomes gradually absorbed, the walls of the aorta and those of the duct contract upon it to their complete obliteration. Foerster adopts this theory as applicable to those cases in which both the duct and the aorta are completely closed.

2. In other cases, however, the coagulum may not extend beyond the duct, the walls of which, thickened from the irritation attendant upon the presence of the clot, contract upon it as it becomes absorbed, puckering the adjacent walls of the aorta in an irregular and scar-like manner, so as partially to constrict the vessel.

3. A more generally applicable explanation is, that this deformity is a partial preservation of the fœtal condition by which the aorta conveys blood to the head and upper extremities, and the pulmonary artery, continuous through the duct with the descending aorta, supplies the lower extremities and abdominal viscera. It is the view of Reynaud and Rokitansky, and is adopted by Dr. Peacock and most modern authors, and may be described as follows:—

At the termination of fœtal life, with the expansion of the lungs the blood-stream is diverted from the *ductus arteriosus* through the two branches of the pulmonary artery. The blood thus diverted returns to the left ventricle and increases the volume to be transmitted through the first and second portions of the aorta by the amount destined for the supply of the lower half of the body. In the normal condition of things the isthmus of the aorta—that portion connecting the great brachio-cephalic trunk with the *ductus arteriosus* as it joins the descending aorta—now rapidly expands so as to become a part of the main arterial channel, while the starved ductus dwindles. If, however, as sometimes happens, whether from defective nutrition, rigidity from inflamma-

tory thickening, or any other cause, this *isthmus* fails to widen, the increased volume of blood finds exit through collateral channels into the descending aorta, the branches of the arch expand, and their twigs, communicating with branches from the descending trunk, enlarge so as to supply to it the blood required for the lower limbs and viscera. Thus far the process takes places in the first days of extra-uterine life, but the narrowed portion of the aorta, having the extra pressure of blood thus removed from it, still further atrophies from disuse, and in course of time may become quite closed, although its channel is probably in these cases never completely obliterated. An increased muscular power of heart is required to compensate for the increased resistance to circulation necessitated by the transmission of the blood through circuitous and divided routes instead of by one short and broad channel to the descending trunk.

It has already been stated, that this defect in the aorta is not necessarily attended with any symptoms, and that when they arise they are those of lesion secondary to the aortic deformity, of hypertrophy and dilatation of the heart, rupture of the heart or aorta, or great dilatation of the main vessel above its narrowed portion. It is unnecessary to refer to these symptoms in detail.

Diagnosis. — The diagnosis has not often been made, but when attention is directed to such a case the physical signs pointing to the constriction and its situation, though few, are tolerably significant. They are those of hypertrophy of the heart, most distinctly of the left ventricle, usually attended with some dilatation, throbbing of the great vessels of the neck and upper extremities; while arteries not usually visible, the transverse cervical, and thyroid, and the small anastomosing vessels at the margin of the sternum and epigastrium are enlarged, tortuous, and pulsating. With this activity of circulation in the upper half of the body is contrasted the febrile pulsation of the abdominal aorta, iliac and femoral arteries, all pulsation in the popliteal and the tibial vessels being often absent. A systolic, or rather post-systolic, murmur may be heard over the aorta and also over the enlarged vessels. The presence of aneurism or of some other mediastinal tumor pressing upon the aorta will be excluded by the absence of any other signs of pressure upon the nerves, or food or air tubes, or upon the lungs.

Prognosis.—In a considerable proportion of cases death occurs from some lesion apparently altogether unconnected with the aortic deformity, eleven cases out of the forty referred to by Dr. Peacock having died from cerebral disease, bronchitis, pneumonia, &c. But the circulation is carried on at high pressure, at least as regards the heart and first portions of the aorta, and sudden death, from syncope or rupture of the heart or aorta, occurred in eight of the forty cases. In the rest, the heart in time gives way under its excessive toil, degeneration and dilatation succeed to hypertrophy, and death slowly occurs in the way usual to such affections. Supposing, therefore, that the diagnosis be made while the patient as yet appears to be in fair health, there is a very fair chance that if, bearing in mind the very mechanical nature of the result of this malformation, he be warned against such exercises as increase the circulation, and encouraged in calm intellectual pursuits and a sedentary profession or business, he may possibly live to the average period of life. The danger of sudden death should undoubtedly be mentioned to his friends, or to the patient himself. There is, of course, no special treatment other than "expectant" to be adopted, but this palliative treatment is of the greatest importance.

The most concise account of this affection of the aorta is contained in an original article by Dr. Peacock in the "British and Foreign Medico-Chirurgical Review" for April, 1870. Dr. Peacock gives a brief abstract of all the cases recorded up to that time, with full references to the authors.

Rokitansky is the principal authority on the subject. "Path. Anatomy," vol. iv., and his work "Ueber einige der wichtigsten Krankheiten der Arterien," 1852.

See also section on "Coarctation of the Aorta" in Dr. Walshe's work on "Diseases of the Heart."

In Foerster's "Handbuch der Pathologischen Anatomie," p. 726, will be found a brief description, with references to fifty-two cases by different authors.

ANEURISM OF THE ABDOMINAL AORTA.

By Dr. William Murray, F.R.C.P. Lond.

The pathological anatomy of Aneurism, the degeneration of arteries, and the process of erosion in bony structures, are subjects too general to be handled in an article on the diseases of a single bloodvessel: the following remarks will not therefore be so extended as to embrace those general conditions to which the aorta is subject in common with the whole arterial system. These will be alluded to when they present features peculiar to the abdominal aorta; but the discussion of the several diseases to which this lower half of the vessel is subject will chiefly engage our attention. The latter afford so deep a study in pathology, diagnosis, and treatment, that a distinct treatise might well be written on them, especially as they often involve excruciating agony to the sufferer, and have in most cases a fatal issue.

The Anatomical Characters of the Disease.—Atheromatous, calcareous, or ossific (Virchow) changes in the coats of the aorta are doubtless the chief predisposing causes of Aneurism; they rob the vessel of its elasticity by destroying its middle coat, where resides the tissue on which the dilatation of the artery and its subsequent recoil depend, and thus they lead either to a permanently dilated condition of all the coats of the vessel (at the diseased spot), constituting a *true Aneurism*, or the degenerated coats give way and a *false Aneurism* is produced. In addition to this source of origin, a tolerably healthy aorta may be ruptured in its middle or internal coats, and thenceforth an aneurismal pouch may be formed at the seat of the accident; and this fact is worthy of remembrance, because an Abdominal Aneurism arising from muscular exertion or external violence may thus occur in a healthy vessel, and be subjected to successful treatment, whereas the occurrence of the disease apart from straining or violence, points strongly to a diseased condition of the vessel, and augurs ill for the future course of the disease. It is also important to remember that Abdominal Aneurism is much less often complicated by valvular disease of the heart or extensive arterial disease, than is found to be the case in Aneurism of the thoracic aorta and its branches.

Simple dilatation of the abdominal aorta is rare, and the cases of true Aneurism bear a small proportion to the cases of false Aneurism in this vessel. Dr. Sibson's tables show 60 per cent. of Aneurisms in the abdominal aorta to have been sacculated, and 10 per cent. non-sacculated; as we may take it for granted that almost all the sacculated Aneurisms were false, and that some of the non-sacculated were also false, we see how small the number of true Aneurisms in this situation becomes. The opening into the Aneurism may be at any part of the circumference of the aorta; in sacculated aneurisms it is as frequently on the anterior as on the posterior aspect of the vessel, and it may be exceedingly small, or as extensive as the size of the vessel will allow; in one case the whole of the posterior aspect of the vessel had disappeared. The coats of the artery in true Aneurism are, of course, continuous throughout; but when, as does occur, the true Aneurism is sacculated, the inner coats become extremely delicate, and the external coat becomes thick and strong. If the inner coats become thickened they are never thereby increased in strength or consistency. In the cases I have examined, when the Aneurism was sacculated and of moderate size, the internal and middle coats were prolonged but a short distance into the sac; here they became soft and pultaceous, or rough and adherent to the fibrine or other contents of the sac, and, on tracing them throughout the sac, fragments were here and there discernible in patches. The sac may be empty, or merely lined by a thin layer of fibrine, when the Aneurism is small and communicates with the artery by a large orifice so as to permit a free current of blood through the cavity of the sac. In other cases, where the current has not been so free, concentric laminæ of fibrine are found, tough and old, immediately beneath the external wall, but softer and stained with blood towards the interior of the sac. The sac may contain coagulated blood in quantities varying according to circumstances, and some have observed a distinct vascularity in the external layers of fibrine. I would lay particular stress upon the presence of *laminated fibrine and coagulated blood* in these Aneurisms, because one or other of these

in any given case is the chief factor in curing the disease; they are therefore to be looked upon as the result of nature's unaided attempts to provide a means of cure. In some rare instances the Aneurism has been formed by a hernial pouch of the internal coats protruding through an aperture in the external coat; in other instances the blood finds its way between the internal and external coats, and thus forms *a dissecting Aneurism.* Laennec saw an aorta in which the internal and external coats were thus separated from the arch to the bifurcation; and a case is quoted in which the blood thus dissected its way for a few inches and then passed into the original channel of the vessel by another opening just above the bifurcation, and thus establishing a fresh course for itself, obliterated the natural channel of the aorta. The natural tendency of the disease is to increase, despite the reparative deposition of barriers of fibrine or coagula; on this account the sac may attain a great size, but in doing so its walls usually give way, and a *diffuse Aneurism* is formed. In some cases the original walls disappear entirely, and their place is taken by adjacent textures without the occurrence of any great extravasation of blood. And thus the walls of an Aneurism may come to be formed of fasciæ, bones, viscera, layers of fibrine, &c.

The changes which occur in the sac are either conducive to the cure of the disease or to the occurrence of rupture. The latter is by far the most common tendency; for, despite the reparative deposition of barriers of fibrine, the Aneurism increases in size under the pressure of a current of blood too rapid to coagulate or deposit fibrine, and eventually the sac gives way, either forming a diffuse Aneurism, or destroying existence at once by the loss of blood in large quantities, or by sucessive hemorrhages letting life ebb out more gradually. If a diffuse Aneurism be formed, the diffused blood may coagulate, layers of fibrine may be deposited, and these, together with adjacent parts, may contribute to prevent further extravasation of blood, so that life may be considerably prolonged; in the end, however, a diffuse Aneurism is almost always fatal, either by the enlargement of the tumor or by its rupture. The other termination to which the disease progresses is that of cure. Hodgson relates a curious case of a small Aneurism which had eaten its way into the body of one of the dorsal vertebræ, and had there become completely filled with layers of fibrine, which presented a smooth even surface to the channel of the aorta. Again, the Aneurism may become so placed as to press upon its aperture of communication with the aorta, and thus may lead to its own cure by compression. From whatever cause the deposition of fibrine arises it will lead to the safest cure of the disease, but generally the filling up of the sac will be aided by coagulation of blood between or within the layers of fibrine. The sac may become obliterated by inflammation and suppuration of its walls and contents. Haldane reports a case in which three Abdominal Aneurisms underwent spontaneous cure by calcareous degeneration of fibrine which had coagulated in their interior.[1]

Seat of the Disease.—We have already said that an Aneurism may commence at any part of the circumference of the aorta, and Dr. Sibson shows that it occurs just as frequently on the anterior as on the posterior aspect of the vessel. Generally the tumor inclines to the left side, but in some cases it has been seen projecting across the front of the spine towards the right side. It may be seated beneath the pillars of the diaphragm, and being embraced by them, may project into the chest as well as the abdomen. Most frequently the disease occurs near the origin of the cœliac axis, and it often involves the orifice of that vessel; the origin of the superior mesenteric artery is also a common seat—indeed, it may be said with truth that the disease usually occurs above the renal arteries. I *have* seen several cases, however, in which the disease was seated below the renal vessels, and one in which it must have been as low as the origin of the inferior mesenteric artery. As a rule, the arteries near the Aneurism become involved in it, and sometimes they are dilated; when the Aneurism is large they are often stretched over it, and in most of these cases they are obliterated either by pressure or coagulation of fibrine. Pressure upon other parts occurs as the Aneurism enlarges; it may become doubled on the aorta, and thus compress that vessel itself; or, as already observed, the various branches of the aorta may be compressed and obliterated—compression of the bile-ducts may occur, leading to jaundice; of the duodenum and pylorus, causing nausea; of the cardia and œsophagus, producing vomiting and dysphagia; or of the renal vessels (generally without uræmia, or suppression of urine). It is curious that compression of the renal vessels so seldom leads to serious results. I have a specimen in which the left renal artery is obliterated, and the corresponding kidney is dwindled to one-third its natural size, while the artery of the opposite side is pervious, and its kidney is hypertrophied to double its natural size.[2] In this case

[1] Edinburgh Medical Journal, Jan. 1863.

[2] See a specimen, in Path. Division of the Newcastle Museum.

no renal disorder manifested itself by symptoms during life. I do not know of any proof that the thoracic duct suffers from compression in the abdomen, although such a complication with consequent emaciation is highly probable, nor have I heard of the *pancreatic duct alone* suffering in a similar manner. The left kidney may be considerably displaced, and pushed over to the right, or the liver may be pushed forward, and the disease may thus simulate enlargement of that organ. The vena cava is so far removed from the aorta above the level of the renal arteries, that it is seldom compressed; and dropsical accumulations, or enlargement of the superficial veins, are on this account rare in Abdominal Aneurism; lastly, the colon may be obstructed in its descending division with the occurrence of symptoms of obstruction during life.

The aneurismal sac will protrude anteriorly, forming a considerable pulsating tumor, if it spring from the anterior aspect of the vessel: if, on the other hand, it spring from the posterior aspect of the vessel, it will be bound down by fasciæ or other structures and protrude but little; while, however, these posterior Aneurisms pulsate and protrude but little, they lead to more serious results by pressing on important deep-seated parts. As the nervous pains which form the chief symptoms of Aneurism are the result of this pressure, it may be well to indicate the anatomical relation of anterior and posterior Aneurisms (*i. e.*, Aneurisms springing from the anterior and posterior aspect of the aorta) to the nerves in which the pain is chiefly seated. An anterior Aneurism which springs from the anterior aspect of the aorta and protrudes forwards, will necessarily compress the ganglia plexuses and branches of the abdominal sympathetic system: a posterior Aneurism, which springs from the posterior aspect of the aorta and grows in a posterior direction, presses on the roots or branches of the spinal nerves as they issue from the intervertebral foramina, in close proximity to which the aorta is placed at this part of its course. Corresponding with these anatomical facts, Dr. Sibson has shown the rule with regard to these aneurismal pains to be: that posterior Aneurisms excite paroxysmal and radiating, as well as continuous pains, in the back and loins in a large number of cases, and pains in the epigastrium in a small number of cases; while anterior Aneurisms excite pain in the epigastrium in a large number of cases, and pain in the loins with paroxysms of radiating pains in but a small number of cases. Thus, there is established a direct relation between the situation of the Aneurism and the seat of the pains produced by the disease, and this is fairly explicable by the fact that *anterior* Aneurisms press on the *sympathetic* nerves of the abdomen, while *posterior* Aneurisms press on the *spinal* nerves and their branches. All this is said with a full recognition of the fact that erosion of the vertebræ is produced frequently by posterior Aneurisms, and but rarely by anterior developments of the disease—for the truth is, that no relation whatever can be established between the pain of these Aneurisms and the occurrence of erosion. Sibson found, we admit, that of forty-six cases of sacculated Aneurism, in fifteen there was erosion of the vertebræ, and in every one of these fifteen there was a communication with the aorta on its posterior aspect; but Habershon and others have clearly established the fact, that as to cause and effect no relation between pain and erosion of the vertebræ exists, for cases are recorded in which there was pain in the loins of the most acute nature, and after death the vertebræ were found to be free from erosion; and a painless illness has more than once been known to precede death when the vertebræ were found to be extensively eroded. The conclusions to be drawn are these:—1st. That sacculated posterior Aneurisms, as a rule, produce erosion of the vertebræ, and are generally accompanied by lumbar pain, but cases may occur where by pressing on the spinal nerves alone, pain without erosion may be produced; and conversely, sacculated posterior Aneurisms may in rare cases erode the vertebræ without pressing on the spinal nerves, and therefore without producing pain: in short, you may have pain without erosion, and erosion without pain, for the pain depends upon pressure on the spinal nerves, *and not upon erosion.*

Erosion of the vertebræ occurs chiefly in Aneurisms which open into the aorta on its posterior aspect. By this process the bodies of the vertebræ may be completely destroyed, and then the Aneurism may communicate with the spinal canal, or the vertebral column may be considerably displaced, and angular curvature may be thus produced. In a case already referred to, the body of one of the lumbar vertebræ was completely hollowed out by a small Aneurism, and the cavity thus formed was lined by a fine smooth membrane. I need scarcely say that erosion of bones is a process distinct from caries, consisting of a combination of absorption and molecular destruction under the influence of pressure. *True caries* of the vertebræ has been observed in connection with aneurismal pressure, and this is not at all to be wondered at, if the aneurism occur in a strumous subject, or if the bone has previously been in an unhealthy condition.

The rupture of Abdominal Aneurisms is more frequently followed by the forma-

tion of a *diffuse Aneurism* than in the rupture of any other vessel; this is due chiefly to the tough and yielding structures which surround the aorta and its branches. The rupture, and consequent hemorrhage, may take place suddenly and fatally, or gradually, by successive gushes of blood, and without immediate death. This difference in the mode and consequences of rupture is due to a difference in the texture through which it may occur; when Aneurisms open on the cutaneous surface, the skin previously becomes attenuated, it loses its vitality, a slough forms, and thus an opening is made slowly and by a gradual process; on the mucous surfaces the opening is formed in the same manner; but in the serous membranes it always occurs somewhat suddenly by a *rupture or rent*. Thus on the surface of the body and in mucous canals, hemorrhage is gradual and at first very slight, sometimes a mere trickling of blood, but it is sudden and complete when the vessel bursts into a serous cavity. When the opening occurs into areolar tissues, whether subcutaneous, submucous, or subserous, a diffuse Aneurism generally results before the final or fatal rupture takes place. The following statement will give the best idea of the parts where rupture is likely to take place.

Of the cases collected by Dr. Sibson, rupture took place in 77 per cent. Of these, 28·5 burst into the peritoneal cavity; 8, into the mesentery; 9, into the left pleura; 6·5, into the right pleura; 22, behind the peritoneum in the left hypochondrium; 4, behind the peritoneum in the right hypochondrium; 7, into the duodenum; and in 21 cases of rupture behind the peritoneum, 17 of the Aneurisms communicated with the aorta posteriorly, and only 3 communicated anteriorly.

Habershon, Stokes, and others have noticed rupture with extravasation into the iliac fossa beneath the fascia, into the cellular tissue around the aorta (in which case the blood may find its way upwards into the chest), into the small omentum, and into the mesocolon, forming in it, as Stokes says, a pillow of blood. Laennec, Chandler, and Dr. Beatty record cases of rupture into the spinal canal. Rupture may occur into the lungs, and death by hæmoptysis may follow; this happened where pleuritic adhesions caused the base of the lung to form part of the parietes of the Aneurism, through which the blood found its way to the less resisting parenchyma of the lungs. Lastly, rupture into the vena cava is mentioned in Crisp's tabulated cases. Mr. Syme's case of varicose Aneurism between the aorta and the vena cava is, as far as I know, unique.

THE CAUSATION OR ETIOLOGY OF THE DISEASE.—The chief predisposing cause of Aneurism, here, as elsewhere, is degeneration of the arterial coats, but this is not the sole cause, as the aorta in the abdomen, though much less frequently degenerated than the thoracic aorta, is, nevertheless, as frequently the seat of Aneurism as that vessel; this proclivity to Aneurism is due, no doubt, to the position occupied by the abdominal aorta: closely bound to the spinal column in its most mobile part, it is subjected to every position the body may assume—at one time being greatly on the stretch, at another almost bent upon itself—nor are the variety and extent of the movements to which this vessel is subjected the sole causes of this disease, for the rapidity with which they are suddenly performed is often of itself sufficient to lacerate its brittle inner coat. This latter cause is seen to act in cases where the patient has suddenly attempted to regain his balance, or where he has made a sudden start or effort, from which he is often able to date the commencement of his malady.

We may, therefore, lay it down that muscular exertion is the chief exciting cause of the disease, and that this may operate in two ways: 1st. By suddenly subjecting the vessel to a severe strain. 2d. By continually subjecting it to a variety of movements which increase the strain upon its internal coats. Fully according with these statements, we find that intemperance, severe privations, advancing age, irregularities of life, and dissipation, syphilis, and rheumatism (all of which induce gradual degeneration of tissue), are frequently adduced as predisposing causes of the disease. We find that occupations and conditions of life leading to great muscular exertion are prolific as exciting causes of the disease; for instance, of 49 cases, Crisp found that 47 were between twenty and fifty years of age, and that 22 of these were between thirty and forty years of age; so that, while age undoubtedly contributes to produce the disease, it is not *old age* especially, but a certain time of life (when degeneration may have set in) which is generally accompanied by great bodily activity. In short, a small amount of degeneration accompanied by great bodily exertion more easily leads to the disease than a large amount of degeneration with slow and feeble bodily exertion. Corresponding proof of this is found in the frequency of the disease in males as compared with females. Crisp found 8 female to 51 male cases; Habershon, 2 females to 11 males. As a rule, when the disease occurs in females they are young, and have led very hard and irregular lives of dissipation. Gairdner mentions such a case in a young woman aged only sixteen years. The occupations which seem to predispose to the disease are all of a labo-

rious nature; the men are smiths, strikers, excavators, navvies, porters, paviors, founders, &c. The disease *may* occur apart from these exciting causes, and when it does so we may be sure there is more certainty of the aorta being extremely diseased, and far less chance of the disease being cured than when it is caused by muscular exertion, &c.

In this category of causes we ought to include external injuries, such as blows, by which the disease is not infrequently produced; and we cannot lay too great stress upon the bad effects of overstraining, such as the following.

A healthy young fellow is employed as a pavior, and on lifting his huge wooden rammer for a blow loses his balance; with the rammer uplifted he makes a vigorous effort to keep his feet, during which he "strains his back;" from that day he begins to complain of symptoms of Aneurism, and eventually he turns out to have the disease.

SYMPTOMS.—I propose to discuss the symptomatology of the disease under two heads.

1st. Those symptoms and signs which by their presence indicate the disease. *These are the positive symptoms.*

2d. Those symptoms which by their presence *contra*-indicate or negative the existence of Aneurism, and by *their absence*, in doubtful cases, are therefore evidence in favor of the existence of Aneurism. *These are the negative symptoms*, and are of course produced by other diseases which closely resemble Aneurism in their main characters; when these symptoms positively present themselves, they often exclude the possibility of Aneurism; when they are altogether *absent*, we have an ominous sign that the disease exists.

1st. *The positive symptoms.* Pain is generally the earliest symptom, the most prominent symptom during the course of the disease, and the symptom which by its increasing intensity gives the severest sting to the patient's dying moments. With regard to its frequency, we may say it occurs in five-sixths, if not in seven-eighths of the cases on record; its frequency and its general characters are chiefly determined by the seat of the Aneurism.

The absence of pain in some cases is, however, a well-established fact, and when this is combined with an absence of other leading symptoms, the patient may live, till within a few hours of his death, without the slightest suspicion that any serious malady exists. Such a case has occurred to my knowledge where rupture and loss of blood were the cause of death. We have before demonstrated the relation which exists between the seat of pain and the seat of the disease, and have shown that pain is a more severe and more frequent occurrence in posterior than in anterior Aneurisms. As the nature of these aneurismal pains is peculiar and characteristic, their consideration is of great importance. They are of two kinds. 1st. *A continued pain* in the back, loins, epigastrium, or hypogastrium, as the case may be. 2d. *An intense paroxysmal pain* in the back or loins, which radiates to the front of the belly, to the testicle, or down the thigh, according to the spinal nerves through which it may be produced. The first is wearisome, and exhausts the patient by depriving him of his rest; the second is agonizing, and leaves him prostrate after every paroxysm. The pain is unaccompanied by febrile excitement, acceleration of the pulse, rigors, or perspirations; it usually increases as the disease progresses, and before death agony of the most acute nature has often to be endured: in one case this was so severe that an exhausted and dying patient fairly leaped out of his bed, and in another case it brought on an attack of raving delirium.

We conclude that the rule with regard to pain in Abdominal Aneurism is, that it may be seated in the back or in the belly, indicating in the former site a posterior Aneurism, and in the latter an anterior Aneurism, and that in either case it may be of two kinds—paroxysmal or continued. The situation of the pain occasionally varies: it may be seated in the hip, in the iliac regions, changing from one side to the other (Beatty's case); it may simulate colic, being increased after eating, or by constipation of the bowels, and may be accompanied by nausea, anorexia, vomiting, or flatulence. The region of the liver may be painful and even tender, with pain about the shoulder and scapula, thus simulating hepatic disease; the chest may be the seat of pain with dyspnœa, or the cardiac region with palpitation. One other strong feature of the pain is its tendency to "catch the breath" during the descent of the diaphragm, and in such cases we may suspect the seat of the disease to be underneath the crura of the diaphragm.[1]

The paroxysmal pain may closely simulate that of renal or biliary calculus, lead colic, or simple neuralgia.

All these somewhat anomalous pains may seriously complicate the diagnosis, and in such cases a careful consideration of other symptoms will be necessary. Change of posture often affects the pain considerably. Patients usually experience relief by lying on their face or on their right side, by resting on the hands and knees, or by sitting almost double. On the other hand, lying on the back, or standing in the erect posture, are usually

[1] See cases by Dr. Ogle, in Lancet.

painful positions. The pain is almost always increased by bodily exertion, and in some cases even the slightest movement is attended by pain; pressure usually increases it, but in some instances pressure has afforded considerable relief; sometimes the surface over the Aneurism is exquisitely tender; but all I have seen were quite free from this, and might be manipulated gently with the greatest freedom from pain.

The pain varies in *character* as well as in situation; the words boring, burning, catching, pulsating, twisting, lancinating, are used by patients to convey to our minds the different kinds of pain felt by them. It is often dull pain when continuous, and sharp when paroxysmal. It varies in *intensity* also,—it is sometimes absent, at other times trivial; in most cases very severe, and occasionally unbearable. We would urge the great necessity of recognizing the import of pain such as is here described in all obscure cases, and of making it an incentive to a most careful physical examination of the abdomen, in order to determine whether or not an Aneurism exist.

A *pulsating tumor* forms another most important symptom of this disease. The pulsation may be visible even at a distance, forming a distinct heaving projection of the abdominal wall; on the other hand, it sometimes gives no visible evidence of its existence.

To the applied hand a distinct heaving impulse is communicated, and at the spot a more or less defined tumor can often be felt. The character of the pulsation is very marked; felt equally in all directions —laterally as well as in front—the tumor seems with each pulsation to expand under the hand, and when the fingers are applied to its sides they are separated with each pulsation. In some cases, where the sac has pressed on the cardiac region, a double shock or pulsation has been communicated to it from the heart. There may be a thrill or vibration with each pulsation, and if by pressure the sac can be emptied, its distension will be accompanied by a purring thrill. The tumor may be movable when it occupies one of the branches of the aorta, *e. g.*, the sup. mesenteric; in opposition to this, however, we have cases by Courato and others, diagnosed to be Aneurisms of this vessel by their non-mobility along with the diaphragm: most frequently the tumor is fixed. The site of the pulsation varies considerably; most frequently it occupies the epigastric region and inclines to the side, so as to be covered by the margins of the left false ribs; in rare cases it inclines to the right side, and in so doing may push forward the right lobe of the liver with each impulse; it not unfrequently occupies a spot a little above and to the left of the umbilicus, or it may be so deeply seated in the epigastrium as only to be felt on deep pressure in that region. The mass may present itself in the left lumbar region, or in the groin, simulating lumbar abscess in the one case, and psoas abscess in the other; or it may project considerably towards the right lumbar region, and so simulate enlargement of the kidney. I have read of one case in which the tumor occupied the position of the spleen, and was mistaken for disease of that organ; and in another case, under my care, whenever the patient lay upon his left side the tumor fell under his left ribs and entirely disappeared. Added to the difficulties which may arise from the various situations of the tumor, is the established fact that, in some cases, neither pulsation nor tumor can be detected. Hope mentions a remarkable case of this kind, where the Aneurism was found to be bound down by the pillars of the diaphragm, the lumbar fascia, and bands of adhesion—an occurrence by no means impossible when the Aneurism is small and projects backwards from the posterior surface of the aorta. As before mentioned, the tumor may sometimes be emptied by pressure, but this will depend upon the amount of fibrine in its interior.

The Physical Characters of the Tumor.—The tumor may be soft and fluctuating, and in such cases it is easily emptied of its contents by pressure, or it may be dense and resistant to the touch; its surface is generally smooth, although in rare cases it may be lobulated. Percussion does not often afford valuable evidence of the disease, for the presence of intestinal flatus, and the deep situation occupied by the tumor when it is above the renal arteries, interfere with its indications. The percussion note is generally dull over the tumor, but this dulness may be limited to one part, such as the summit of the tumor, and there is generally a line of resonance running between the tumor and the liver. The characteristic expansile pulsation of the tumor may be replaced by a forcible short or jogging shock communicated to the applied hand.

A bellows murmur is frequently to be heard in connection with Abdominal Aneurism. It was observed in 25 per cent. of the cases collected by Sibson, but I cannot help thinking that it occurs more frequently than this, for I have never seen a case without it. The character of the murmur varies; according to Dr. Walshe it may be, 1. A single systolic murmur. 2. A dull, muffled, systolic sound, convertible into a murmur by a little pressure. 3. A sharp, abrupt, systolic murmur, audible at the left lumbar spine, or much more marked there than in front. 4. A systolic murmur below the sac, and none immediately over it. 5. Occasion-

ally a dull second sound. Dr. Walshe has never heard a murmur diastolic in time.[1] A shock may be perceived by the ear, as well as a murmur; it may be single and systolic, or double; the presence of a second diastolic shock is pathognomonic of the disease; as before said, a thrill may accompany the murmur. Where murmur is inaudible, pressure with the stethoscope over the Aneurism may produce it, and in all cases the patient should be made to assume the recumbent position, which generally increases both the murmur and the pulsation of the tumor. Dr. Corrigan attributes this development of the murmur and pulsation in recumbency, and their disappearance in the erect posture, to the removal of hydrostatic pressure from the walls of the cyst, which permits a more free passage of blood in and out of it, with less tension and more vibration of its walls. The murmur varies in intensity: it may be feeble, or so loud as to be audible at a distance, and its intensity sometimes diminishes with the growth of the Aneurism.

It is very important to bear in mind, in obscure cases, that all physical signs may be absent—impulse, murmur, and dulness; and in such cases a diagnosis of the exact nature of the tumor cannot possibly be made. Murmur is sometimes the only physical evidence of the disease. In all suspected cases, therefore, the stethoscope ought to be applied, and its application to the spine and left vertebral groove ought not to be neglected, as murmur in that region is of grave import.

To these, the cardinal symptoms of the disease, we must add others of less importance. *Respiration*, if the sac be so high or bulky as to interfere with phrenic action, will be quickened and impeded. Sibson found dyspnœa mentioned in eight per cent. of the cases. *Cough* may be excited when the walls of the thorax become in any way connected with the sac, but, as this is rare, we find cough much less frequently present than dyspnœa. *Dysphagia* was observed in about six per cent.; and from the close anatomical relation which the œsophagus bears to the aorta we need not be surprised at this. Nausea, vomiting, loss of appetite, flatulence with spasmodic pain, and constipation, are all common enough occurrences in the course of the disease. Mayo observed numbness of the lower limbs; and in other cases, coldness, formication, pricking, numbness, and even paralysis of the legs has occurred. Ascites or anasarca of either limb are exceedingly rare, and, as will be seen, these occurrences are more indicative of the absence of the disease than of its presence, *i. e.*, they are negative symptoms which when present almost preclude the existence of the disease. As the result of pressure, we ought to mention Professor Seaton's curious instance of contraction of the right pupil in a case of Abdominal Aneurism; probably the sympathetic nerves were the channel through which this curious phenomenon was brought about.

[Another effect of pressure is the occlusion, partial or total, of the thoracic duct. This was diagnosticated during life, and verified after death, in a case which I saw in the Pennsylvania Hospital, under the care of the late Dr. W. Pepper. No doubt the emaciation, in many cases of Abdominal Aneurism, is considerably accelerated by this cause.—H.]

It is a curious fact that all these symptoms may for a time disappear, and the patient get apparently well; it is curious, too, that during seasons of great excitement, when the mind is directed to other things, the pains may be completely forgotten. The cases of the gentleman who spent a day in the hunting-field, and the barrister who made a powerful speech in court, a short time before death, abundantly illustrate this. As the disease progresses and the pain increases, insomnia and restlessness at night tend to exhaust the body; the patient becomes emaciated, exhausted, worn out, and would doubtless die of slow exhaustion did not death from rupture put an end to his sufferings.

2d. We now come to *the negative symptoms*, by which we mean those symptoms which seldom or never occur in Aneurism, and therefore negative the existence of the disease; these symptoms being generally present in diseases which simulate Aneurism, are of great importance in the diagnosis of doubtful cases. They speak against the existence of Aneurism, and for the existence of other diseases.

We first remark the absence of general arterial excitement, with undue fulness or rapidity of the pulse—78 to 88 seems to be the average rate of the pulse, and the pulse-respiration ratio generally remains at the normal standard. Undue rapidity of pulse is therefore a negative symptom. The temperature of the body is, as far as I have observed in two cases, normal, and from the exhausted state of the patients, it may be below par; great increase of temperature indicates some other affection. The heart's action may continue to be quite normal; indeed, we may say disease of the heart is the exception rather than the rule in this disease.

Anasarca of the extremities, so important in many abdominal diseases, *is very rare* in Aneurism, and only occurs when the cava becomes involved in or communicates with the tumor.

Enlargement of the superficial veins of

[1] Diseases of the Heart, p. 494, 3d ed.

the abdomen is also exceedingly rare in Aneurism, and very common in other diseases of the abdomen. Ascites is still more rare, and being so common in abdominal disease, its absence is seriously significant of Aneurism. Dr. Stokes lays great stress upon the absence of effused lymph and the friction-sound which it produces when it is present in the peritoneal cavity. Unless the disease press on the emulgent vein of the kidney the urine is healthy; and as, clinically, very few exceptions to this rule occur, the absence of sediment of pus, mucus, and albumen, ought to prevent us from assigning a renal or vesical origin to the pain. Jaundice, want of bile in the stools, general constitutional disturbance with complication of other organs, and enlargement of the liver, are opposed to the probability of the existence of Aneurism.

Mobility of the tumor rarely occurs in aneurism of the aorta itself, although occasionally this is observed in Aneurism of its branches; but mobility is, as we shall see, common enough in some other abdominal tumors. Stokes points out that aneurismal tumor never begins below and increases upwards; this upward growth of a tumor would therefore argue powerfully against Aneurism.

The general cachexia of cancer, or the tubercular diathesis, are very rare in Aneurism, as are also deposits of cancer or tubercle; so that in all doubtful cases, careful inquiry into the history of the patient, and careful physical examination of the body, are necessary.

The diseases which may be mistaken for Aneurism are so numerous and diversified that it may be well to divide them into—

1. Affections depending on increased aortic pulsations only.

2. Affections wherein a tumor, itself pulseless, receives an impulse from the aorta.

3. Affections wherein pulsation, not derived from the aorta, exists.

4. Affections wherein a pulseless tumor simulates a non-pulsating Aneurism.

5. Affections where pain without tumor exists.

1. Increased *Aortic pulsation* is not expansile, the lateral impulse and the movement in a forward and slightly downward direction being wanting. Murmur is very rarely present (never occurring over the spine, and scarcely ever diastolic), and the most careful manipulation and percussion will fail to detect lateral enlargement in calibre. The absence of pain, of the evidence of arterial disease, and the occurrence of the disease in young, anæmic, and female subjects of nervous temperament, or its development during attacks of dyspepsia, flatulence, or constipation, and its entire removal by treatment, are indications sufficient for our guidance in ordinary cases. In more obscure cases, the most careful inquiry will often fail to produce a decided opinion.

2. Tumors which receive an impulse from the aorta.

Under this head are cancer of the stomach and pylorus; cancer of the pancreas, liver, and small omentum, involving the duodenum. Tubercular deposits in the mesenteric or lumbar glands; omental tumors or cysts, ovarian tumors, and tumors attached to the uterus, or even the enlarged uterus itself; enlarged kidney, distension of the pelvis of the kidney, a movable kidney, distension of the colon from flatus, worms, or feces (the latter being by far the most common), or cancer of the transverse colon, each of these affections may receive an impulse from the aorta, and may compress the vessel so as to lead to murmur also. By attention to the symptoms before delineated, many of these may be excluded; and, as the diagnosis in each case will require a full consideration of each suspected disease, all I shall attempt to do here will be to lay down a few general rules for our guidance.

A. In but a few of these cases does the impulse possess the heaving, expansile character of Aneurism; and lateral impulse is rare.

B. The tumor in most of the above cases is movable, and when moved the murmur is modified thereby; in Aneurism of the Aorta itself this is never the case; and in Aneurism of its branches, movement does not cause the murmur to disappear; by causing the patient to rest on his hands and knees the tumor may fall away from the aorta, and pulsation will then cease if it be not aneurismal.

C. In many cases symptoms contra-indicating Aneurism will be present.

D. In all cases a more careful inquiry into the history of the case, the constitution of the patient, and the character of the tumor, is necessary, for in many cases this will give great aid to diagnosis.

E. Clear out the bowels, examine the urine, remove dyspeptic symptoms, regulate the uterine functions, and in so doing make careful inquiry into the condition of the organs involved.

3. This class of cases includes those tumors which possess in themselves a heaving impulse, due to their active vascular condition. The relation between a diffuse Aneurism and a vascular cancer is sometimes very close, even when external; and when vascular organs like the liver or kidney becoming affected with hæmatoid cancer, acquire a heaving, expansive pulsation, the diagnosis is very difficult. Cancer of the mesenteric and lumbar glands may also simulate Aneurism. It

is in these cases that the presence of constitutional disturbance, quick pulse, hot skin, perspiration, and of diathesis, is so useful an aid to diagnosis; the symptoms of disease of the organs involved would also aid us if present.

4. Cases where a pulseless tumor resembles a non-pulsating Aneurism.

Of this class are psoas and lumbar abscess, possibly leading to displacement of vertebræ, like angular curvature, causing pain in the back like that of Aneurism, and perhaps appearing suddenly as if the Aneurism had ruptured. We must include in this class any of the tumors in class 2 which may have acquired the symptoms of Aneurism without pulsation. The absence of murmur in these cases, and the presence of cancer or tubercle in other organs, would aid the diagnosis.

The 5th class of cases is a difficult one. It includes all cases of obscure pain in the belly, back, loins, hips and thighs. Cases of lumbago, sciatica, neuralgia of the abdominal wall, intestinal pain, the pain of calculus, various hepatic and nephritic pains, spinal pain, &c. &c. In such cases a careful physical examination of the abdomen, for other evidence of the disease, is the best safeguard in avoiding error; but beyond this, and the special symptoms of these diseases which may accompany the pain, I know of no point on which to establish a diagnosis.

Course, Duration, and Termination of the disease.—Beginning suddenly, or developing gradually by obscure symptoms, the course taken by the disease varies; it may be so mild as never to betray its existence till the final rupture takes place, or it may entail almost constant suffering on its victim. In pronouncing an opinion it is of great importance to remember that even in severe cases periods of convalescence occur in which all symptoms disappear, and the patient feels quite well. Many cases seem to exist for a long time without serious disturbance, when suddenly, after the Aneurism has reached a certain size, all the symptoms set in with great severity. The duration of the disease is variable; cases are on record which lasted seven years, and even eight years, but this is far above the average. The practical question seems to be, how long will a patient live after an Aneurism has produced severe or decided symptoms of its existence? This period rarely exceeds eighteen months, and after the formation of a decided pulsating tumor, with paroxysms of pain, the majority of patients die within three months. The termination of the disease has hitherto, in almost all cases, been in death. A few cases are recorded where, without aid, nature managed to fill up the sac by layers of fibrin, &c., and thus to establish a cure. In other cases again, by proper diet and regimen, and the employment of various modes of treatment, the disease has been cured.

Death may occur from exhaustion, or from hemorrhage; and death from hemorrhage may be sudden or gradual. When gradual, the patient may at first experience relief of his pain, or shock with rigors may be experienced at the time, and last more or less for a few days, during which the heart beats rapidly and feebly, the first sound being almost lost. The pulsation in the tumor, after rupture, becomes weaker, the murmur is lessened, and a new, soft, semi-solid swelling is formed without pulsation or murmur. Dr. Walshe has observed pulsation of expansile character in the extravasated blood, but the rule is, I believe, as stated above. Dr. Lyons found the symptoms of rupture followed by dulness on one side of the chest, and diagnosed rupture into the pleural cavity, which proved to be correct. Hemorrhage into the peritoneal cavity is generally followed by sudden death, and hemorrhage into the lung, stomach, or bowel, will be followed by discharge of blood from those cavities. Convulsion may accompany loss of blood. Dr. Stokes thinks that a sudden loss of blood is more dangerous than the loss of larger quantities by degrees. In one case Dr. Stokes observed fainting on three successive days before death: he believed them to correspond to successive discharges of blood from the Aneurism. When the blood forms a tumor in the lumbar or iliac region, the pulsations in the femoral may be impeded, but this is not always the case.

TREATMENT.—Like most uncured diseases, Aneurism of the Aorta has been subjected to various methods of treatment. It will be needless to dwell upon those methods which have become obsolete, and have been rejected by the experience of the profession. Of these the treatment of Valsalva has been most commonly adopted, for in most cases it affords temporary relief, and in some cases seems to have established a cure, where the strength of the patient, aided by his faith and perseverance in the means employed, permitted of a fair trial. We should scarcely have adverted to the plan of Valsalva, had not Tufnell, of Dublin, brought out some rather startling cases showing that a modification of Valsalva's treatment may prove successful. Dr. Tufnell's plan is that of Valsalva without depletion, and consists chiefly in rest, plain but good diet, and soothing medicines when needed. The patient should be placed in the recumbent position, and this he must maintain for some weeks, or even months. Now, despite the success which has attended Dr. Tuffnell's efforts, I can-

not think his treatment will ever become popular, for few patients will be found willing or able to rest night and day in one posture for a period of three months, and the irksomeness of the patient's own existence during that time is not the only difficulty, for the patience of relatives will also be severely tested in giving all due attention to the case; however, when no better means can be employed this may be tried. The administration of medicines has hitherto done but little for internal Aneurism, and further researches in this respect are needed. Dr. Owen Rees has given acetate of lead, and Roberts, Balfour, Begbie, and others have shown how decidedly the symptoms of internal Aneurism improve on the administration of large doses of iodide of potassium. In my own experience the iodide has, in some cases, relieved the symptoms; in others, the symptoms have altogether disappeared for a time, and in one case I believe a cure has been effected. This experience, I think, fairly represents that of the profession generally on this subject. When other means of cure fail, or when they are inappropriate, there still remains one procedure which holds out a fair hope of recovery to the patiect. *Compression*, which has done so much for the cure of Aneurism of other arteries, has acquired a peculiar honor in proving applicable to Aneurism of the Abdominal Aorta; in other arteries it supersedes another less safe, but equally effectual mode of cure (ligature); but in the aorta it renders curable a disease hitherto entirely beyond our reach. As a student in some of our best hospitals, I never heard so much as a hint that efforts for the cure of such cases were worth entertaining. Now, however, there is hope for many cases. Already the aorta has been successfully compressed several times for the cure of Aneurism—both for Aneurism in its own course, and for the iliac Aneurism. As this method of treatment, though easy in its application, requires much care and attention to details, I propose to add as much information on the subject as we at present possess, in the hope that ere long our experience of the treatment will be much enlarged.

Let us premise by a few remarks on the anatomy of the abdominal aorta. The vessel extends from the last dorsal vertebra to the middle of the fourth lumbar vertebra; it lies a little to the left of the median line, having the right crus of the diaphragm, the vena azygos and the thoracic duct between it and the vena cava. The vena cava is nowhere in exact contact with the aorta, and gradually diverging as it ascends, at the level of the renal arteries the greater part of an inch intervenes between it and the end of the aorta. During part of its course the aorta is embraced by the pillars of the diaphragm, so that for about two inches only part of its circumference, the anterior aspect, is in the abdominal cavity; this embraced part of the artery is remarkable for being covered by important organs, and for giving off the most important branches of the aorta. It is covered by the pancreas and the splenic vein, the left renal vein, the third portion of the duodenum, and some of the most important plexuses of the sympathetic nervous system.

The arteries given off from it are placed as follows: 1. The phrenic—immediately the aorta appears from beneath the diaphragm. 2. The cœliac axis—one inch from the commencement of the abdominal aorta. 3. The superior mesenteric—three-fourths of an inch below the cœliac axis or one inch and three-quarters from the commencement of the aorta; and the renal arteries, half an inch lower than the mesenteric, or two inches and a quarter from the point at which the aorta enters the abdomen.

From this point downwards for three inches at least, no large vessel arises from the aorta, nor is the vessel, during this part of its course, covered by important parts.

Lastly, we have the inferior mesenteric artery, a little more than five inches below the commencement of the aorta, the vessel itself terminating at the distance of seven inches from its origin.

From these facts it will be seen that the aorta may be compressed with the greatest safety and facility in its lower two-thirds, that is, in the last five inches of its course. Let us refer for a moment to the points on the front of the abdomen, to which this and other points correspond, so that we may determine from the external evidence of the site of an Aneurism, how far it is amenable to treatment by pressure, and with what amount of safety pressure may be applied.

The aorta extends from the end of the sternum to the umbilicus (seven inches). Allowing one inch for the length of the ensiform cartilage, its tip will correspond to the origin of the cœliac axis. Three-fourths of an inch lower is the superior mesenteric artery, and half an inch lower than it, or four inches and three-quarters from the umbilicus, is the origin of the renal arteries. Allowing an inch for the point on which the pressure is applied, we have here a clear space of more than half the aorta where Aneurisms may be easily and safely treated by pressure, for the inferior mesenteric artery being by its anastomoses well supplied with blood, can be occluded without the slightest risk. The rule is, that you must not apply the tourniquet higher than one inch and a quarter from the tip of the ensiform cartilage, the point of origin of the renal arteries; in

fact, you cannot apply it higher than this point, so that we may conclude it is safe to apply it as high as you can. For an account of the vessels by which the circulation is carried on inside and outside the abdomen after the aorta is occluded, I must refer the reader to my work on *The Rapid Cure of Aneurism*, and for a still fuller account of the anastomosis between the lumbar branches of the aorta above and the branches of the internal iliac below, I would refer to the researches of Prof. Turner, of Edinburgh. It may not be out of place here to add a few words on the application of pressure to the aorta, as its success depends entirely upon an accurate attention to the details of the procedure.

Before proceeding to compress, it is important to ascertain that the heart, liver, kidneys, and other organs are in a healthy state. It is also important to clear out the bowels both by a purgative and by a stimulating enema, as distension of the abdomen by flatus adds to the difficulty and danger of applying firm pressure. The use of chloroform is absolutely essential for more than one reason. It not only enables the patient to bear severe and long-continued pressure, but it also removes the resistance of the abdominal parietes. The instrument to be used may be a large Signorini's horse-shoe tourniquet, or Lister's tourniquet for the abdominal aorta, or a tourniquet with a fine adjustment, invented by Dr. G. Y. Heath and myself, and made by Coxeter. The strength of the rack and pinion, or screw of the instrument, is of very great importance in all cases.

In applying the tourniquet, complete arrest of the circulation through the Aneurism should be aimed at, as it is of the greatest moment to secure complete stagnation of a mass of blood in the Aneurism for a period of time sufficient to produce coagulation. It is very probable that coagulation sets in rather suddenly when once the conditions for it have been secured, and one object should be to encourage this tendency in the blood by retaining it in a stagnant condition as long as possible without the slightest movement or disturbance.

After considering the experience derived from recent cases, I am of opinion that we should proceed as follows in carrying out the pressure treatment :—

1st. Apply the pressure for four hours, and if on removing the tourniquet no impression has been produced on the pulsation, the first attempt must be considered at an end, but if the pulsation is somewhat diminished, the instrument should be reapplied for another hour. In one case when pressure was being applied to the aorta for the cure of an iliac Aneurism, when I arrived I found the assistants who had charge of the case had just removed the instrument in despair, but perceiving a distinct lessening of the pulsation, I insisted on a reapplication of the instrument, and in twenty minutes the Aneurism was consolidated, and the patient got well.

2d. If the first attempt has been unsuccessful, a few days must elapse before another trial is made ; on this occasion the pressure should be maintained for six or eight hours.

3d. If this fail, a final effort must be made, and the pressure extended to a period of twelve hours. There is but little danger if the process be tried in the above cautious manner, as indications of inflammation, exhaustion, or shock would at once put a stop to our efforts. Pressure so prolonged as to exhaust the patient, or so frequently applied as to prevent perfect recovery in the interval, is specially to be avoided. I need scarcely add that everything depends on the personal superintendence of the surgeon who undertakes to treat the case. He must give up his ordinary work for the day, and devote himself to this alone. Assistants cannot be expected to give that concentrated attention to the tourniquet which is essential to the proper application of pressure.

If we fail to cure the Aneurism by any of the above efforts, we must endeavor to alleviate the sufferings of the patient as much as possible. Leeching and cupping are sometimes useful in relieving the pain of plethoric patients. Rest is an all important condition for the relief of pain. Nocturnal pains and insomnia are best combated by opiates, bromide of potassium and chloral in appropriate doses ; belladonna combined with opium is of great use, and liniments of aconite or belladonna and chloroform are useful external applications, but the subcutaneous injection of morphia is our best remedy for pain. It is important to keep the patient's mind free from undue anxiety, and to maintain the general health by diet and regimen. Medicines which control arterial excitement, such as aconite, hydrocyanic acid, green hellebore, and digitalis, are often of great use, but in spite of these and all other remedies the patient will often suffer severely before he dies.

For much of the pathological anatomy on this subject we are indebted to the labors of Dr. Sibson and Dr. Habershon, and to Mr. Timothy Holmes we are indebted for the fullest exposition of the surgical aspects of the subject, as well as for the first suggestion that pressure might possibly be applied to the abdominal aorta for the cure of Aneurism. Up to the commencement of 1873 at least twenty Aneurisms had been treated by

pressing the abdominal aorta, of these only two died, and in them the fatal result was evidently brought about by re-applying the tourniquet too soon. I cannot too strongly urge the importance of avoiding this; nothing is gained by a speedy re-application, and great risk is thereby incurred. I need not say that the advocates of a new method of treatment should, above all things, avoid the arrest of its early development and tender growth, by too rudely or hastily putting it to the test in inappropriate cases, or by pushing it too far.

Since the above date several cases have occurred. Before referring to them I ought to mention the case cured by Moxon and Durham in Guy's Hospital, and to the case treated by Sir J. Paget without success. I ought also to refer to the case where Mr. Bryant applied distal pressure with fatal but not altogether discouraging results. A full discussion of the treatment used in these cases is contained in Mr. Holmes's "Lectures on the Surgical Treatment of Aneurism."

Since 1873 Dr. Headlam Greenhow has successfully carried out the treatment in a case in the Middlesex Hospital, and Dr. G. H. Philipson has succeeded in curing a case in the Newcastle Infirmary. To these might be added one or more cases cured by continental surgeons, and I must not fail to refer to the success of Mapathea and O'Ferral in Dublin in applying pressure to the aorta for the cure of iliofemoral Aneurisms. Most of the above cases illustrate one important point, viz., that an Aneurism often continues to beat for several hours after the pressure has been removed, and then suddenly and finally ceases to do so. To account for this, several suggestions may be offered. It may depend on a gradual contraction of the walls of the Aneurism, fresh laden with barriers of fibrin; or on the sudden formation of a clot of blood on recently-deposited fibrin, or blood-clot; or on some more obscure alteration of the relationship of the contents of the sac to the blood-current. For a fuller discussion of this interesting question I must again refer you to my work on the rapid pressure treatment.

DISEASES OF ARTERIES.

By John Syer Bristowe, M.D., F.R.C.P.

(1) *Inflammation.*—Arteritis, or inflammation of the arteries, is a disease which physicians and surgeons regarded formerly as of common occurrence; but they not only included under the term true cases of the disease, but attributed to Arteritis cases in which, though arterial disease was present, it was not inflammatory, and probably a still larger number in which there was no arterial affection whatever. They regarded, in fact, mere *post-mortem* blood-staining of the lining membrane of the arteries as evidence of inflammatory congestion, and all degenerative changes of the same tissue as the results of the deposition of inflammatory products. It need scarcely be said here that the lining membrane of arteries is now known to be devoid of capillary vessels, and incapable, therefore, of vascular injection; and that any redness it may present is simply due to its imbibition after death of the coloring matter of the blood which has lain in contact with it; and that atheromatous and other degenerative changes have either no connection with inflammation, or only such a connection with it as have cirrhotic and such like deposits in the liver and other organs.

Inflammation of an artery manifests itself chiefly by changes in the outermost and middle, or vascular coats. In the early stage, the cellular coat, which is that in which the vasa vasorum are chiefly distributed, presents changes similar to those which occur in inflamed connective tissue of other parts; these are, specially, redness from hyperæmia of the capillary vessels, and swelling in consequence of inflammatory exudation. Similar changes, though less in degree, affect the outer portion of the middle coat. But the inner layer of the middle coat, and the internal coat, undergo little if any appreciable modification. If, however, the thickening of the walls be at all considerable, then the calibre of the vessel becomes diminished, and the lining membrane corrugated. The inflammatory changes may become arrested at the point here indicated, and the artery then either revert to its original healthy state, or its walls become permanently indurated and

thickened ; or, on the other hand, the inflammation may lead to softening, and even to suppuration—the latter condition affecting specially the outer portion of the arterial parietes. The processes just described as occurring in the arteries of man, have been proved experimentally, by Virchow and other observers, to be capable of production in the arteries of dogs and other of the lower animals.

Associated generally with arteritis, especially with inflammation of arteries less in size than the aorta, we find the deposition of fibrin on the inner surface of the inflamed tract. This soon increases in quantity, and occludes the channel of the artery. A fibrinous concretion is thus produced, adherent to the surface, blocking up the vessel, sometimes more or less defined and rounded at the extremities, sometimes shading off into ordinary undecolorized coagulum. After a while such a clot is apt to undergo disintegration in its interior, to become a cyst occupied by puriform fluid, and thus to resemble accurately the softening clots which occur so frequently in the interior of the heart. Sometimes, on the other hand, it undergoes changes resembling those which fibrinous deposits in the interior of an aneurism undergo ; it contracts and hardens, and blending with the arterial walls, forms ultimately with them a mere fibrous cord. Sometimes again, the obstructing clot becomes wholly or in part removed, and the channel of the vessel consequently restored.

It was formerly supposed that clots forming at the seat of inflammation, in arteries and veins, were due in great measure to inflammatory exudation taking place from the lining membrane of the affected vessel, and the experiments of Gendrin seemed to prove the validity of this explanation. But Virchow's more recent investigations, the results of which seem to have been confirmed by Henry Lee and Callender, show that if a vein, from the interior of which blood is excluded, be irritated, the substance of its walls becomes infiltrated with inflammatory products, but no exudation whatever takes place on the surface of the lining membrane. It is certain, therefore, that the lining membrane of veins, and, it may be added, that of arteries, either never furnish any inflammatory exudation, or furnish it rarely and with difficulty, and almost certain, therefore, that the plugs which are associated with arteritis are due to the mere coagulation of blood.

But even if it be admitted that these plugs are merely coagulated blood, it by no means follows that they are not in many cases the consequence of arteritis. It is impossible, indeed, on any other view to explain how it is that in Arteritis commencing from without, clots form in the interior of the affected portion of vessel. Doubtless the lining membrane of the artery particles in the inflammatory process and the formation of a clot at the site of inflammation is due to some coagulating influence which the affected wall hence exerts over the blood with which it is in contact.

The causes of arteritis are as various as the causes of most other inflammations. Sometimes it is the consequence of irritation acting from within, as when an accidental thrombus forms at the part or an embolus becomes impacted there. More frequently it is the result of some cause acting locally, but from without, as when an artery is mechanically or otherwise injured, when it becomes surrounded by or imbedded in cancerous or tubercular formations, or when it becomes involved in suppuration of tissue, or in any other process of inflammation. Sometimes again, it is doubtless due to special conditions of general unhealthiness ; and there is some reason to believe that certain forms of anæmia, and that syphilis, may be enumerated among them.

The results of arteritis are also various. Sometimes, no doubt, the arterial walls return to their original healthy condition; sometimes they become indurated and perhaps thickened, with loss of contractile power and of elasticity ; when obstructed by clot, they sometimes, as before stated, remain permanently obstructed, and if life be prolonged, become reduced to mere fibrous cords ; sometimes, on the other hand, their walls become softened and ulcerated, and perforation may take place, followed by hemorrhage, and its consequences.

The most important results, however, of Arteritis are generally those secondary phenomena which take place, in consequence of the obstruction of the artery, in those tissues or parts to which the obstructed artery leads. These will be best considered under the head of "Thrombosis and Embolia."

The symptoms, immediately due to Arteritis, may be very briefly enumerated. They consist in a greater or less amount of general febrile disturbance, with (if the artery be within reach) pain and tenderness at the seat of inflammation, and perhaps fulness and hardness and loss of pulsation. But these rarely exist in an uncomplicated form ; for on the one hand they are often, from the very earliest moment, mixed up with, and masked by, the symptoms of that morbid condition with which they are associated, or out of which they have arisen ; while on the other hand, when inflammation affects an artery leading to any important part, the symptoms due to its obstruction soon by their gravity overshadow the symptoms due directly to arteritis.

The treatment of simple Arteritis may also be dismissed in a very few words. It should consist, first, of such constitutional management as is appropriate for other forms of inflammatory affections, and second (when the artery affected occupies a limb or is otherwise accessible) of local abstractions of blood by leeches or otherwise, and according to circumstances, of moist warmth, such as may be produced by poultices, or of cold applications, especially of ice.

In speaking of Arteritis we have hitherto avoided all reference to those morbid conditions of the internal coat which Virchow regards as the results of endo-arteritis, and which are so frequently the seat of atheromatous and other degenerative changes. These consist of cartilage-like, slightly translucent circumscribed thickenings of the internal membranes, which vary in size, are more or less rounded in outline, and project in a wheel-like form into the arterial canal, and are found under the microscope to be due mainly to overgrowth of the cell-elements of the part. This latter fact shows that they are growths, and not, at all events in their earlier stages, degenerations; and since they are developed slowly, and at the same time (at all events when they are limited to the larger arteries) without obvious symptoms, they may doubtless be regarded as chronic inflammatory changes, allied to those which constitute cirrhosis.

Again, laminæ of true bone are occasionally formed in the lining membranes of arteries; as also in that of veins. And here, as in the last case, we have obviously to deal, not with degeneration, but with growth and development. The laminæ are the result of some slow inflammatory process, or at all events of some process related to inflammation.

(2) *Degeneration.*—Degenerative changes occur mainly in the internal and middle coats of arteries, and are for the most part attended with the deposition of fatty or of calcareous particles, and the gradual disintegration of the tissues in which the particles accumulate. Degeneration is sometimes a primary change, but more frequently probably the result of antecedent chronic endo-arteritis. In the former case the normal structural elements of the arterial walls immediately decay, in the latter the decay takes place in the substance of a something due to the overgrowth of such elements. This distinction, which Virchow clearly demonstrates, is undoubtedly a real one; at the same time it is, as yet at all events, of little practical importance; for the two conditions are constantly associated, and they lead to the same issues.

Fatty degeneration of arteries usually goes by the name of atheroma. Virchow, who points out the inappropriateness of this term as usually employed of arterial disease, endeavors to limit its use to those cases in which the degeneration occurs in the deeper layers of the internal coat and leads to the formation there of a cavity containing fatty detritus—cases in which, as a matter of fact, the degeneration is almost without exception secondary. And he calls those cases fatty erosions in which the process begins on the free surface, and in which as a rule the degeneration is primary. But for reasons which have been already referred to we shall employ the word atheroma in its ordinary sense.

Atheromatous degenerations appear in the early stage as opaque white, or yellowish-white spots, seated either in the substance of the internal coat, or between that and the middle coat. They are irregular in size and shape, and form elevations, which encroach more or less on the arterial canal, but present, nevertheless, a perfectly smooth and polished free aspect. They are at this time probably few in number and widely scattered, but there are certain situations which they specially affect, among which are the margins of the orifices of origin of branches. At a later stage, they have increased in extent, partly by the formation of fresh spots, partly by the coalescence of neighboring ones. Their thickness has at the same time increased and increased irregularly; so that the inner surface of the affected artery assumes an uneven and more or less tuberculated character. If a vertical section of the diseased arterial walls be now made, two or three varieties of condition will be observed, existing either alone, or more commonly in combination. In one case, the morbid process will be found to be entirely superficial. In another case the degenerations will be found to involve the deeper layers of the internal tunic. The membrane at the seat of disease will then be found thickened to two, three, or four times its original thickness, sometimes to a greater degree still, attaining it may be in the aorta a thickness of a quarter of an inch. It will be found opaque, yellowish-white, softer than natural, pulpy, or even semi-fluid in consistence; and it will, unless, the softening be sufficient to destroy this characteristic, be found to consist of superposed laminæ, arranged nearly parallel with the surface, and continuous at their edges with the laminæ of the healthy portion of the membrane. It will generally be observed, also, that a thin layer of comparatively healthy membrane separates the more diseased tissue from the canal of the artery. In another case, but more rarely, the morbid process attacks the middle coat, and may be limited to that coat.

The further changes which take place in atheromatous patches may be briefly stated. The more superficial ones soften

on the surface, become eroded, and then gradually disappear, leaving an ulcer-like pit or depression. The deeper-seated sometimes soften, and presently discharge their contents through a small orifice in the lining membrane into the channel of the artery. Sometimes it would seem that more or less of the atheromatous material gets absorbed, and the surface assumes in consequence an irregularly depressed, puckered appearance. Sometimes the diseased patches undergo cretification and the artery assumes in their situation the so-called "ossified" condition.

Calcareous degeneration of arteries generally accompanies, or rather follows on, atheromatous deposit. Sometimes, however, it seems to be developed independently of atheroma. Like atheroma, it generally commences in the internal coat, and may be limited to it. It forms hard brittle plates of irregular shape, and of various sizes, which in the first instance are limited for the most part to the deeper layers of the internal coat. At all events, in the early stage, the calcareous plates are separated from the current of blood by a thin layer of the lining membrane. After a while, however, this layer becomes also infiltrated, and the earthy matter comes into direct contact with the blood. The calcareous accumulations then form plates corresponding in their general shape to the portion of the artery in which they have been formed, having a tolerably smooth internal surface, and an outer surface, which is more or less nodulated; occasionally they form nodulated projections even on the free surface. Not infrequently erosion of the artery takes place at the circumference of such plates, and their rough edges become exposed to the blood; sometimes the plates become detached. In some of the smaller arteries more particularly, as in those of the extremities, calcification takes place in the middle coat sometimes primarily, sometimes in association with similar disease of the internal coat. In this case the calcareous matter tends to assume the form of rings, and obviously infiltrates the muscular fibres of the part.

Whenever atheromatous or earthy deposition takes place, even if it be confined to the lining membrane, some change in the nutrition of the middle tunic seems either to accompany it, or to follow quickly upon it. For the portion of this tunic which is subjacent to the deposit, even if it looks healthy, loses more or less completely both its elasticity and its contractile power. It need scarcely be added, that this loss of power occurs also, and even more markedly when the middle tunic is distinctly involved in degeneration.

Atheromatous degeneration consists essentially in the transformation of the cellular elements of the tissue into oil (olein) and cholesterine, and the softening and breaking down of the intervening parts. The oil (which is said to be olein) occurs in the form of globules of various sizes, some so small as to constitute mere molecular matter, others so large as to exceed the size of a pus-corpuscle. The cholesterine occurs in two conditions; in one it forms the ordinary thin imperfect rhomboidal plates; in the other (mixed probably with albumen), it forms globules of various sizes, which, like those in the so-called fatty kidney, present a cross when examined with polarized light.

At some period in the course of atheromatous degeneration, the degenerated patches begin often to be the seat of calcareous depositions, they shrink probably in thickness and gradually concrete. This earthy transformation consists in the gradual deposition, among the other degenerative products, but chiefly in the intercellular tissues of earthy globules, and at the same time, of the gradual disappearance by liquefaction, and absorption probably, of the cholesterine and oil. The earthy globules run together, blend into botryoidal masses, and at length form patches of earthy material which still give evidence of their mode of origin in the persistence of irregular angular cavities, in the nodulated character of their margins, and in the presence of free earthy globules in the neighborhood of these margins. The process of calcification here described is similar to that observed in other forms of pathological calcification, as on the surface of serous membranes in the teeth, and so on: it is similar also to the mode of calcification which Mr. Rainey has shown to be normal in shells, and in various other forms of healthy hard tissues including bones. Pathological calcifications of this kind in many tissues, and even in veins, have been shown to become occasionally converted into true bone, so that true ossification of an artery is by no means an improbable event. Virchow, also looking at the question from a somewhat different point of view, considers that the earthy depositions taking place in the lining membranes of arteries result in true ossification. When calcareous deposit takes place independently of atheroma, or so much in excess of oily deposit as from the beginning to mask the presence of the latter, the process of calcification is still essentially the same as that which has been described. The earthy matter is deposited, that is to say, in globular particles and masses, which, by further deposition on their surface, grow together. When the calcareous change takes place in the middle coat, the muscular fibres (as has before been pointed out) are the special seat of

this change and become converted into rigid spindle-shaped bodies.

All arteries are liable to the various forms of atheromatous and earthy degeneration which have just been considered, but they are not all equally liable. Again, although, when the disease is present, there is a tendency for it to become general, it by no means very infrequently happens that it is limited to one particular section of the arterial system, and attains there even a very advanced condition. Among the arteries specially liable to be affected, and in which as a rule the disease is found most developed, may be enumerated the aorta, the large trunks immediately springing from the aorta, the arteries of the brain, the coronary arteries of the heart, the splenic artery, and the arteries of the extremities. To these may be added, diffused arteries, such as those of stumps, and the arteries distributed in the walls of the senile uterus.

When atheromatous and calcareous deposits are at all extensive, they lead to many more or less important results. Thus, they produce often extreme irregularity of surface, and sometimes to these irregularities, clots become adherent, and occasionally undergo that central softening which will be found described in connection with cardiac clots often, especially in small arteries such as those of the brain, of the heart, or of the extremities), the lining membrane becomes so much thickened, that the passage of the blood becomes impeded, or even altogether arrested; sometimes, the deposit of calcareous matter becomes so extreme, that entire arteries are converted into rigid cylinders; sometimes again, the atheromatous degeneration is so abundant, that the softened lining membrane forms in the interior of the artery a number of undulating partially detached excrescences, projecting loosely into its interior. And it is worthy of remark, that most of the conditions just referred to occur not simply in arteries of large or of medium size, but even in the minutest arteries and in the capillaries. The latter two kinds of vessel, it is well known, often undergo the fatty form of degeneration; but they also occasionally undergo the purely calcareous form of degeneration. We have seen them in the brain converted into rigid needle-like tubes, in which, under the microscope, the calcareous matter consisted entirely of calcareous globules, which were deposited apparently beneath the lining membrane, and had coalesced more or less completely with one another.

The causes of these degenerative processes in arteries are obscure. It is certain that they constantly attend on old age; and then, according to the situation of the vessels chiefly affected, lead to cerebral apoplexy, to senile gangrene of the extremities, to angina pectoris, or other forms of cardiac disease, or to aneurisms. But it is also certain that they do not attend on old age exclusively; that they are occasionally observed in infancy; and that they not unfrequently lead to fatal results in adolescence and in the prime of life. There is reason to believe, that in the latter cases, the disease is due to certain cachexiæ, inherited or acquired; and that among these must be included the syphilitic, the gouty, and that which accompanies the various forms of Bright's disease. There is no doubt, indeed, that degeneration of arteries is a constant accompaniment of chronic renal disease; and Dr. Kirkes has endeavored to account for this, erroneously, we suspect, on mechanical grounds.

The symptoms, due to degeneration of arteries, simply, are very indistinctly marked. Persons whose arteries are in this condition live often for years while the disease is in progress, and yet suffer little inconvenience from it. The symptoms, indeed, which call attention to the presence of degeneration are almost entirely due to the conditions which complicate degeneration, to rupture, to aneurism, to obstruction of arteries, to enlargement of the heart, and so on. Yet, sometimes, when the superficial arteries are the seat of degeneration, the fact that they are so is indicated by their form and their rigidity; sometimes, when the cerebral arteries are affected, transient brain symptoms point to the presence of this condition; sometimes, when the aorta is the special seat of mischief, something in the rhythm, in the sounds, and in the dimensions of the heart, suggests the presence of aortic disease.

There is even less to be said in regard to the treatment of arterial degeneration, than in regard to its symptoms. All that need be stated on this head is, that the patient in whom such arterial disease is suspected should guard against all excitement, mental and bodily, that he should give way to no excess of any kind, and that he should endeavor to live quietly, and regularly, and temperately.

There is another form of arterial degeneration, known as the waxy, lardaceous, or so-called "amyloid" degeneration, which, so far as we know, has only been hitherto detected certainly in the capillary vessels, and in the minutest arteries. So far as this is a disease of the vascular system, it may be regarded in the light of a pathological curiosity only, inasmuch as it leads to no symptoms referable to the bloodvessels. It is seen constantly in the kidneys, in the liver, and in the spleen, when these viscera are the subjects of the form of degeneration in question. It seems, indeed, to commence in the capillaries and small vessels, and to in-

volve subsequently only the other tissues of these organs. The affected vessels become thickened and pellucid, and glassy-looking, and absorb iodine with characteristic readiness, assuming a peculiar reddish-brown hue. Virchow maintained some years since, that the matter deposited in waxy degeneration is cellulose; this view, however, has been disproved, and he has since retracted it. Dr. Edmund Montgomery demonstrated several years ago, at the Pathological Society, that the waxy or "amyloid" material contained much cholesterine, combined with albuminous matter. And he regarded it as identical, or nearly so, in chemical composition with the fatty-looking globules, acted on by polarized light, which occur amongst other globules in ordinary atheroma.[1]

Waxy degeneration is due apparently to the influence of certain conditions of system; especially of those which attend the later stages of syphilis, and chronic tubercular diseases, and of that which is induced by wasting suppuration, especially when the bones are involved.

(3) *Changes of Dimension.*—Alterations in the calibre of arteries take place very frequently, both as physiological and as morbid processes. As instances of the former may be mentioned, the enlargement of the collateral arteries of a limb in consequence of the obliteration of the main trunk, the alternate enlargement and diminution of the uterine arteries which attend the development and subsequent shrinking of the gravid uterus; and the atrophy which ensues after amputation in the arteries and stumps. The latter class of changes, however, the class (that is to say) which includes morbid dilatations and morbid contractions, is that which mainly interests us here.

(*a*) *Enlargement.*—An artery may be more or less generally enlarged, under which circumstance it is commonly spoken of as being dilated; or it may be enlarged at one or more distinct points, and the word aneurism is then employed to designate every such enlargement.

Dilatation. — Dilatation, in the sense just indicated, is not uncommonly met with in the degenerated arteries of old persons, especially perhaps in the aorta, and in the arteries at the base of the brain. The arteries thus affected are generally somewhat unevenly dilated, and present in consequence an irregularly nodulated or knobby contour. Dilatation is apt to occur also in a series of arteries belonging to some circumscribed locality, when by their aggregation they produce that condition which is commonly known by the name of cirsoid aneurism, or aneurism by anastomosis. The arteries in this disease, which are generally in their origin arteries of minute size, become generally and extremely dilated, they elongate and become tortuous, and ultimately by the formation of new anastomoses, or the enlargement of those which originally existed, communicate freely with one another in all directions. This dilatation does not appear to be attended with any structural disease of the arterial walls. They are, however, probably always very much thinner than those of healthy arteries of the same size.

Aneurism.—Circumscribed dilatations of arteries, or aneurisms, present many varieties of character dependent on their causes, the structure of their parietes, their form, and some other conditions. Hence, in reference to their origin, they have been called idiopathic or traumatic, in reference to their walls, true or false or diffused, and in reference to their form, sacculated or fusiform; and they have been classified by different authors in accordance with one or other of the plans thus indicated. The several names just enumerated are in common use. Of them, some are employed only in their literal sense, and scarcely need, therefore, any explanation; but two or three of them not only convey no very obvious meaning, but have been used in such different, nay opposite, senses by different authors, that their retention is a source of constant confusion. The names to which we here specially refer are those of true, false, and diffused aneurisms. By Scarpa, and other earlier writers, the term true was applied to those ordinary forms of aneurism in which, as a rule, the middle coat of the artery is deficient, or in which, at all events, the walls of the aneurism do not include all the layers constituting the arterial wall; and the term false was used of that comparatively rare form of aneurism, in which the walls are formed of all the arterial tunics. Hodgson, however, and subsequent writers on the subject, have, unfortunately, exactly transposed the use of these words, and have called that true aneurism which Scarpa described as false, and that false which he described as true. The terms true and false are still constantly employed, and generally inexactly. Obviously it would be convenient to drop them altogether. The term "diffused aneurism," again, has been used in various senses. Thus, by some, it has been used to signify that diffusion of blood which takes place when an artery, or an aneurism, ruptures into the cellular

[1] Since the above was written, Dr. Marcet has shown that the lardaceous material is essentially a form of albumen combined with much less potash and phosphoric acid, and with more soda, chlorine, and cholesterine than an albuminous structure in health. Path. Trans. vol. xxii. p. 1.

tissue—a condition to which, as Mr. Holmes properly insists, the term of ruptured artery or aneurism is properly applicable. By others, however, it is employed to designate those cases in which after such a rupture the patient survives sufficiently long for the space into which the blood has escaped to become circumscribed by inflammatory induration, and thus to be converted into a cavity maintaining a free communication with the ruptured vessel. In this latter sense we shall continue to employ it.

An ordinary or *Sacculated Aneurism* is a circumscribed dilatation of an artery, involving generally a well-defined area of the arterial walls, and limited generally to one side, or a portion of one side only. When small in proportion to the artery from which it springs, it may be more or less hemispherical, or thimble-shaped, its orifice being its broadest part. But sometimes while it is yet small, and generally when it becomes large, it assumes a rounded, or even completely globular form, and then opens into the artery with which it is connected by an oval orifice, less in breadth than the aneurism itself—sometimes very much less—and with its long diameter corresponding in direction with the channel of the artery. Sometimes from the enlarging sac yielding more readily at one or more points than elsewhere, the original aneurism may have other aneurisms, as it were, springing from it; sometimes the irregular resistance opposed to its growth by surrounding parts compels its enlargement in certain directions rather than in others; and from these, or other causes, aneurisms may come to assume almost any variety of shape. Their size varies within very wide limits. As a rule, it may be considered that the largest arteries yield the largest aneurisms; but this is a rule liable to exceptions, of which, perhaps the most notable is, that aneurisms springing from the commencement of the aorta are almost always fatal from rupture while they are still very small. They vary, roughly speaking, from the size of a child's, or of an adult's, head to that of a pea or less. Their orifice is generally round or oval, though perhaps somewhat irregularly so. Sometimes this is bounded by a well-defined or tumid margin; but sometimes the cavity of the artery gradually dilates into the aneurism, and the orifice (though still defined above and below by an abrupt line) is undistinguishable laterally, and formed simply by the divergence in this situation of the arterial walls. Various descriptions have been given of the composition of the walls of such aneurisms as are under consideration. Some authors, indeed, enumerate every conceivable variety of composition, from that in which it is formed of all three coats, to that in which it is formed of one coat only. We need not follow them. As a rule, an idiopathic aneurism, when in an early stage of formation, or while it remains small, presents a lining membrane which is continuous at the margins of the aneurismal orifice with the lining membrane of the artery; and if the artery be macerated, or is slightly decomposed, the membrane may be detached, and will be found to form a complete cast of the cavity. This lining membrane is generally thicker and softer than that of the artery itself, and perhaps more translucent; but it presents little or no structural difference. In larger aneurisms the inner surface is still generally, in the greater part of its extent, more or less polished; but though still necessarily continuous with that of the artery, becomes for the most part quite inseparable from the tissues which it lines. Very often, even in commencing aneurisms, the middle coat of the artery terminates in the thickened rim which bounds its orifice; sometimes it may be traced for a variable and even considerable distance on to the aneurism; and sometimes flakes, of what appears to be the middle coat, are scattered here and there irregularly over the whole circumference of the tumor. It may be stated that, as a rule, the middle coat is either deficient or presents traces of its presence only. The external arterial tunic is that which mainly and most uniformly forms the wall of any aneurism. At first probably it exists there in an unaltered condition. But as the tumor grows, and as this coat becomes stretched, additional connective tissue becomes added to it and incorporated with it, so that it becomes thicker and more resisting than the outer coat of the artery itself. And as the tumor continues to grow and presses upon neighboring organs and tissues, these or portions of them become compressed and indurated, and contribute to the formation of its walls.

A *Diffused Aneurism* may be of any size or any shape; it may originate as such directly from an artery, and not very unfrequently it becomes superadded to an ordinary aneurism as a consequence of rupture of its walls; indeed, it is a very common thing to observe, in large aneurisms, that the proper parietes are here and there, over some well-defined area, deficient, and replaced by condensed, blood-infiltrated tissue belonging to the parts in which the aneurism is imbedded. The parietes of a diffused aneurism consist simply of the tissues which happen to have limited the escape of blood, and which have become to some extent infiltrated with blood, indurated and, according to their age, more or less fibrous.

A *Fusiform Aneurism* is an aneurism in which the entire circumference, or the

greater part of the circumference, of an artery becomes for a limited and tolerably well-defined part of its length, dilated. Such an aneurism is generally more or less irregular in form, and indeed approximates in structure and in mode of formation to that condition which we have already described under the head of Dilatation. It may, however, like an ordinary sacculated aneurism, become very large; and this enlargement may be due either to its progressive general increase, or to the superaddition of a sacculated aneurism. As might be supposed, aneurisms of this kind may comprise in their parietes all the arterial coats, and probably in the beginning always do so; but, in their onward progress, their parietes naturally tend to become identical with those of the sacculated variety.

A modification of the fusiform aneurism is occasionally observed in the aortic arch in cases where the aorta is greatly contracted or obliterated at the point of entrance of the ductus arteriosus. The arch in these cases becomes generally and considerably dilated, sometimes so much so as to be capable of containing the fist; the walls undergo great attenuation, but (excepting accidentally) remain free from atheromatous or other unhealthy deposit, and whole.

Aneurisms may be produced by accident, or spontaneously; and perhaps not very unfrequently in the course of the complete development of an aneurism both causes may have operated in various degrees. In other words, accidental occurrences are more liable to produce aneurisms in arteries which are already diseased, and in a condition favorable to the spontaneous origin of aneurism, than they are in healthy arteries; and when aneurisms have already formed, their enlargement is not very unfrequently in part due to the occurrence of accidental ruptures. Accidental aneurisms are often met with in the popliteal artery, and other large arteries of the extremities, and in the aorta, as the result of a strain, in which probably the middle coat has been lacerated; and it may occur in any artery as the consequence of a wound. In cases of idiopathic aneurism, the essential cause of the disease is, doubtless, deficiency of resisting power in the middle coat of the artery at the seat of aneurism, as compared with the dilating power of the blood to which it is opposed. The operation of this cause is shown in its simplest form in the instance already quoted, in which an aortic arch, otherwise healthy, becomes dilated in consequence of the obliteration of its further extremity. But idiopathic dilatation and idiopathic aneurism most commonly arise in connection with atheromatous and ossific deposits. When these are all extensive, the middle coat, even when not obviously itself the seat of such deposits, loses both its contractile and elastic powers, and is then apt to yield under the constant impulse of the blood. It becomes attenuated, its fibres separate from one another, and, as the tumor becomes larger, either wholly or in part disappear. For a time, no doubt, the thickening of the inner coat, due to atheromatous or earthy change in it, protects the feeble middle coat from the injurious operation of the dilating force. But, after a while, the atheromatous inner coat becomes eroded and removed, or the atheromatous collection between it and the middle coat becomes discharged into the artery, or the bony plate becomes partially or entirely detached; and in one of these ways, or in some other way, the protecting influence is removed from the enfeebled middle coat, which then begins to expand. It is not, however, absolutely necessary that the removal of the internal coat should even in cases of atheroma precede the formation of aneurism; it is merely necessary that its own resisting power should not be sufficiently great to compensate for the loss of this power in the middle coat. It may be added, that any other condition tending to enfeeble the middle arterial tunic may be a cause of aneurism. Thus aneurisms arise as a result of inflammation involving the walls of arteries; and Tufnell, Holmes, and J. W. Ogle have all published cases in which aneurisms seem to have supervened in this way from embolia. Sometimes an artery may be opened by ulceration commencing from without, and thus a diffused aneurism may be produced.

When once an aneurism has begun it almost always undergoes gradual enlargement. Up to a certain point, as has been already shown, its parietes are for the most part derived solely from those of the artery out of which it has originated; but soon, in its progress, it begins to press on and displace neighboring organs, and then to appropriate them, as it were, in the formation of its walls. Its enlargement may be almost unlimited when it is developed in the substance of cellular tissue, as behind the peritoneum, or when its chief growth is subcutaneous; but when it projects towards serous surfaces, or presses upon mucous channels, it tends comparatively early to open into them. The ultimate tendency, indeed, of all aneurisms is to rupture. Often, as has been pointed out, partial ruptures occur into cellular tissue, and the additional cavities thus formed become circumscribed and taken into that of the original tumor. Often an aneurism opens by a sudden tear into a serous cavity, as that of the pericardium, the pleuræ, or the peritoneum; but perhaps more frequently

the rupture takes place into the sub-serous tissue, the blood accumulating therein and finally escaping thence by a subsequent rupture into the serous cavity itself. Often again an aneurism, after gradually pressing upon a mucous canal, opens into it through the formation and separation of a slough between them. In this way thoracic aneurisms frequently open into the trachea, bronchial tubes, or œsophagus; and abdominal aneurisms occasionally discharge themselves into some lower part of the alimentary canal. Occasionally, too (but this is usually a very late event), an aneurism bursts externally, having previously by its pressure caused the formation of a cutaneous slough. In rare cases an aneurism opens into an artery, a vein, or even into the heart itself. These latter events are most common in aneurisms of the aortic arch. But aneurisms produce mischief not merely by bursting and causing fatal hemorrhage. They are apt, by pressing on various organs, to produce effects referable to these organs; thus when developed on the under surface of the brain, they may produce eclampsia, or interfere with vision or with hearing, or lead to some other nervous phenomena; thus when developed in the thorax they may compress the trachea or the œsophagus, and so impede respiration or swallowing, they may involve the recurrent laryngeal nerve or the sympathetic, and induce certain characteristic symptoms which will be elsewhere detailed; and they may compress, and lead to obliteration of venous trunks, and thus induce dilatation of tributary and anastomotic veins, and œdema of the parts beyond the seat of obstruction. The effect of aneurisms on bones is curious. It is well known that, as they enlarge and press upon bones, they cause their erosion and gradual disintegration; that in this way a thoracic aneurism will destroy more or less completely the bodies of two or three vertebræ, and may even, in consequence of this destruction, burst into the vertebral canal; that it will destroy portions of the sternum, of the ribs, or of the clavicles. Whilst this destruction is in progress, the eroded surface of bone lies exposed in the aneurism, forming a segment of its walls; and not unfrequently a partially destroyed clavicle or rib is found projecting into the interior of an aneurism.

Occasionally aneurisms undergo spontaneous cure. To understand this it is necessary to consider the changes which take place within their cavities. Sometimes after death aneurisms are found empty, or filled only with perfectly coagulated blood or mere *post-mortem* coagulum. Sometimes, on the other hand, they are lined with laminated clots. These consist of a series of more or less imperfect layers, which lie one within the other, concentric with the aneurismal walls. They are pretty firmly attached to one another, but admit of separation, and the outer one is generally somewhat firmly united with the lining membrane of the aneurism. They consist of a toughish fibrinous material, of which the outer layers are thin and more or less buff-colored, the inner becomes thicker and softer and redder as they approach the channel through which the blood is still passing. The amount of these clots varies very much; sometimes there are merely two or three layers, so that the cavity of the aneurism is scarcely encroached on by them; at other times they fill the greater part of the cavity; and occasionally they are so abundant as completely to obliterate it: the innermost laminæ filling up the orifice of the aneurism and lying flush with the general surface of the artery, or even forming an irregular projection into its channel. Clots of this kind are very commonly observed in sacculated aneurisms, but they are not invariably present in them, and very large sacculated aneurisms are sometimes wholly free from them. Fusiform aneurisms are almost always without them. It need scarcely be said that these clots are distinctly deposited from the blood. The cause of their deposition is probably twofold. In the first place it is a recognized fact that blood tends to coagulate upon rough or diseased surfaces; thus upon mere atheromatous patches occurring in an otherwise healthy artery coagula are sometimes seen to have formed. In the second place, from the fact that these coagula are far more common in aneurisms which communicate with an artery by a comparatively small orifice, than in those which are mere fusiform dilatations, it may be taken for granted that stagnation of blood tends to encourage the deposition of fibrine. The formation of these clots depends then, probably, in some cases on one of these causes, in some on the other of these causes, but generally, doubtless, upon the concurrence of the two. It may be added that the presence of a layer of fibrine is probably the most efficient cause of all in determining fibrinous deposition; just as a nucleus of calcareous matter in the bladder attracts to itself similar matter which otherwise would have been retained in solution in the urine.

The formation of these clots in quantity no doubt frequently opposes an important barrier to the growth of an aneurism; and as has been shown they sometimes form so abundantly as completely to obliterate its cavity. In the latter way a spontaneous cure may sometimes be effected; but it is doubtful if such cures would generally prove permanent; for they are mostly observed in persons who have

been confined to bed some time previous to death, and the clots on which they depend are generally not very difficult of detachment. But indeed, on the other hand, it is occasionally in consequence of such a detachment that an aneurism becomes cured; the mass of clot, or a portion of it, shifts its position, and blocks up the artery upon which the aneurism is seated, leading at the same time to obliteration of the arterial canal and of the aneurism. Sometimes, again, an aneurism becomes cured by itself compressing the arterial tube either above or below its orifice.

Aneurisms may occur in any artery; but much more frequently in certain arteries than in others. Generally, it may be said that those arteries which are most prone to atheromatous and earthy degenerations are those which are most prone to aneurismal dilatation. Such are the aorta, especially its arch, the innominate, carotid, and subclavian arteries, the cœliac axis, the common iliacs, and the arteries at the base of the brain. But the proneness to aneurism is not solely in proportion to the proneness to degeneration; for in consequence of the comparative violence of the impulse to which arteries near the centre of circulation are exposed, these incur a special risk which those further removed escape; and again, in consequence of the position and connections of certain arteries (as the popliteal) they are exposed to danger of various kinds of violence to which many other arteries are exposed little, if at all. But we repeat that aneurisms may arise in almost any artery. Besides those named, or indicated, aneurisms are met with in the mesenteric artery and its branches, in the coronary arteries of the heart, in the ophthalmic artery, and in one case we found one in the course of one of the small arteries in the substance of the cerebellum. The pulmonary system of arteries is very rarely the seat of aneurisms; still, they are occasionally met with both in its trunk and in its branches. These arteries are more frequently dilated.

Aneurisms occur much more frequently during middle age and in declining years than in the earlier periods of life; but they are far more uncommon (in the laboring classes especially) between the ages of thirty and forty. And still younger persons, even children, do not wholly escape. They affect men more frequently than women. This is especially true of aneurisms of the extremities, which are mostly the result of violence.

The local indications of an aneurism, when it is seated in some accessible part, consist in the presence of a dilating pulsatile tumor, attended frequently when it is developed in the course of large arteries by a murmur synchronous with the systole of the heart. But in the case of many aneurisms, as of those within the skull, and in other inaccessible situations, these indications fail us entirely. The other symptoms of aneurism can scarcely be usefully considered here, inasmuch as they are in part those due to degenerated arteries generally, in part those which are due to the pressure of the aneurism on surrounding organs and tissues (and these symptoms will necessarily vary with the situation of the aneurism), and in part also those which may arise from their rupture (and these again must vary according to the part into, or in connection with which, the rupture takes place).

It is difficult also to consider usefully in a short space the treatment of aneurism. Indeed, we are compelled to pass over in silence the important subject of their surgical treatment. With regard to aneurisms which are beyond the reach of surgery, the essential objects to be held in view are, first, to prevent their increase; and second, to promote their obliteration by the deposition of clots within them. These ends can only be attained, so far as we know, by maintaining as much as possible rest of body and of mind, especially by quieting the circulation and preventing any such bodily movements as are likely to affect the aneurism immediately. When aneurisms approach the surface, the application of ice or other sedatives over them, galvano-puncture, and the introduction even of foreign bodies, as of threads or wire, may aid coagulation. At one time it was supposed that the frequent abstraction of blood, and the use of a low diet, were important adjuncts in promoting the cure or retarding the progress of aneurisms. Such was Valsalva's method. But modern physicians, and among these must be specially mentioned Dr. Stokes, have generally found reason to disapprove of this plan of treatment, and to prefer (as it seems to us with reason) the use of a nutritious and even of a generous diet, and abstinence from bleeding and other hurtful drains on the system.

Before concluding the subject of aneurism, it is necessary to consider two or three abnormal conditions of artery to which the term Aneurism is with more or less inappropriateness commonly applied. These are dissecting aneurism, aneurismal varix, varicose aneurism, and cirsoid aneurism, or aneurism by anastomosis.

Dissecting Aneurism occurs chiefly, if not entirely, in the aorta. It commences (generally in connection with degenerative disease, but sometimes when the vessel is simply dilated) in a rupture of the internal coat, through which blood is forced between the middle and outer coats, or rather (as Dr. Peacock has

shown) into the substance of the middle coat, where it accumulates, separating the internal and external coats from one another. Sometimes the resulting separation is slight, extending beyond the ruptured orifice; sometimes it is very extensive, "dissecting the coats of the aorta from the arch to the bifurcation, and extending even into the iliac arteries. Such an aneurism may after a while undergo a further rupture, either externally through the outer coat, or internally through the inner coat, and thus communicate by a second orifice with the interior of the artery. Sometimes the accumulation of blood in the substance of the walls is so great that the artery becomes obstructed.

Varicose Aneurism and *Aneurismal Varix*, are names which have been applied to the very rare conditions which follow on the establishment of a communication between a neighboring artery and vein. Such a communication may (as the result of disease) take place between an aortic aneurism and the superior cava, and (as the result of accident) between certain of the arteries and veins of the extremities. It used occasionally to result from wounding the artery through the vein, at the bend of the elbow, in the operation of phlebotomy. To the case in which the artery opens immediately into the vein, the term aneurismal varix is applied; to that in which an aneurismal cavity lies between the communicating vessels, the term varicose aneurism.

Of *Cirsoid Aneurism* we have already spoken. This is sometimes a congenital affection; sometimes arises without any obvious cause at various times after birth, and even in adult life; and it may appear in various parts of the body, as in the orbit, in the fingers, and elsewhere, but most frequently in the scalp. A remarkable case is recorded in the "Pathological Transactions," where nearly the whole of one of the lower extremities was involved.

(*b*) *Contraction and Occlusion.*—These conditions may be produced in a variety of ways, of which some have already been partially considered. Their more important causes we will here enumerate, leaving what little we have to say about the symptoms and treatment of obstruction till we come to consider the subjects of thrombosis and embolia. Occlusion of arteries is sometimes a congenital defect; sometimes it is the result of injury; sometimes it arises from the pressure of some hard tumor growing external to the artery, or from the artery becoming compressed by, or involved in, some carcinomatous or other growth; not unfrequently it takes place in the course of atheromatous and earthy degeneration, as a consequence either of excessive thickening of the lining membrane of the artery, or of the partial detachment of diseased patches, or of the formation of clots in connection therewith. To such obstructions of the arteries of the lower extremities, senile gangrene is perhaps always due; a similar condition of the coronary arteries of the heart leads now and then to local patches of degeneration and to rupture of the heart; and a similar condition of arteries of the brain, to circumscribed softening of that organ. Lastly, occlusion is not unfrequently due either to the formation of clots in arteries (thrombosis) at the seat of obstruction, or to the impaction in arteries of clots and other matters brought to them from some comparatively remote portion of the vascular system (embolia). These last two causes of obstruction will be considered under the head of "Thrombosis and Embolia."

DISEASES OF VEINS.

By John Syer Bristowe, M.D., F.R.C.P.

(1) *Inflammation.*—The term "Phlebitis" was formerly used, like the term "Arteritis," in a much wider and much looser sense, and much more inaccurately, than it is for the most part at the present day. It signified then a disease of the gravest import; for it was the common designation of most cases of what is now called pyæmia, of many obscure fatal cases in which it was erroneously supposed that inflammation had crept from some distant vein to the heart, and of many cases of what would now be termed thrombosis or embolia. It included also true Phlebitis, or inflammation of the veins: the disease which alone we now propose to consider.

Phlebitis, in this latter limited sense, is a morbid condition of vein, due some-

times to constitutional, causes, sometimes commencing from within the vein, sometimes from without, attended with important changes in the venous walls, and frequently with more or less of inflammatory mischief external to the vessel, and with coagulation of blood or other morbid phenomena within it.

The changes which indicate the presence of inflammation affect the outer vascular region of the venous walls in a far higher degree than they do the inner region which is devoid of capillary vessels. When inflamed, the walls become thickened (sometimes several times thicker than in health), congested externally, infiltrated with inflammatory exudation, and softened; sometimes suppuration takes place in them, sometimes they become disintegrated, or ulcerated and perforated, or entirely destroyed. If the inflammation be a chronic process, the exudation in the walls assumes a fibroid character, and the walls get indurated as well as thickened.

Generally while these changes are in progress, the connective tissue, with which the vessel is surrounded, partakes in the inflammatory processes, and consequently becomes congested, infiltrated, and, according to circumstances, indurated and brawny, or the seat of suppuration. At the same time, too, changes for the most part take place within the vessel. Blood coagulates there and clings to its lining membrane; a clot filling up, and it may be distending and occluding the vein, becomes thus established, and secondary phenomena due to the arrest of the circulation ensue. Clots thus formed undergo various changes, which will be afterwards more fully considered; sometimes they remain solid, and gradually contract and indurate; sometimes they soften and become reduced to a puriform fluid; and sometimes, we believe, they undergo actual suppuration.

The causes of phlebitis are very various, and influence to a great extent its degree, its character, and its progress. Thus, in some instances, it is due simply to the affected vessel being pressed upon, or surrounded, by some tubercular, cancerous, aneurismal, or other growth. Then generally the walls of the vein become greatly thickened and indurated, and its channel filled with a clot, which is usually hard and fibrinous, and closely adherent to the lining membrane. Sometimes, however, the vessel becomes flattened and so obstructed, and clots are formed only beyond the seat of obstruction. Sometimes, too, the portion of vessel chiefly involved becomes entirely destroyed and no longer traceable.

In many cases phlebitis is consequent on the vein being involved in inflammation (erysipelatous or other) affecting primarily the organ or tissue in which the vein is included; and the phlebitis then tends to partake more or less in the character of the surrounding inflammation. The sheath of the vein becomes first inflamed, and subsequently the inflammatory process invades successively the outer, middle, and internal coats; leading sometimes only to their congestion and to their thickening, but sometimes resulting in distinct suppuration. In the latter case the sheath of the vessel may become uniformly infiltrated with pus, or present more or less isolated collections of that fluid, and pus may be developed in the substance of the outer tunics; and occasionally as a result of this the venous walls undergo erosion or ulceration from without inwards, and a communication, or communications, become established between the interior of the vein and the parts external to it. When the inflammation extends thus from without, it sometimes happens that the outer walls only of the vein are involved; the walls become thickened in the aggregate, but the lining membrane remains smooth and polished, no coagulum forms, and the channel remains free. More frequently, however, the walls become affected in their whole thickness, and at the seat of the disease a clot forms, which adheres and blocks up the canal of the vein. This clot, as before stated, tends to soften either generally or in certain spots, and thus to assume the character of an abscess, or of a collection of pus bounded on all sides by coagulum, or by a layer of inflammatory lymph. There is no doubt that in nearly all these cases the pus-like fluid, and the more solid material bounding it, are simply the consequence of degenerative processes taking place in clots deposited from the blood. But occasionally true pus is certainly met with in this situation. Sometimes this is due to the opening of an abscess into a vein and the consequent conversion of a limited portion of the venous system into an abscess; but sometimes it is due, we believe, either to the development of pus from the lining membrane of the vein, or to suppuration occurring in the substance of the clot. This latter condition we have undoubtedly observed in cases of erysipelas.

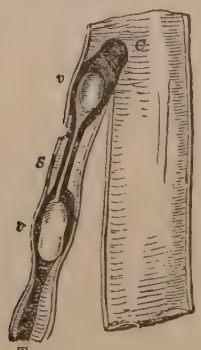
Again, phlebitis may result from local irritation, from poisoned wounds, or from other injuries. The inflammation then affects principally the sheath of the vessel, which becomes congested and infiltrated with inflammatory products, and sometimes undergoes suppuration; and it tends to travel along the sheath, so that presently a considerable length of vein, or a system of veins, may become affected. The same changes take place in this case in the venous walls, and in the interior of veins, as have been already considered.

In other cases, again, the inflammation doubtless commences from within, the outer portions of the walls of the veins becoming involved secondarily to the inner. This is the case, probably, when without any cause, originating, so far as we can see, in the walls themselves, thrombi form in certain veins, as in the iliac veins, for example, of phthisical patients. Such thrombi distend and occlude the vessels which subsequently only to the formation of these thrombi become thickened and give other evidence of inflammation.

There are yet other cases, probably, in which thrombi form, wherein the inflammation associated with their formation is a primary inflammation of the venous walls, dependent probably on some constitutional affection. Such cases, however, are necessarily obscure, and it is difficult to distinguish them from those which have just been considered.

We may add here a few words on the different conditions of system in which phlebitis is most apt to supervene, and on the veins which are most prone to be affected. Phlebitis frequently takes place, as has been already stated, in the soft tissues, in connection with various forms of inflammation, such as erysipelas, diffuse cellular inflammation and carbuncle. It takes place frequently, too, in connection with inflammation, and especially with suppuration, of bones and joints; under this head may be included phlebitis of the lateral sinuses of the skull, consecutive to suppuration in the ear. It is peculiarly apt to supervene in the puerperal state, frequently affecting the uterine veins, and frequently also

[Fig. 132



Thrombus in Saphenous Vein. S. Vein. T. Thrombus. C. Conical end projecting into femoral vein. At v, v, the Thrombus is softened. (Virchow.)]

affecting other of the systemic veins, but more particularly the iliac, producing, or aiding to produce, the condition known as phlegmasia dolens. Phlebitis, too, is very liable to occur in the course of phthisis and of heart disease. In the former of these affections especially, it is not uncommon to meet with it in the iliac veins, and in the venous trunks connected with the upper extremities. It is also discovered now and then, both in these diseases and in others, in the renal veins, in some of the veins connected with the liver, and other visceral veins. Lastly, we may point out that phlebitis is liable to arise in veins already otherwise diseased, as, for example, in varicose veins.

Just as inflamed arteries, which have become obstructed with clot, lead in consequence of this obstruction to secondary affections in organs and tissues to which they are distributed, so inflamed veins, when they have become similarly occluded, produce in consequence of their occlusion secondary phenomena in the regions which they drain. The obstruction of a vein necessarily leads in a greater or less degree to stagnation of blood, first in the veins, and next in the capillaries which form part of its system. This stagnation of blood, with the consequent dilatation of the vessels in which it is stagnant, may be the only secondary phenomenon; and it may soon disappear, provided either the vein becomes pervious again, or the anastomotic veins are sufficiently large or numerous to take on readily its suspended functions. In other cases the veins beyond the seat of obstruction become dilated and tortuous, and serum exudes into the connective tissue, causing anasarca. This dilatation of tributary veins, with localized anasarca, is well seen, in the legs when the obstruction takes place in the iliac veins, in the arms when it occurs in the subclavian and axillary veins, and in the head and neck and arms when the venæ innominatæ are the seat of disease. But the formation of phlebitic clots is attended with a class of dangers different from those which have just been considered, and different from any which attend the formation of clots in arteritis. I allude to the dangers of embolia, to the dangers, that is to say, of the detachment of the phlebitic clots or of fragments of them, of their transference by means of the circulation to other parts of the system, of their impaction in small arteries, and of the effects more or less serious to which they may then lead, in the areas which the obstructed arteries happen to supply; and I allude also to the supervention of pyæmia, which mostly, as before pointed out, has its starting-point in some local phlebitis taking place in connection with some unhealthy inflammatory process.

The local symptoms of phlebitis consist in pain and tenderness in the course of the affected portion of vein, with distinguishable fulness and hardness, if it be superficial, and often with redness or livid

discoloration in the integuments over it. These symptoms are attended also with more or less general febrile disturbance, and followed soon, generally, by distension of the veins beyond, and by anasarca. If the phlebitis be of that kind which, commencing in the venous sheaths, tends to spread along them from the smaller to the larger veins, the symptoms become altogether more grave; the pain and tenderness, the hardness and superficial congestion, which mark the seat of disease, are observed to spread and to become extensive; abscesses may form here and there around the veins, and may even lay open portions of them; the constitutional symptoms are those of high fever, which tends to assume the typhoid character.

The symptoms of phlebitis, however, in complicated cases, are very often difficult, or even impossible, to recognize; sometimes, no doubt, because the symptoms themselves are very trivial, frequently because the complications are of so grave a character as to include and mask them. The latter event takes place particularly when the phlebitis occurs as a part of erysipelas, or of diffuse cellular inflammation, or of other kinds of inflammatory affections; it occurs also when pyæmia, or even sometimes when embolia, supervenes on phlebitis.

But little need be said in reference to the treatment of uncomplicated phlebitis. If the vein be within reach, leeches, or ice (inclosed in a bladder or India-rubber bag) may, in the early stage of the affection, be applied along its course. At a later stage poultices or warm-water dressing may be serviceable. Rest should, of course, be enjoined. The constitutional treatment must be made to depend partly on the character and degree of the constitutional symptoms due to the disease, partly on the patient's general condition of health, partly on the special dangers to be apprehended and guarded against. If the general symptoms be trivial, little or no medical treatment is called for; but the more they assume a typhoid character, the more stimulants and nourishment, and medicines tending to the same end as these, are required. If the patient be suffering from tubercle, cancer, or heart disease; from the syphilitic or alcoholic or other cachexy; or from the effects of privation, the constitutional treatment must have special reference to these conditions, to the patient's general condition of health, and to the special dangers to be apprehended and guarded against.

(2) *Degeneration.*—Degenerative changes in veins are infinitely more rare than they are in arteries. Indeed, ordinary atheroma is probably never met with here; although it would seem that some degree of fatty degeneration, or at least of deposition of oily molecules, is not unfrequently to be seen microscopically in the walls of varicose veins, and in their valves. Depositions of earthy matter, although very rare, are yet, undoubtedly, of occasional occurrence. And it is worthy of remark that one of their favorite seats is the walls of varicose vessels; those vessels in fact in which fatty matter is now and then discovered. The calcareous deposits form irregular plates (originating apparently in or beneath the lining membrane) almost always considerably thinner than the calcareous plates in arteries, and often presenting a nodulated or "stalactitic" character towards the channel of the vein on which they tend to encroach. These calcareous plates have the same composition, and doubtless the same mode of development, as those in arteries. They consist mainly of carbonate and phosphate of lime, are deposited in globular masses which tend to coalesce, are formed in fact much as bones is formed, and are sometimes converted into unmistakable bone. They are usually observed in small amount, and often a single calcareous mass alone is discovered.

(3) *Concretions.*—Phlebolithes are globular or ovoid or irregular concretions, not unfrequently observed in the interior of veins, especially in the interior of veins which are dilated or varicose. Their most common seat probably is in the veins about the neck of the bladder, and other pelvic veins; they are met with, however, occasionally in the varicose veins of the lower extremities, and in the veins of the lungs and of the spleen. They are sometimes attached at one or more points to the lining membrane, are sometimes inclosed in a capsule formed by the obliteration of the vein above, and below the concretion, and sometimes lie loose either in the channel of the vein, or in a pouch connected with it. These bodies vary from the size of a horse-bean downwards, but are sometimes considerably larger. They are of a yellowish-white color, hard and calcareous, and on section appear to be made up of concentric layers like a urinary calculus. They consist chemically of carbonate and phosphate of lime, with some magnesia, and a variable proportion of organic material. How these bodies are formed has been matter of dispute. By some writers it was imagined they were formed externally to the lining membrane of the vein, into which they subsequently became prominent, then pedunculated, and finally detached. The more common opinion, however, and that which is doubtless the correct one, is that they originate in transformed clots. And, indeed, according to Rokitansky, there is commonly a roundish cavity or irregular

fissure within the nucleus, which is itself dry and of a rusty brown or dull yellow color. A clot of which this nucleus is the remnant is probably first formed; this undergoes degeneration, collapses, and furnishes a nucleus around which fibrinous layers from the blood are successively and slowly formed, and in which calcareous salts become deposited.

(4) *Adventitious Growths.*—Veins, like arteries, may be involved in carcinomatous and other growths, and like arteries, may become obliterated in consequence. But veins are additionally liable to become the actual seat of such growths. Thus, sometimes when a vein gets surrounded by a malignant tumor, this gradually invades its walls, and ultimately projects into it, and then either forms pedunculated or sessile outgrowths, or fills the channel, renders it impervious, and extends along it. Sometimes, again, secondary cancerous growths originate in the substance of the venous walls, and ultimately form polypi, hanging into the cavity of the vein. We have seen a good example of this condition, in connection with a trunk of one of the pulmonary veins, in a case where the lung was the seat of carcinoma. The same thing may occur also in connection with tumors which are not malignant. Thus in a case of myeloid disease of the humerus, we once found the veins ramifying in the deltoid muscle, and some of the large veins of the upper arm, filled with myeloid growth, identical with it. Tubercle, so far as we know, is never met with in the veins.

(5) *Changes of Dimension.*—Veins undergo changes of dimension both from physiological and from pathological causes. To the former class of causes are to be attributed that ordinary enlargement of the veins which attends the growths of the body, that enlargement which takes place in anastomotic branches when a trunk is obstructed, those changes of dimension of the uterine veins which attend the corresponding change of dimension of the uterus itself, that diminution of size which occurs after amputation in the veins of stumps, and so on. To the latter class of causes must be assigned the varicose condition of veins which is so often met with, and some forms also of contraction of veins.

(*a*) *Enlargement.*—Varicose veins are veins which have irregular dilatation. Veins thus affected are elongated and unnaturally tortuous: their diameter is larger than natural, and often very considerably larger; and at the same time they present, irregularly distributed over their surface, hemispherical dilatations or aneurism-like pouches, and occasionally even flask-like diverticula communicating with them by a comparatively small orifice. In veins furnished with valves the dilatations occur more particularly in connection with the sinuses immediately above them. Coincidently with this dilatation, the walls become attenuated, and the valves (at first perhaps simply rendered inefficient by becoming too widely separated from one another) become atrophied and shrivel up. The middle coat of veins is probably that (like the middle coat of arteries) by which dilatation is in the normal condition chiefly opposed; and it is therefore in connection specially with the yielding of this that dilatation occurs. Its fibres become divaricated; and, as already pointed out, they undergo some degenerative process indicated by the deposition in them of fatty particles. Sometimes, on the other hand, the walls of the dilated veins become thickened instead of attenuated, a change which is due to thickening of the outer coat. Dilated veins, like dilated arteries, may, by their pressure on parts external to them, cause the absorption of these parts. In this way the enlarging vessels sometimes approach the surface of the skin, or that of some of the mucous membranes, and even cause the absorption of bone. We have pointed out already that varicose veins are specially liable to become inflamed; they are specially liable also to have coagula deposited within them. These may occur as casts of veins, blocking them up; or may be produced in the dilatations only; sometimes, according to Rokitansky, laminated coagula like those of aneurisms are formed within the pouches. Phlebolithes, as we have already pointed out, are sometimes found in varicose veins, and are probably derived from such clots as have just been described. The varicose condition affects veins very variously both in extent, in degree, and as regards the order of veins affected. Sometimes the larger veins only are thus dilated, and when such veins are seated in some superficial part, large tortuous, soft, knotty, bluish cords, projecting above the normal level of the skin, indicate their presence. Sometimes the smaller veins only are varicose, and then, if superficial, they form in different situations pencils as it were of reddish or bluish vessels, larger than natural, thickly clustered and radiating, it may be, from a point or line. Sometimes one or two veins only, or portions of them, present the varicose condition; sometimes nearly all the veins of a limb may be involved in the disease; sometimes there seems a still more general tendency for the veins to become dilated.

The essential cause of the dilatation of veins is the same in principle as that of

the dilatation of arteries, namely, inability of their walls, from deficiency in them of resisting power, to withstand the pressure which the blood within them exercises upon them. This inability may depend either on actual loss of power in the walls, or on loss of power due to their over-distension, in consequence of impediment to the onward flow of blood. It may depend therefore on constitutional causes, or on accidental local conditions of disease. But there is an additional circumstance, which has a very important influence in determining the formation of varices, and in increasing their bulk when once they have begun, that is, the pressure to which veins are exposed in relation to the height of the column of blood they have to support. It is, we need scarcely say, a well-known hydrostatical fact, that the pressure exerted by a fluid (whether circulating or still) against any point in the walls of a receptacle containing it, is in exact proportion to the height of the column of fluid above that point; and hence it is clear that those portions of the venous system, which are most dependent or nearest the ground, are exposed to greater pressure from within than those which occupy a higher situation. No doubt this is to a great extent counteracted, in those veins which from their position are most subject to its operation, by the presence in them of valves. Still, it is not wholly counteracted even in healthy veins; and in those which have become sufficiently dilated to render the valves within them useless, it must act to its fullest extent. It is, doubtless, owing in great measure to the operation of this cause that varicose veins are specially frequent, and become specially large, in the lower extremities.

Varicose veins sometimes get well spontaneously, especially after the disappearance of the cause which has induced them. Sometimes a cure is effected by the gradual return of the enlarged veins to their normal size, sometimes by an attack of inflammation in them leading to the deposition of a clot, and to their obstruction and subsequent obliteration. Sometimes they remain more or less stationary. But more commonly, if left alone, they continue to enlarge, and tend ultimately to burst externally and to cause dangerous, even fatal, hemorrhage. The presence of varicose veins leads also to unhealthy conditions of the parts with which they are connected.

The symptoms due to varicose veins may be gathered from the foregoing account of their morbid anatomy. They consist locally of enlargement of the veins, swelling and œdema of the associated tissues, aching pains, sometimes itching, tendency to inflammation of surface, eczema, excoriation and ulceration. To these may be added special symptoms, due to the impairment of function of any organ or part with which the varicose vessels happen to be connected.

It is not easy to lay down any general rules with regard to the treatment of this affection. If it arise in constitutional conditions, there is reason of course to suppose that constitutional treatment may be of service. Looking upon the disease then as a disease indicating debility, it is natural to assume that tonics and other remedies tending to give strength may prove serviceable. And in the present state of our knowledge it is no doubt wisest to act on this assumption. If, on the other hand, it depend on any local impediment to the flow of blood, it will be right, if possible, to remove or counteract this local condition. Further, it is generally desirable to support the affected veins, by the application over them of uniform and moderate pressure, in order to obviate the debility of their walls; and, as far as possible, to maintain the part affected in either the horizontal position, or some other position tending to relieve the vessels from undue pressure of their contents.

Before leaving the subject of varicose veins, it is desirable to say a few words in regard to some of the situations in which they chiefly occur, or in which their occurrence is specially interesting. The most common seat of varicose veins is doubtless the lower extremity. Both legs seem to be equally liable to be affected. The disease here presents great varieties; sometimes a small portion of a vein, or a small group of veins only is affected; sometimes nearly all the veins are involved; and all varieties are met with between these extremes. There seems little doubt that the superficial veins are those which, as a rule, are primarily and principally affected; but it has been pointed out by Briquet and by Callender that the points of chief distension are those in which the superficial veins are joined by branches from the muscular and other subjacent tissues; it has also been pointed out that in many cases the deeper-seated veins are equally involved with the superficial veins, and that occasionally the disease is actually limited to those which are deep-seated. Among the conditions tending to produce varicose veins here, and especially, of course, tending to produce them in such persons as are constitutionally predisposed to their occurrence, may be enumerated, occupations requiring long-continued maintenance of the erect position, pregnancy, ovarian diseases, and generally the presence of abdominal tumors producing pressure either on the vena cava or on the iliac veins, and probably also cardiac and other diseases in which the free passage of blood from

the right to the left side of the heart is impeded. Varicose veins in the leg produce swelling of the leg and sometimes a slight degree of anasarca; they are apt also to lead to inflammatory conditions here, to induration of the cellular tissue, to congestion of the surface, excoriation and eczema, and not unfrequently to ulcers. These ulcers are generally very intractable. It is in the leg, too, more than anywhere else, that rupture of the dilated veins is liable to occur. In the treatment of varicose veins of the leg the constant use of support is very essential. Generally the constant wearing of an evenly applied bandage from the foot to the upper part of the thigh, or of a well-fitting laced stocking, is indicated. Sometimes the obliteration of the trunk veins by surgical means becomes imperative. The details of surgical treatment we do not profess to discuss, but we may enumerate the more important surgical expedients, such as the application of pressure in the course of a vein, the tying of veins, the formation of an eschar over them, the introduction of foreign matters such as threads into them for the purpose of producing coagulation, and the like. When the surface of the leg is congested or inflamed, the limb should be maintained at rest and in the horizontal position, and cooling and such other applications as are applicable in superficial inflammation should be employed. In the treatment of varicose ulcers support and pressure are of the first importance. If a vein burst, the wound should be treated exactly like the wound after the ordinary operation of phlebotomy. The patient, too, should be placed in the horizontal position and the limb elevated.

Varicose veins not unfrequently arise in the spermatic cord, producing the disease termed varicocele. This occurs almost always upon the left side, and is supposed to be determined here in part by the great length of the left spermatic vein, and in part by the fact that this vessel opens into the renal vein instead of opening like its fellow directly into the cava. The veins in this disease become very large and tortuous, and are described as feeling like a bundle of worms; the testicle to which they belong becomes the seat of much aching pain, and ultimately its nutrition becomes impaired, it shrinks in size and undergoes atrophy. In this affection the testicle should be supported; and, as in the former case, it may be necessary to employ operative measures.

Hemorrhoids or piles have generally been regarded as a varicose condition of the hemorrhoidal veins; and there is no doubt that they are often produced, and always increased, by constipation and by any other condition which impedes the passage of blood along these veins, or the veins into which these empty themselves. Hemorrhoids, however, are not so much varicose veins as they are a hypertrophic condition of the mucous membrane, or of the integuments, in the neighborhood of the anus, attended with much congestion of the capillary and other minute vessels, and in some degree also with a varicose condition of the veins.

Varicose veins may occur in other situations besides those which have been specified, and lead to grave results. Thus, there is reason to believe that the veins of the stomach and of other portions of the alimentary canal occasionally become dilated, and induce dyspeptic and other obscure symptoms. We have met with a case in which the veins of the œsophagus were varicose, and where death was due to the rupture of one of them. The veins about the bladder and the prostate are not unfrequently varicose. Those of the labia pudendi certainly often become varicose in the course of pregnancy. Again, varicose veins are occasionally observed in the upper extremities, and even in the neck. As the result of actual obstruction of veins, they may in fact be met with in almost any situation; we have already specially pointed out some cases of this kind, and we may add to the list, the occurrence of such veins in the abdominal parietes in certain cases of hepatic or of splenic disease, and the dilatation of veins in the neck and upper extremities which follows upon obliteration of the innominate veins, or vena cava descendens, produced by aneurismal or other tumors in the neck. Rokitansky says that the veins of the pia-mater become varicose in drunkards.

(*b*) *Occlusion.*—Occlusion of veins has been already considered incidentally in various parts of the foregoing account of the diseases of veins. It has been shown to occur sometimes as the result of phlebitis attended with the formation of clots; to be produced sometimes by the pressure of a tumor growing external to the vein, sometimes by the growth of carcinomatous or other tumors into the interior of the veins. The results of occlusion and its symptoms have also been considered incidentally. They are principally, dilatation of the veins on the distal side of the seat of obstruction, enlargement of anastomatic veins, œdema of the tissues through which the passage of blood is obstructed, and such further phenomena as attend, on the one hand anasarca, on the other hand varicose veins.

CARDIAC CONCRETIONS.

By John Syer Bristowe, M.D., F.R.C.P.

The condition of the blood in the heart's cavities, at the time of death, as to quantity and quality, and the relation which its varieties of condition bear to the cause of death, are necessarily matters of much pathological interest; they are matters also of some practical interest; and, in both these points of view, have been made of late years the subject of a good deal of careful observation. Yet it is curious that nearly all systematic writers on heart-diseases—and even the more recent of them—have either passed this subject over in almost complete silence, or, if they have been tempted to enlarge upon it, have displayed a lack of knowledge in regard to it which the character of their works in other respects would scarcely have permitted us to suspect.

It is not proposed, in the limited space which has been necessarily allotted to the present article, to treat exhaustively the subject under consideration, still less to criticize at any length published opinions upon it which seem to us erroneous. It is intended rather to give a general brief account of the whole subject, and to enlarge upon those points only which seem to have some special interest and importance.

Morbid Anatomy.—At the time of *post-mortem* examination, the cavities of the heart may be found either contracted and empty, or dilated and containing an amount of blood proportionate to their dilatation. And, in the latter case, the blood may be found either quite fluid, or imperfectly coagulated, or coagulated and moulded to the surfaces with which it is in contact, or in the form of "globular concretions," or in a tough laminated condition, or mixed, it may be, with concretions (emboli) brought hither from remote parts of the vascular system. It may be added, that two or more of the above conditions frequently coexist in the same case, and such of them as are not incompatible with one another, even in the same cavity. It is desirable, however, to discuss them separately.

Emptiness of Cavities. — The cavities which are most frequently found empty of blood are the ventricles. This emptiness is much more common in the left ventricle than in the right; but not unfrequently both cavities are in the same condition. The auricles are generally full, if not distended.

Fluid and semi-fluid Blood.—The blood contained in the heart's cavities may be fluid or semi-fluid. That is to say, it may be nearly as fluid as when it freshly escapes from a vein, it may be more or less treacly, or it may contain floating in it soft loose masses of dark-colored imperfectly-formed clot. It is in these cases usually that the lining membrane of the heart and large vessels becomes stained with the coloring matter of the blood.

Moulded Clots.—The blood may have undergone more or less complete coagulation. Its condition, however, varies very considerably in different cases. Sometimes the coagula are small, and the cardiac parietes are contracted, or collapsed upon them; sometimes they are large and distend the cavities to the full; sometimes they exist therein alone; sometimes they are surrounded by a greater or less quantity of serum or of uncoagulated blood; sometimes they are of a uniform red-black hue; sometimes they are partly decolorized; sometimes they are wholly fibrinous.

These coagula, whatever their color or consistence, are always accurate, or nearly accurate, casts of the cavities which contain them, and are generally attached to the surface, not by any organic connection, but by being dove-tailed, as it were, with its inequalities. Those of the corresponding auricles and ventricles are continuous through the auriculo-ventricular opening; and are, moreover, prolonged to a greater or less extent into the venous and arterial trunks. In the aorta they sometimes extend throughout nearly its whole length, in the pulmonary artery to its smallest subdivisions. The prolongations into these tubes are cylindrical, but generally a good deal smaller in diameter than the tubes themselves; and those portions of them which correspond to the arterial valves, have always the form of the valves distinctly impressed upon them.

Moulded coagula are sometimes, as has been just pointed out, of a uniform red-black hue. They have then much the appearance and consistence of black-currant jelly, are soft and tremulous, and consist of a uniform mixture of chiefly the

fibrine and the red corpuscles of the blood. Sometimes they are partly decolorized, or, in other words, a partial separation of their component elements has taken place. The fibrine may then have separated, much as it does in the formation of the buffy-coat after bleeding, producing a thin almost colorless layer on that portion of the clot which, during its formation, has lain uppermost. Or it may happen that the whole surface of the clot is fibrinous, while the interior remains colored. Sometimes again, and this is the most remarkable case, the whole, or nearly the whole, of the clot is fibrinous. Such clots are sometimes loose in texture and watery and retain more or less of the coloring matter of the blood: sometimes straw-colored, jelly-like, and elastic; sometimes close-grained, buff-colored, opaque, and tough.

Moulded clots may be found in all the cavities of the heart: but those which are fibrinous are chiefly found in the ventricles, and more frequently in the right ventricle than in the left; they may, however, occur in the left ventricle even when they are absent from the right. The clots which extend into the larger vessels are generally in the greater part of their extent identical in character with the cardiac clots, with which they are continuous. But even when almost purely fibrinous, they mostly pass off at their extremities into colored clots. This is especially the case with those occurring in the veins.

Softening Clots.—Softening clots, globular concretions, purulent cysts (for all these names, and many others, have been applied to the bodies now about to be described) are coagula, which have undergone changes, by which they have become converted into roundish masses, softened for the most part internally into a puriform fluid, attached firmly to the parietes, and occupying, with scarcely an exception, those portions of the cavities which lie out of the direct current of the blood.

These bodies may occur singly in a cardiac cavity, or in considerable numbers, and may vary from the size of a pin's head up to that of a pigeon's egg. They are almost always attached to the surface, and generally spring distinctly from the interspaces between the carneæ columnæ, or the musculi pectenati. Their attachment to the surface, though sometimes in part due to slight adhesions, is mainly effected by this entanglement with the fleshy columns; and, indeed, where several of these bodies are present in a cavity, they are probably always continuous with one another by means of processes extending beneath those muscular bands which are attached to the cardiac walls by their extremities only.

Their free surfaces are sometimes smooth, sometimes more or less ribbed; generally they have an opaque buff-color, but they may present more or less of a brick-red tint, or may be variegated with irregular streaks of red and white. On section, they present considerable variety of appearance. Sometimes they are solid throughout, and repeat on their sectional surface, the characters already displayed by their external aspect; more commonly, however, they are more or less softened, at one time converted into a thin-walled cyst, at another time broken up irregularly into a series of small intercommunicating cavities. The walls of the cyst are identical in character with the substance of the unsoftened concretions, but their inner surface is soft and flocculent. The contained fluid is thick and puriform, and varies in color from a pale buff to a brick-red, or even chocolate, hue.

Under the microscope the solid portions of these concretions are found to consist of a fibroid network similar to that of ordinary coagulated fibrine. "This, however, is intermixed with a large quantity of granular matter, which renders the fibroid structure more or less indistinct. They contain also oil, compound granular cells, and a few imperfect cells which appear to be the remains of white corpuscles. In some cases there are many altered blood-corpuscles, and now and then solitary and clustered needle-like crystals." "The puriform contents of the cysts present considerable variety as to their microscopic elements. When white or buff-colored, they consist almost solely, if not solely, of molecular matter, oil, and broken-down corpuscles, with which are frequently mixed compound granular cells, and colorless acicular crystals. When presenting a brick-red or chocolate hue, they exhibit, in addition to the elements just mentioned, numerous blood-corpuscles, more or less altered, and consequently more or less indistinct, and occasionally also ruby-colored, rhomboidal, hæmatoid crystals." In one instance which we have met with, the fluid contents consisted almost entirely of well-marked pus-like corpuscles.

It is asserted by Rokitansky,[1] that these concretions are almost always limited to the left ventricle. This, however, is an error. They do, it is true, occur here more frequently than in any other one of the cavities of the heart; but they occur much more frequently in all the other cavities collectively, than they do in the left ventricle. They are not unfrequently found in two or three cavities, and occasionally in all of them at the same time. In order of frequency they affect, we believe, first, the left ventricle; second, the

[1] Path. Anatomy (Sydenham Society's Translation), vol. iv. p. 217.

right ventricle; third, the right auricle; and last, the left auricle. With regard to their position in the cavities of the heart, there is no doubt that, with scarcely an exception, they occupy those situations which are most favorable to the stagnation of blood. "In the auricles they chiefly affect the auricular appendages, and in ventricles they almost always occupy the spaces and interstices between the carneæ columnæ."[1] Occasionally, they are developed around some of the chordæ tendineæ; and one or two cases are recorded, in which they have been found detached.

Laminated Clots.—Laminated coagula, such as are found in aneurisms, are of very unfrequent occurrence in the heart, and of recorded cases, the most common are certainly those in which the coagula have formed in the interior of aneurismal dilatations, or of actual aneurisms developed in connection with the ventricles. Still a small number of cases have been met with, in which cavities otherwise healthy have become almost obliterated with coagula of this kind. We are acquainted with this condition only as affecting the left auricle, secondarily, to extreme contraction of the mitral orifice—under circumstances, therefore, not dissimilar from those which lead to the formation of such coagula in the interior of actual aneurisms. In one such case,[2] where the mitral orifice was so contracted as scarcely to admit the tip of the little finger, the left auricle was greatly dilated, and full of firm laminated coagulum, which formed two perfectly distinct masses—one extending from the auricular appendage backwards, the other forwards from the posterior and inner part of the cavity. They were slightly adherent to the parietes, and were in contact with one another by their free surfaces, which were consequently flattened. The cavity of the auricle was thus obliterated, or, at least, reduced to the imperfect and irregular channel left between these mutually compressed masses.

Embolic Concretions.—Sometimes, though very rarely, concretions which have been moulded in remote parts of the vascular system are found entangled amongst ordinary cardiac clots. The only instance in which we have certainly met with this condition was a case of scarlet fever with sloughing of the tousils. In this case small opaque shreds, and portions of cylinders, consisting entirely of corpuscles resembling those of pus, were found in the right ventricle, embedded in ordinary *post-mortem* clot. We have never met with tubercle or carcinoma in the heart thus conveyed.

ETIOLOGY.—It is obvious that at the moment of death the heart's ventricles are either contracted, or in various degrees dilated; and that their emptiness or fulness of blood at the time of *post-mortem* examination must be in great measure determined by these conditions. It is obvious, too, that in those cases in which the cavities are found full of blood from the stagnation in them simply of the blood arrested in its course at the moment of death, the state of this blood as to fluidity or coagulation must depend in great measure upon the conditions of sickness under which death has occurred. All these are matters of interest, and worthy of investigation; but they are not included within the scope of our present article, and we are compelled therefore to dismiss them.

But of the *clots* found in the heart after death, some have evidently been formed in it during life, and may possibly have had some influence in destroying life. Amongst these must be included such as are wholly or for the most part fibrinous, globular concretions, and laminated coagula. How and by what means these are produced we have now to consider.

Clots moulded to the cavities of the heart, if they be of uniform consistence and of a uniform reddish-black color, have doubtless in all cases been formed *post mortem*, from fluid blood contained in the cavities at the time of death; and the same explanation doubtless holds good of those cases also in which such clots present a layer of fibrine (a buffy coat in fact) on that part of their surface which has lain uppermost. In all cases, however, where the clots are purely fibrinous, or where the fibrinous element is in excess, or where the fibrine which has separated occupies any other position than the upper surface, the separation of the fibrine and therefore the coagulation of the blood, must have taken place during life, while the blood was still in process of circulation. That this must be so is evident from the consideration that there is no means by which stagnant fluid blood can, in coagulating, manifest separation of fibrine except upon its upper surface, still less achieve the perfect separation of its fibrine from all its other constituents. It is further proved by Dr. Richardson's examination[1] of these fibrinous clots, which shows that the amount of fibrine contained in them is several times greater than can be accounted for by the quantity of blood

[1] The passages included within inverted commas are quoted from the author's papers, "On Softening Clots in the Heart," contained in the 7th and 14th volumes of the Pathological Society's Transactions.

[2] See Path. Trans. vol. xi. p. 65.

[1] See Dr. Richardson's Lectures in the British Medical Journal for 1860.

which the heart's cavities are capable of containing. It must not be forgotten, however, that all the fibrine met with in such cases in the cavities of the heart rarely, if ever, exceeds the amount of fibrine contained in the blood which passes through the heart in the course of half a dozen beats; and that, therefore, the whole of a large fibrinous clot may have been whipped out of the blood in the course of the minute or two of circulation which precedes death. It is certain, then, that such clots are formed during life, but by no means clear how long their formation actually takes. Some of them are doubtless, as has been just suggested, formed in the course of the few moments immediately preceding death; but it is exceedingly probable that others have taken some considerable time in their formation.

What it is that determines this coagulation of the blood during life is by no means easy to determine. Dr. Richardson, in the paper before referred to, enumerates several classes of cases in which fibrine is peculiarly apt to be deposited during life in the heart's cavities, the most important of his classes being that of acute inflammatory affections, including pneumonia. We have no doubt that, in all the cases which he enumerates, fibrinous clots are not unfrequently observed; but, indeed, they are constantly met with in the *post-mortem* room, not only in them, but in almost every form of disease. They are by no means constant, even in cases of pneumonia. Mr. Henry Lee believes them to be characteristic of purulent infection of the blood. This, however, is obviously an error; for while it is common to meet with them in cases where no such infection can be suspected, in cases of pyæmia they are altogether exceptional. We shall not pretend to offer any satisfactory explanation of the causes of the formation of these *ante-mortem* clots in some cases, and their non-formation in others. But we may admit generally with Dr. Richardson, that there are diseases in which, from some cause or other, there exists a tendency to the separation of fibrine; and further we may suggest that slowness in dying may in such cases to some extent determine this separation.

With regard to the formation of the rounded concretions, which are generally softened in their interior into a puriform fluid, many fanciful theories have prevailed. Thus it has been supposed that they are softened tubercle, or pus, conveyed to the heart from a distance and there encysted. Their contents, however, are never tubercular, and rarely if ever purulent; and although they may be occasionally met with both in phthisis and in pyæmia, their occurrence in these diseases, especially in the latter of them, is exceptional. Again, it has been supposed that their formation is due to local endocarditis. But, in reply to this supposition, it may be pointed out that they rarely, if ever, accompany undoubted cases of endocarditis; and, moreover, that they are almost without exception found in just those parts of the heart's cavities in which true endocarditic deposits probably never take place. That they are merely altered clots is evident, both from their microscopical constitution and from their identity, in the changes which they undergo, with clots formed in other parts of the body, whether in the vessels or by extravasation. It is evident, too, that the condition in which they are found after death is the result of processes which must have required days, or even weeks, and possibly a still longer time for their completion. It is evident, further, from the mode in which they are attached to the cardiac walls, that they must have been formed in the position in which they are discovered *post mortem*. The cases in which they are most commonly observed are cases of heart disease, of renal disease with dropsy, of chronic bronchitis, and of chronic phthisis, cases in which death is often slow, or in which struggles, as it were, between life and death are apt to occur from time to time for some while before death actually supervenes. It seems probable that the foundation of these concretions is laid at one or other of these moments of seemingly impending death, by the coagulation at that time of blood in the cavities of the heart; that the patient rallies from his apparently moribund condition, and that the clots, at once the evidence and the result of that condition, remain; that the clots then during the remainder of the patient's life gradually undergo those changes, which clots in the brain and elsewhere are liable to undergo; that they become torn into smaller masses, probably in consequence of the constant movements of the cardiac walls; that these masses become rounded partly in consequence of the contractile force inherent in the fibrine of which they chiefly consist, partly by the attrition to which they are exposed by the constant movement of the fluid blood over their surface, and that after a while their interior undergoes softening and disintegration.

The laminated clots, of which I have quoted an example from the left auricle, are evidently of slow growth, and originate long anterior to death. Indeed, they are obviously formed on the same principle as that which determines their formation in aneurisms, and are the result, as in aneurisms, of a slow process of deposition from the blood.

Symptoms and Effects.—We now have to consider the important question, whether the clots which are formed in the

heart prior to death have any effect in producing death, and if so, whether their presence during life can be recognized by any characteristic symptoms. All who have enjoyed much clinical, combined with *post mortem*, experience of disease, will admit, as regards the vast majority of cases in which *moulded fibrinous clots* are discovered in the heart, that their formation has taken place during the process of dissolution, and as a part of that process, that their formation has been unattended with symptoms referable to themselves, and that if they have exerted any influence adverse to life, it can only have been in the sense of preventing any tendency to rally, in other words, of confirming the act of dying. It does not however necessarily follow that cases do not occasionally happen, in which in the course of certain forms of disease, or even apparently in health, such clots form, and by the impediment which they oppose to the circulation of the blood through the heart, cause death. To the consideration of this point we will shortly recur. Meanwhile we will discuss the effects of those forms of clot—*globular and laminated concretions*—which beyond all dispute must have been in existence a considerable time anterior to death. In regard to the laminated concretions, it may be stated, we think with some degree of certainty, that their presence is attended with no special symptoms. No doubt they add to the embarrassment of the heart, but they add only to the embarrassment of an already embarrassed organ; they merely increase the severity of symptoms which are already severe, and therefore, if combined with mortal disease, merely cause the disease to anticipate its final series of events. It is worthy of remark, however, that life is maintained in these cases even when the auricular cavity is so encroached on as to be no more than a mere channel between the veins and the auriculo-ventricular opening. Globular concretions equally as a rule produce no special symptoms. Certainly they are constantly met with *post mortem* in cases which have been under continued observation, and have presented no special symptoms indicative either of their formation or of their presence. No doubt their presence in the cavities of the heart has a tendency on the whole to impede the action of the heart and to affect the circulation injuriously, especially if they be large, or if they occupy certain situations. But impediment, real or virtual, to the circulation, probably always exists prior to the formation of these bodies, so that their addition tends to aggravate symptoms already established rather than to develop new ones. It seems not improbable that they may now and then interfere with the normal function of some of the valves, and so lead to the production of endocardial murmurs, but with this result we have no practical acquaintance. Again, two or three cases are recorded in which they have been found detached in an auricle, and lodged in, and thus obstructing, a contracted auriculo-ventricular orifice. And it has been surmised that they may occasionally become ruptured, and, by the escape of their contents into the circulating blood, produce symptoms of pyæmia. It may be considered, therefore, that, excluding a small number of quite exceptional cases, the presence of these clots in the heart cannot be recognized by peculiar symptoms, but may be surmised, and often correctly, in cases where the struggle between life and death has been greatly protracted, especially if the patients be suffering from any of the diseases in which morbid anatomy shows that these clots are chiefly produced. It is important, however, to bear in mind that although these concretions doubtless originate in ordinary fibrinous coagula, there are few if any cases in which the moment in which they were first formed can be even approximately determined.

Let us now return to the question as to the influence of moulded clots in producing death. It seems to us that with the facts before us—first, that coagula of this kind are constantly observed in the *post-mortem* room as the mere accompaniment and result of the dying process; second, that (as has been shown) whole cavities may become obliterated by coagula without directly causing death; third, that (as has also been shown) in the majority of cases in which it can be clearly demonstrated that concretions have been formed some considerable time before death, their formation has not produced marked symptoms, we ought to require very strong testimony indeed to convince us in any case that concretions found in the heart at the time of death, have caused death, still more to convince us that those clots which resemble in every point the clots which are the mere result of dying, have had this effect. It is no doubt convenient and seductive, when we meet with a case of fatal illness, to be able to point to some obvious pathological phenomenon attending it, and to believe that in that we recognize the cause of death. Not long ago fatty heart furnished the ready explanation of most sudden deaths, now fibrinous concretions in that organ begin to rival fatty heart in popularity. We have no hesitation in stating our conviction that in the great majority of cases which have been recorded of death from the formation of fibrinous concretions in the heart, these concretions have been developed in the ordinary way, and have had no more to do with the death of the patient than the rigor mortis has. We are not pre-

pared to deny that death is sometimes actually caused by such a deposition of fibrine, but we can state positively that no such case has come under our observation, and we believe that the great majority of recorded cases are cases in which the sequence of events—cause and effect—have been misunderstood and transposed. In the remarks which have just been made, we wish it to be distinctly understood that we refer exclusively to cardiac concretions, and not to concretions blocking up the pulmonary artery and limited to that artery. This latter subject will be discussed under the head of "Thrombosis and Embolia." We may add, for the convenience of those readers who are interested in the subject, that they will find an ingenious account of the symptoms which are supposed to attend the formation of moulded cardiac concretions, in Dr. Richardson's lectures, already more than once referred to in the course of this article.

THROMBOSIS AND EMBOLIA.

By John Syer Bristowe, M.D., F.R.C.P.

The terms "Thrombosis" and "Embolia" (or Embolism) have been introduced by Virchow: the former, to signify the coagulation of blood in arteries or veins during life: the latter, to signify the transference either of clots, or of other solid matters appearing within the vascular system, from one part of that system to another part, in the direction of the circulating current, and by means of it. These subjects have already been partly considered under the heads of "Pyæmia," "Cardiac Concretions," "Diseases of Arteries," and "Diseases of Veins;" we propose however here to treat them as a whole, and, as they are intimately related to one another, to combine their description in a single article.

The local phenomena, which attended the coagulation of blood in the vascular system during life, are essentially the same, in whatever part of that system coagulation takes place; and the changes which clots undergo are also essentially the same, whether the clots occur in the arteries, in the veins, or even in the heart. These have been already in great part described. A clot, consisting either of nearly pure fibrine or of all the solid elements of the blood combined, forms, and is moulded as it forms, to the surface against which it lies; to which also it is from the beginning, or becomes ere long, adherent. The changes which such a clot undergoes in the course of time vary. They consist, sometimes in its gradual contraction and organization; the fluid matters become absorbed, the cellular elements disintegrate and disappear, the fibrinous portion undergoes condensation, and ultimately the clot becomes converted into, or replaced by, ordinary connective tissue. They consist sometimes in the softening and breaking down of the clot internally; the central parts become converted into a thick puriform fluid, sometimes red, sometimes nearly white, consisting chiefly of disintegrated cell elements merely—such as granular matter, oil, cholesterine, débris of corpuscles, and perhaps hæmatoid crystals; the clot may thus come to form, either wholly or in part, a mere fluid-holding bag, in which condition it may remain for a considerable time; but gradually, here as in the former case, the fluid portion undergoes absorption, the contents dry up and the cyst-walls collapse upon them. Sometimes, further, clots become the seat of calcareous transformation; and this may occur both in those which have softened internally and in those which have maintained the solid form; particles of earthy matter are deposited, which gradually increase in number, and ultimately by their aggregation transform them into calcareous masses. There is reason, as has been already shown, to believe that phlebolithes are formed in this way; and phlebolithes are occasionally the seat of true ossification.

A full account of these clots, as they are met with in the heart's cavities, has already been given.

In the aorta and pulmonary trunk they are unfrequent, except where they are met with as fibrinous or more or less colored cylinders prolonged from the interior of the respective ventricles or from the neighborhood of the semilunar valves. Such clots, like the corresponding cardiac

clots, are manifestly formed during life, though often during the last moments only of life, they always present the impress of the arterial valves, and, though generally much smaller than the channel in which they lie, sometimes almost fill it. Older clots are sometimes observed in the aorta. These are isolated roundish concretions, adherent to the surface (mostly if not always in connection with points of disease), projecting into the canal, but not materially obstructing it. In other arteries, however, and especially in the smaller arteries, these clots generally form solid cylinders, equal in diameter to the vessel in which they lie, adherent more or less to its surface, and more or less completely obstructing its channel. The obstruction, however, is generally at first incomplete, and the constant impulse of blood against the proximal extremity of the clot tends gradually to force a certain proportion of blood between it and the arterial walls. In this way, blood in small quantities flows for a time through irregular channels over the surface of the clot; soon, however, it coagulates there, and thus the original clot becomes incrusted with an irregular layer of more recent coagulum, the vessel becomes distended and the occlusion becomes complete. Further, additional coagulum tends to be deposited in connection with the extremities of the primary clot; this deposition ceasing generally, on the proximal side, at the point of anastomosis nearest the seat of obstruction.

[Fig. 133.

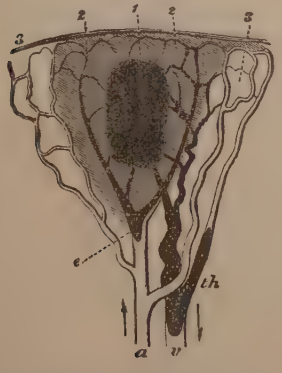


Diagram of a Hemorrhagic Infarct.—*a*. Artery obliterated by an embolus (*e*). *v*. Vein filled with a secondary thrombus (*th*). 1. Centre of infarct which is becoming disintegrated. 2. Area of extravasation. 3. Area of collateral hyperæmia. (O. Weber.)]

The brief account which has just been given, applies with almost equal exactness to the clots which form in veins. A very important additional fact, however, in regard to these, has been demonstrated by Virchow; the fact, namely, that they tend to increase by the deposition of fresh clot at their proximal end, until the vein becomes filled up as far as its mouth, and that, in continuation of this process (from the blood which passes along the trunk vein with which the obstructed vein communicates), additional coagulum is gradually added to that which has been already deposited, until from the plugged orifice there projects into the interior of the trunk a rounded mass of laminated coagulum, which may attain a very considerable size.

The causes of Thrombosis have already been to some extent considered. Sometimes the coagulation seems to be consecutive to mere stagnation of blood, or sluggishness of circulation, occurring in certain conditions of disease. Such probably is the case in regard to softening clots in the heart; such probably, also, is the case in regard to the clots which plug certain of the veins in phthisis and some other affections; and such, also, doubtless, is the case when arteries, leading to districts of disease in which the capillaries are obstructed, become themselves filled with clot. Sometimes the coagulation is determined by mere roughness of the surface over which the blood passes. This is observed when isolated clots become adherent to atheromatous patches, and when extensively atheromatous or ossified arteries become obstructed with clots. Sometimes the thrombus is the result of inflammation of the walls of the artery or vein in which it is found, the coagulation of the blood being consequent on some altered relation between the walls of the vessel and the blood within them. Indeed, phlebitis and arteritis are probably the most frequent causes of thrombosis. Further, the formation of clots in arteries and veins takes place occasionally in the course of some cachectic conditions of system, such as those connected with syphilis or anæmia. It may, of course, be a question, whether or not the coagulation in these cases even may not be the result of inflammation.

The embolus, or obstructing mass, which, conveyed from a distance, becomes lodged in some vessel, and, for the most part, occludes it, may consist of any solid material derived either directly from the blood, or from the walls of certain parts of the vascular system. But in order for Embolia to take place, it is obvious that the solid matter must be formed in such a situation as shall admit, first, of its detachment, second, of its conveyance by means of the circulating fluid, third, of its impaction in some vessel too minute to admit of its further progress onwards. Hence it follows that an embolus must always be looked for in some part of the pulmonary or systemic arterial system, or in the portal system, and that

its source must be sought for, as a rule, either in the veins or in the heart; occasionally in the large arterial trunks.

A very frequent source of Embolia is the formation of clots, from whatever cause, in the systemic veins. Thus, sometimes phlebitic or other clots become dislodged, then swept away in mass by the blood, and ultimately fixed in some part of the pulmonary arterial system. More commonly, however, as Virchow has shown, thrombi, which have become friable in texture, undergo disintegration, so that fragments only of them become detached and carried onwards; and he has shown that this process chiefly occurs in connection with those bulbous extremities of thrombi which project from the occluded veins into the trunks with which these veins are connected. In this case the emboli are likely to be numerous and small; and it is likely that many of the smaller twigs of the pulmonary artery will be occluded rather than one or two of the larger branches only. The same processes may take place in connection with the pulmonary veins and systemic arterial tree; but Thrombosis in these veins, and Embolia from this source in the systemic arteries, are certainly not common.

Another frequent source of emboli is furnished by the interior of the heart. Sometimes, there is reason to believe, the softening clots, which have been already described, and which it has been shown may be detached, may be conveyed onwards, and produce arterial obstruction. The most frequent source by far, however, in connection with the heart, is the vegetations which form on the valves in the course of rheumatic and other inflammatory conditions. Sometimes the soft granulations of recent inflammation, which are often clustered, and often loosely attached, become separated and washed away with the current of blood; sometimes, on the other hand, fragments of older concretions break off—concretions which have become condensed and friable, or tough. In either of these cases, it may happen that the detachment of one or two large masses may lead to the blocking up of some arterial trunk, or (and this is more commonly the case) that the separation of a number of small fragments may cause the occlusion of one or many small vessels. Since cardiac vegetations, as the result of inflammation, are much more common on the left side of the heart than on the right side, it necessarily follows that Embolia originating from the heart is much more commonly met with in the systemic arteries than in the arteries of the lungs.

The last source of emboli is the crumbling away or disintegration of atheromatous or cretaceous deposits, such as one meets with in erosion or ulceration of the lining membrane of the heart or arteries. The minute particles, or, at least, some of them, not unfrequently become arrested, like other emboli, in the arterial twigs, and lead to their obstruction. As in the last case, Embolia from this cause is much more frequently observed in the systemic arteries than elsewhere.

An embolus, of whatever kind it may be, and whatever may be its source becomes swept along with the blood, from one vessel to another vessel, until it reaches one which from its size opposes a bar to its further progress; in this it becomes wedged, and obstructs it wholly, or almost wholly. Very often it becomes fixed on the spur formed by the bifurcation of an artery. Soon after its arrest the embolus becomes invested in clot; blood gradually coagulates on its proximal side as far back as the next anastomosis of the obstructed artery; it coagulates, also, generally on the distal side as well, sometimes only as far as the next branch, sometimes throughout the whole series of vessels which the primarily obstructed artery supplies. In the case of obstruction of vessels from emboli, equally as in that from thrombi, blood will, in a greater or less degree, insinuate itself for a time between the embolus and the vascular wall; this blood, however, generally soon coagulates, and becomes continuous with that at either extremity of the embolus. The clot in which the embolus thus becomes imbedded, and which completes the obstruction which the embolus had begun, may vary in character from an ordinary colored clot to a purely fibrinous one. At first it is easily distinguishable from the embolus within it; but, like other deposited clots, it soon undergoes degenerative changes, and gradually approximates, more or less, in character to the embolus itself, which may thus be rendered quite incapable of separate recognition.

Generally, at all events in the more obvious cases, the embolus is a definite mass, which becomes impacted in the form in which it had separated. Not unfrequently, however, especially when the more minute arterial twigs become obstructed, these twigs are found distended with an aggregation of small angular masses, which would seem to be either the débris of a larger embolus, or the minute particles due to the erosion or crumbling occurring at the seat of the primary affection. It seems not improbable that both of these latter explanations may hold good of certain cases; that sometimes, as Virchow suggests, an embolus which has become impacted breaks up into fragments, under the constant pressure from behind to which it is exposed, and that these fragments become

then driven onwards into the minuter vessels beyond; that occasionally also, perhaps the débris, separated from an eroded surface, become in the process of separation loosely cemented together by coagulum, and the soft mass thus formed becomes driven into the minuter arteries, and moulded to them.

The local indications of the presence of a thrombus and of an embolus are as nearly as possible identical. In both cases the vessel becomes obstructed and distended; in both, inflammation of the walls (even if it did not previously exist) becomes excited, and they undergo thickening; and in both, if the vessel affected be superficial, it may be felt to be enlarged or hardened, and will probably be found to be painful and tender. The most important results, however, of these affections are those which depend on the obstruction of vessels—results which manifest themselves in connection with the parts which lie on the distal side of obstruction, and especially in those regions, the vessels of which are tributaries or effluents of those which are obstructed. It has already been pointed out generally what these results are. When a vein is obstructed, the return of blood is prevented in a greater or less degree, the vessels behind become distended with blood, dilated—it may be varicose—and the tissues behind become the seat of congestion and of dropsical effusion. When an artery is the seat of obstruction, the direct passage of blood to and through the parts to which the artery leads becomes arrested. In some cases, of course, this disturbance of the circulation is temporary only; anastomozing branches enlarge, and, by transmitting an increased quantity of blood, make up between them for the loss to the circulation of the obstructed vessel. But in all cases disturbance takes place to some extent; the nutrition of the districts to which the artery leads becomes impaired, the blood stagnates in its vessels, these become distended with blood, owing to the reflux into them from neighboring vessels, and not unfrequently ruptured so that extravasation takes place; sometimes inflammatory processes, with exudation of lymph, or suppuration, supervene; and very often molecular death and gangrene ensue.

The gravity of the consequences of obstruction of vessels, whether arising from Thrombosis or Embolia, depends partly on the size of the vessel obstructed, partly on the importance to life of the organ or part to which the blocked-up vessel belongs, partly on the suddenness with which occlusion takes place. Obstruction from one or other of these causes may affect any vessel. As regards obstruction of veins, all that might otherwise have needed to be said here has already been said under the head of Phlebitis, from which venous Thrombosis can scarcely be separated. But some of the more important cases of obstruction of arteries we propose now briefly to consider seriatim.

Obstruction of Arteries of Heart, Liver, Spleen and Kidneys.—In each of these cases, what Virchow terms capillary emboli, derived from cardiac granulations, are far from uncommon. Occasionally large masses become impacted in certain of their vessels; and occasionally (in the kidneys especially), without any cardiac disease whatever, the larger number of the principal arteries of the organ become obstructed by the formation of firm fibrinous adherent clots within them. The consequences of obstruction of the arteries of the organs just enumerated are not very dissimilar from pyæmic affections of the same organs; they may consist in all (but more especially in the heart and kidneys) of minute abscesses, about as large perhaps as pins' heads, or of minute abscess-like points, in which the puriform matter is composed of mere disintegrated material; they may consist also in all (but more particularly in the spleen, kidneys, and liver), of so-called "fibrinous blocks." These vary in size, but are often very large—a cubic inch or so in bulk. They present, for the most part, well-defined limits, vary between a pale buff color and a deep brick-red hue, and appear to consist essentially of the normal tissues infiltrated with some of the elements of blood. The pathological phenomena here described are very frequently observed, but the symptoms to which they give rise are not very obvious.

Obstruction of Arteries of Brain.—Far more important than the obstructions which have just been considered are the obstructions which, as Dr. Kirkes originally showed, take place occasionally in the arteries of the brain. The obstructions here are, without doubt, frequently embolic, and take place distinctly in the course both of chronic diseases of the aortic or mitral valves, and of acute inflammatory attacks of these parts. When such is the case the embolic fragment is almost invariably discovered in one or other of the middle cerebral arteries, or their branches—according to Dr. Kirkes, most commonly in the artery of the right side. But the obstruction also frequently takes place wholly independently of heart disease, and without any possible source of Embolia, and is clearly then due to Thrombosis of the affected vessel. Such Thrombosis may occur in any of the arteries at the base of the brain, and we have seen a case in which the cerebral portions of both internal carotids and the basilar artery became thus successively obstructed. The

effects of obstruction of arteries on the portions of brain-substance to which the obstructed arteries lead consist, in the first place, of patchy congestion, and, in the second place, of marked softening, attended with yellowish, or slightly greenish discoloration, and the appearance of numerous compound granular cells. The affected portions of brain are usually small and circumscribed, but are sometimes extensive and diffused. They are most frequently observed (in connection with obstruction of the middle cerebral artery), in the corpus striatum.

The symptoms of obstruction of the cerebral arteries always appear suddenly. The patient is seized with a kind of "fit," sometimes apparently epileptic, sometimes syncopic, but sometimes unattended with either convulsions or loss of consciousness; and on emergence from this sudden attack he is found to be hemiplegic. The symptoms which succeed are little, if at all, different from those which attend on apoplectic attacks; they vary in different cases, as the latter vary, and need not be detailed in this place.

Obstruction of Arteries of Extremities.—The arteries of the extremities become occasionally obstructed by clot. Sometimes no doubt these obstructions are embolic; but far more commonly we believe they depend on arteritis. In the lower extremity, where this condition is most frequently observed, the seat of obstruction is usually, we believe, either the femoral artery in the neighborhood of the origin of the profunda, or the popliteal artery. Occasionally obstruction takes place simultaneously in the corresponding arteries of opposite limbs. The formation of a plug is generally, perhaps always, ushered in by acute pain at the spot which the plug occupies. This is followed by impaired circulation in the limb beyond, loss of pulsation in the distal portion of artery, pallor, coldness, numbness, and ultimately, it may be, gangrene. In certain cases, however, the patient recovers from the effect of the occlusion, as patients recover from that produced by the ligature of an artery.

Obstruction of Pulmonary Artery.—The branches of the pulmonary artery are the recipients of all the emboli derived from the systemic venous system. Embolia, therefore, is in them of common occurrence. Thrombosis also not unfrequently takes place in them. The blocking-up of minute arterial twigs in pyæmia leads, as has been already shown, to the morbid changes in the lungs indicative of that malady. The formation of clots in some of the arteries, in the course of mitral and other forms of obstructive cardiac diseases, are constant accompaniments of pulmonary apoplexy and probably in most cases precede and cause it. Occasionally the impaction of a large embolus in one of the larger branches of the pulmonary artery, or the development therein of a thrombus, leads to inflammatory, and other, mischief of a comparatively large portion of the lung, or even of an entire lobe. We have seen such a case, in which a large portion of lung, the main artery of which was obstructed by an embolus, had become pneumonic, its terminal bronchial tubes had become destroyed by suppuration, and thus converted into irregular cavities, and the investing pleura had become inflamed. The most serious cases, however, are those in which, either from Thrombosis or Embolia, the whole, or nearly the whole, pulmonary circulation becomes suddenly arrested, and rapid or sudden death ensues. A good many cases of this kind are recorded, and they seem to be comparatively frequent among puerperal women, though they are by no means confined to them. Occasionally no doubt the transference of a clot which has formed in one of the systemic veins is the cause of this sudden obstruction, either by the clot itself blocking up the pulmonary trunk, or by serving as a nucleus, around which, after its impaction, further coagulation takes place. Much more frequently, however, we believe, the formation of the obstructing clots commences, and becomes completed, in the arteries in which they are discovered after death. And we ground this belief on the fact that in many cases, where death from this kind of obstruction is unquestionable, the pulmonary clots form an almost continuous system, accurately, or nearly accurately, moulded to the channels in which they are found, and in a greater or less degree adherent to them. It is a very remarkable fact that, in some at least of these cases, the deposition of clots in the pulmonary arteries must have taken place, and been completed to the verge of almost total obstruction during a period of nearly perfect health, and that the patient's sudden death has been due either to an accidental shifting of the clot, or to the coagulation of the streamlets of blood, by the persistence of the flow of which between the older clots and the arterial walls life had hitherto been maintained. The clots here referred to commence sometimes immediately above the pulmonic valves, sometimes at the bifurcation of the pulmonary artery, sometimes separately in each branch of the artery, and are continued more or less uniformly, and for a greater or less distance, along their ramifications. They possess all the characters of clots formed some while before death, and adhere here and there to the arterial walls.

The symptoms which indicate serious obstruction of the pulmonary artery are sudden embarrassment of respiration,

great dyspnœa, with coldness, and pallor, and clamminess of skin; pallor, not lividity, of face; feebleness, rapidity and irregularity of pulse, followed by death, sometimes after an interval of several days, sometimes quite suddenly. As an example of the formation of clots in the pulmonary arteries, or rather of their presence in these arteries, during apparent health, and of sudden death resulting from their presence, we may quote the following case:—

A female servant, thirty years of age, was admitted into St. Thomas's Hospital under Dr. Bristowe's care, on the 18th June, 1860, having suffered from slight symptoms of pleuritis on the right side for about ten days. On admission there was distinct evidence of dry pleurisy on the affected side, but the symptoms soon passed away; the patient got apparently well, and was about to leave the hospital; but before she could leave it, and while assisting in the wards, she was attacked suddenly with faintness and gasping for breath, and in a few minutes was dead. Her death took place seven days after admission.

The body was spare, and without œdema. The pericardium was healthy, the heart of moderate size, with parietes, lining membrane and valves all healthy. All the cavities contained dark fluid blood, without a trace of coagulum. The right lung was adherent to the parietes by an exceedingly delicate, easy-to-be-broken-down membrane. The organ was small, and its lower lobe partially collapsed. Its surface was studded with irregular, and in some cases, large patches of subserous hemorrhage. Its tissue was crepitant throughout, though less so below than above. The bronchial tubes contained a large quantity of frothy mucus. The branches of the pulmonary artery distributed to the organ were in the greater part of their extent filled with decolorized and slightly adherent cylindrical coagula. They commenced, not in the trunk of the pulmonary artery, but in the division of it leading to this lung, formed casts of all the primary branches of this division, and were prolonged thence into many of the secondary and subsequent branches. The coagula, however, did not in all instances form parts of a continuous system; but in many cases the smaller branches, and in a few the larger branches, were occupied by coagula of the same kind as, but having no continuity with, those prolonged from the root of the lung. The clots were for the most part cylindrical, and accurate casts of the vessels in which they lay; still, here and there they presented slight irregularities and enlargements. They were for the most part adherent, though slightly so, to the parietes; but here and there were free, leaving passages between them and the arterial walls; which, together with the intervals entirely free from clot, were filled with dark-colored fluid blood. All the clots presented a reticulated fibrinous surface, and a central black-currant-jelly-like axis. They had evidently formed prior to death. The pulmonary veins were empty. The left lung was in precisely the same condition as the right. There was no important disease in any other organ. The blood in the systemic arteries and veins was generally fluid; but in the left internal iliac vein, extending partly into the common iliac, was a cylindrical coagulum, not completely filling the vessel, but adherent to it, and presenting characters identical with those of the clot in the lung. Again, in the left innominate vein, a mass of coagulum was discovered, completely blocking it up; this was unadherent, and was found, on unravelling it, to consist of a branching system of partly decolorized clots, which could not have been formed there, but must have been carried thither from some of the smaller tributary vessels.

The above case, it may be added, is not adduced to prove that death may take place from the formation of clots within the pulmonary arteries. For the case is one in which there is room for difference of opinion in regard to the source of these clots. We believe, nevertheless, that in this case the clots found in the pulmonary arteries were formed in them; and we believe it partly in consequence of the form and structure of the clots, partly because there is no valid ground for disbelieving that such clots may be deposited during life, as well in the pulmonary arteries as in the systemic veins.

DISEASES OF THE PULMONARY ARTERY.

By R. Douglas Powell, M.D., F.R.C.P.

In any systematic consideration of the diseases affecting the pulmonary artery, that vessel must be separated into two portions, one external to, and one within the lungs. Disease of the pulmonary artery before its distribution in the lungs is so uncommon that, in practice, a murmur most audible over the region of this vessel is regarded as of hæmic origin, or this hypothesis failing, as attributable to some congenital defect about the heart, or to pressure from without, and it is only by this method of exclusion that we force ourselves to admit that the disease has its seat in the vessel itself.

Atheroma.

Etiology.—The etiology of atheroma and aneurism of the pulmonary artery does not essentially differ from that of corresponding affections of the aorta; but in consequence of the pulmonary vessel being more deeply seated, more lax in its capacity, and therefore less liable to direct injury or effective strain, the results of those constitutional influences, gout, alcoholism, syphilis, which lead to atheroma, are much more rarely developed. The milder degrees of atheroma—fatty degeneration of the intima—are, however, not unfrequently seen associated with those heart and lung diseases—mitral constriction and regurgitation, pulmonary fibrosis, or emphysema with hypertrophy of the right ventricle of the heart—which permanently increase the tension of the pulmonary circulation.

The association of such atheromatous patches in the pulmonary artery with those diseases which cause more or less persistent difficulty in the smaller circulation, and which have as their common accompaniment a more or less increased venosity of blood, is, as has been well pointed out by Drs. Wilks and Moxon, a strong argument against the supposition that this artery is protected from atheroma by virtue of the dark blood circulating through it. That the pulmonary artery is not wholly insusceptible to deeper lesions, however, is apparent from a case, to be presently cited, in which, under the combined assaults of rheumatism, alcoholism, and hard work, with a very strong suspicion of syphilis, this vessel, in common with the aorta, became affected with atheromatous disease resulting in loss of substance. Dr. Hope[1] refers to a case in which the vessel was dilated and rigidly ossified, even beyond its primary divisions in the lungs.

Symptoms.—No symptoms have hitherto been traced as referable to atheroma of the pulmonary artery.

Dilatation. Aneurism.

Under circumstances of great pressure within the pulmonary circulation, as in cases of marked narrowing of the mitral orifice, with great hypertrophy of the right heart, a certain general enlargement of the pulmonary artery may take place. Dr. Sydney Coupland has recorded the case of a naval pensioner, aged 75, in whom there was an extreme degree of this general dilatation of the artery and its branches. In this case the main trunk was found dilated to a circumference of 6⅛ inches, the valves being incompetent and the walls of the artery greatly thinned. There was in this case great hypertrophy and dilatation of the right heart, marked emphysema of the lungs, and patchy superficial atheroma of the intra-pulmonary branches. Although some degree of patency of the foramen ovale was also present, Dr. Coupland regarded the emphysema as the real cause of the general dilatation of the vessel.[2] Dr. Conway Evans, in the Pathological Transactions for 1866, describes a case in which there was both general dilatation of the artery and hypertrophic thickening of its walls, with atheroma of the internal coat, associated with a contracted mitral orifice. Dr. Evans refers to other similar cases, recorded by Drs. Quain, Peacock, and Bristowe, in all of which, as in the case he describes, marked hypertrophy of the right ventricle was present.

No case of aneurism of the main trunk of the pulmonary artery is referred to in Dr. Peacock's index to the Pathological Transactions, for vols. xvi. to xxv. inclusive; and Mr. Erichsen, in his selected

[1] Diseases of the Heart, 4th edit. p. 394, 1849.

[2] Path. Trans. vol. xxvi. 1875.

observations on aneurism, only refers to one case, described by Ambrose Paré, in which there was aneurismal dilatation and ossification of this vessel, from rupture of which the patient died suddenly. Dr. Crisp had met with no recorded case.[1]

Narrowing of the Pulmonary Artery.

Etiology.—Constriction of the pulmonary artery is most commonly situated at its commencement, and is generally a congenital disease, associated with other congenital malformations of the heart, especially with imperfect septum ventriculorum and patent foramen ovale and ductus Botalli (Lebert).[2] *Endocarditis* affecting the pulmonary valves and causing them to adhere by their margins, and so as partly to close the orifice, or *myocarditis*, leading to constriction at the conus arteriosus, are the most common causes of narrowing of the pulmonary artery: and these causes come into action before the end of the third month of intra-uterine life.

Symptoms: Diagnosis.—Dyspnœa and cyanosis becoming obvious as soon as the infant commences active movements, with the physical signs of great hypertrophy of the right side of the heart and a systolic murmur heard over the pulmonary cartilage and conducted upwards and to the left, are the principal points to be observed in the diagnosis of this malady. A more full consideration of its clinical and pathological features will be found in the section on congenital diseases of the heart.

Narrowing of the pulmonary artery beyond the valves is, like other diseases affecting this vessel, of rare occurrence. The most common cause of such local diminution of calibre is compression by a tumor, either aneurismal or from mediastinal growth, or possibly consisting of enlarged bronchial glands.

As the following case well illustrates the principal symptoms of compression of the pulmonary artery, and also presents other features of interest, I may perhaps be allowed to relate it here.

T. D., aged 38, a fireman in a pottery manufactory, came under my observation at the Charing Cross Hospital in October, 1874. He was in his work exposed to great changes of temperature, sometimes working at a temperature of 200° or more, and also to pigment fumes and coal-dust, etc. To these causes he attributed a constant cough from which he had suffered for some time. He stated that he often had to lift heavy weights, as much as 2 cwt., which he would carry in his arms supported against the lower chest. No distinct history of syphilis was elicited; three children were living out of a family of nine. Patient had a severe attack of rheumatic fever in 1856 which lasted seventeen weeks, but had since then continued his work without difficulty up to six weeks previous to his attendance at the hospital. He had only for two weeks been quite disabled from work, on account of palpitation and breathlessness.

The following notes were taken a month before his admission into the hospital, where he continued under my care in the temporary absence of Dr. Silver. The patient was a short, stout man, with a somewhat bloated face, of a dusky pallid hue, with decided lividity of lips. On exposing the chest an enlarged vein was observed coursing from the left shoulder along the second intercostal space to the sternum, the veins generally of the neck were full, and the carotids throbbed visibly. Pupils and pulses equal. The chest was expanded. The heart's apex beat at the sixth rib one inch outside nipple line, and, as indicated by shading in the diagram, the cardiac impulse was diffused from this point below the nipple to the epigastrium, and above the nipple to the second cartilage. Over a circular space B, having its centre at the third cartilage close to the sternum, and extending to the cartilage above and below, an impulse was felt synchronous with the apex-beat but more prolonged and attended by a marked thrill. Immediately succeeding this a second short impulse or shock coincided with the second sound of the heart. At the 2d, mid-sternum, A, the systolic impulse was slight, without thrill, and the diastolic shock more faint. The superficial cardiac dulness was bounded, as indicated in the diagram, by a line extending from the second left cartilage near the sternum to the apex beat, and skirting the region of thrill and the left nipple. To the right the area of dulness reached half way to the right nipple at the level of the fourth rib. A systolic rough bruit was heard most loudly over the region of thrill, B, and was succeeded by a second sound so accentuated as to communicate a shock to the ear. The blowing murmur was loud also at the base of the heart with marked accentuation of the second sound, and (?) a very slight diastolic bruit. Sibilant râles were heard over the chest; at the left posterior base there was some dulness extending round to the lower axilla with weakened respiration and subcrepitant râle. The systolic bruit was more audible at the left than the right suprascapular region.

The legs were œdematous, the liver enlarged, and the abdomen somewhat full, though not containing any considerable amount of fluid.

This patient continued under observation in the hospital until his death in December. Increasing dyspnœa with severe paroxysms, marked cyanosis, and extreme anasarca of the abdominal walls and lower extremities without much increase in the amount of fluid

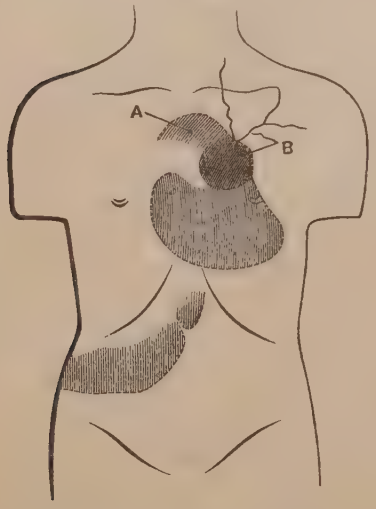
[1] Diseases of the Bloodvessels, 1847.

[2] *Vide* Clinical Lecture, Medical Times and Gazette, January, 1870.

in the peritoneum, œdema of the lungs, increased congestion of the veins of the head and neck, with some regurgitation through the jugulars, were the principal signs of ingravescing disease. The physical signs about the heart did not materially change. The patient became intensely cyanotic shortly before death.

Bearing in mind the extreme rarity of aneurism affecting the main pulmonary artery, yet having regard to the signs of aneurism present, and to the evidence also, in the marked hypertrophy and dilatation of the right ventricle, of obstruction at the pulmonary artery, the diagnosis of aneurism of the aorta pressing upon and narrowing the pulmonary artery was inevitable. From the position of the dulness and thrill, and from the absence of any regurgitant murmur, it was assumed that the aneurism must be projecting forwards from the third portion of the arch. This, however, proved not to be the case.

Fig. 134.



At the autopsy an aneurismal pouch was found arising from the aorta a little above the valves, extending behind the pulmonary artery so as to project three-quarters of an inch beyond it, coming in contact with the left auricular appendix. This aneurism was found to have opened into the pulmonary artery. The left ventricle was greatly hypertrophied, the aorta atheromatous throughout, and from its inner surface several shallow pouches extended, each presenting a thickened margin.

The right ventricle was much dilated and hypertrophied, the pulmonary valves being natural. An oval well-defined aperture half an inch in diameter was found at the distance of three-quarters of an inch above the junction of the left and posterior valves. The vessel was somewhat stretched around the aperture and pouched inwards. Above the anterior valve at the same level was found a depressed smooth surface of irregular outline, having a raised, hard, puckered margin. The left ventricle was greatly hypertrophied. The aorta was slightly contracted at its orifice, but immediately beyond the valves, which were healthy, it was dilated, and from the left side a wide-mouthed pouch extended behind the pulmonary artery, so as to project for three-quarters of an inch beyond it in contact with the left auricular appendix. Several shallow secondary pouches were to be seen on the inner surface of the sac, one projecting into the calibre of the pulmonary artery, and another having given way into it, forming the aperture above described. The liver showed lesions referable to drink and perhaps to syphilis.

This case is of pathological interest inasmuch as it is an example of a rare disease—atheromatous erosion of the pulmonary artery. Clinically, the symptoms and signs were principally those of stenosis of the pulmonary artery and aneurism of the aorta.

In this case the most important signs and symptoms were those most characteristic of constriction of the pulmonary artery beyond the valves. The systolic bruit most intense over the pulmonary cartilages, and here accompanied by systolic impulse and thrill with great accentuation of the second sound, were signs attributable to aortic aneurism; but the marked evidence of hypertrophy and dilatation of the right heart, the general venous engorgement with regurgitation through the veins in the neck and general dropsy, pointed to an obstruction to the exit of blood from the right ventricle. In simpler cases the systolic murmur is characterized as pulmonary by its being conducted upwards and to the left, or to the left interscapular region. Accentuation of the second sound is insisted upon by Professor Quincke[1] as of importance in distinguishing constriction of the vessel beyond the valves from stenosis at or within the ventricular orifice when the second sound is obscured. There may be dilatation of the vessel above the point of constriction.

Dr. Peacock[2] in a tabulated collection of thirty-three cases of aortic aneurism opening into the heart or great vessel includes fifteen cases in which the communication was with the pulmonary artery. The aortic aneurisms in those cases arose with one exception from the ascending aorta, and in most instances immediately above the valves; the perforation of the pulmonary artery took place, with two exceptions, within an inch and a half of the valves. In two instances the vessel was perforated below its bifurcation.

These cases suffice to show that aortic aneurism is the most common cause of

[1] Ziemssen's Cyclopædia, vol. vi. Diseases of the Bloodvessels.

[2] Path. Trans. vol. xix. p. 126.

pressure upon the pulmonary artery. The degree of pressure from this cause must vary infinitely, and the cases must differ correspondingly in the relative intensity of the symptoms referable to the aneurism and to the compression of the pulmonary vessel. In the case above related the rupture of the aneurism only accelerated by a few hours the death of the patient, which was rapidly approaching, from symptoms referable to dilated right heart and obstructed venous circulation.

Murmur Over the Pulmonary Artery.

A pulmonary murmur systolic in time, soft in quality, and of medium or low pitch, is commonly heard over the second and third left cartilages close to the sternum without there being reason to suspect any disease of the vessel. This murmur is very local—not conducted in any direction. It is heard most frequently in young women and children, being then associated with arterial bruits in the neck, and with the venous hum audible over the jugulars (more especially over the right jugular, in which vessel a thrill may commonly be felt). Whatever the exact mechanism of the murmur may be anæmia is its most common cause. The pulmonary artery at its commencement is very superficial, and it is readily conceivable how the rush of a thin watery blood through an orifice so close under the ear should cause an appreciable sound. In children, and even in adults whose cartilages are tolerably resilient, the murmur may be produced or much intensified by pressure with the stethoscope; and it may be removed by such full inspiration as lifts the cartilages and ribs from pressing upon the vessel.

In extreme anæmia murmurs of the same kind may be heard all over the cardiac region, being generated at the several orifices of the heart.

In cases of retracted left lung from old-standing disease a murmur is frequently audible over the pulmonary artery, probably induced by the flattening of the chest wall bringing the cartilages in contact with the vessels. The tension of blood within the artery is always increased in these cases, as shown by the accentuation of the second sound, and sometimes in marked cases of fibrosis of the lung the division of the artery as it enters the affected organ is positively constricted and wrinkled. In other cases an enlarged and hardened gland will intrude upon the calibre of the vessel as it enters the lung. In displacement of heart from fluid effusions or other causes a systolic murmur may be heard over the pulmonary artery.

Pulmonary Artery within the Lung.

Although disease of the main trunk of the pulmonary artery is exceedingly rare, atheroma and even aneurism of its branches within the lung are frequently met with, but only, with equally rare exceptions, in cases of disease disorganizing the lung, uncovering its vessels, and involving their walls in its destructive processes.

It is in the course of excavation of the lung in phthisis that thickenings, erosions, dilatations or actual aneurism of the branches of the pulmonary artery are most commonly met with. We have at once under these conditions the three most important determining causes of atheroma and aneurism, viz.: *increased blood pressure*, on account of the many vessels which are occluded, *local loss of support* from breaking down of the tissues around, and *softening of the arterial wall* by inflammatory changes, also quite of a local character.

In acute ulcerative destruction of the lung such vessels as do not become occluded in good time are apt to become softened or eroded, and, by their rupture, to give rise to copious and sometimes fatal hemorrhage. In cases in which the destruction of the lung is less acute and violent, cavities form with more or less trabeculated walls, the trabeculæ consisting partly of bronchi, but chiefly of vessels surrounded by a certain thickness of condensed tissue. These vessels are as a rule occluded, but exceptions are occasionally met with. We frequently find in chronic cavities a large branch of the pulmonary artery which is quite patent, occupying a trabecula, or coursing along the wall of a cavity immediately beneath the limiting membrane. Such vessels, missing their wonted support on the cavity side, become strained by the blood-pressure. At first the arterial wall thickens,—not from hypertrophy, as Dr. Rasmussen has suggested,[1] for the thickening is limited to the side exposed, and has a uniform smooth section in which nothing but a commingling of connective tissue elements affecting the whole thickness of the wall and obscuring all distinctions between the coats can be seen. It is evidently an inflammatory process of the nature of endarteritis which affects these vessels, and although the thickening appears hard and fibroid, it nevertheless yields before the constant blood-pressure, the calibre of the vessel commencing to dilate at this point. Dilatation goes on

[1] Edin. Med. Journ., paper translated by Dr. Moore, vol. xiv.

in the usual way of aneurism, the originally thickened coat thinning as it becomes spread out before the increasing intrusion of blood, until it forms a brittle, soft, papery layer which cannot be recognized from a fibrinous lamina. Thus a most typical sacculated aneurism may form, projecting into a cavity more or less occupied by laminated fibrin, and which may rupture at any period of its formation.

The more chronic the cavity the more suitable the conditions for the formation of a sacculated aneurism.

In chronic cavities which have from exposure to some evil influences become inflamed or ulcerous, vessels imbedded in the walls, or occupying trabeculæ are rapidly laid bare, and may either become perforated or may dilate into irregularly shaped fusiform aneurisms. An attempt (often successful) at occluding such vessels may frequently be observed in the formation of a firm oat-shaped coagulum which is attached to the internal surface of the artery corresponding with the point of exposure. This plug may gradually enlarge and close the vessel.

There are some specimens in the Brompton Hospital Museum, showing sacculated aneurisms projecting into and occupying bronchial dilatations.

ETIOLOGY.—The etiology of these aneurisms is included in their pathology. They are of strictly local origin, and it is doubtful if any constitutional conditions influence their production.

Age and Sex.—These conditions also only affect the occurrence of aneurism according as they influence the pulmonary disease. In fifteen cases which I have tabulated[1] the ages of the patients varied from fourteen to forty. I have since, however, met with a case of fatal hæmoptysis in an infant seven months old, from erosion of a dilated pulmonary vessel in a cavity.[2] Three of the above cases were females and the rest males: the infant just alluded to, however, was a female, and probably the preponderance of males in so small a number is of accidental occurrence.

SYMPTOMS AND SIGNS.—Copious hæmoptysis, repeated at short intervals, occurring in a case in which there is excavation of the lung, is the most characteristic symptom of the rupture of an aneurism, or a large vessel, within the lungs. The first gush of blood is of a dark venous color but quite pure and unmixed. No other symptom or sign indicates the presence of these lesions of the pulmonary artery save in exceptional cases. In one case that fell under the notice of the author but a few hours before death, a very peculiar interrupted form of amphoric breathing was explained *post mortem* by a small aneurism projecting into the chief bronchus at its point of communication with a large cavity.

DIAGNOSIS.—This can be made with tolerable certainty from the character of the hæmoptysis, as above explained. The more chronic and quiescent the cavity the more likely is sudden hæmoptysis to be derived from this source. Sometimes the excavation containing an aneurism is of very small dimensions.

PROGNOSIS.—Always of course very grave, but by no means necessarily fatal. Cases of the most profuse and oft-repeated hæmoptysis sometimes completely recover.

TREATMENT.—Absolute and prolonged rest with full doses of ergot give the best results. A very nice discrimination is needed in the management of these cases, especially as regards stimulants. It is at the moment of fainting that the best opportunity of coagulation occurs, and one must not be in a hurry to restore the force of the circulation. These patients, too, often recover and make blood very rapidly, and then are apt to get a return of their hæmoptysis. A restriction of diet, especially as regards butcher's meat, is often useful, and no stimulants should be allowed.

[1] On the Pathology of Fatal Hæmoptysis. Path. Trans. vol. xxii. 1871.

[2] Ibid. vol. xxv. Dr. Fagge has since reported a case similar to aneurism occurring in a female child aged 2¾ years. Ibid. vol. xxviii.

ON DISEASES OF THE CORONARY ARTERIES.

By R. Douglas Powell, M.D., F.R.C.P.

The coronary arteries may be affected with any of those lesions—atheroma, fatty degeneration, calcification, occlusion, dilatation, or aneurism—to which other similar vessels are liable: and, as is also the case with like vessels going to important parts, the phenomena indicative of disease are all referable to damaged nutrition and disordered functions of the organ, in the present instance the heart, to which the vessels are distributed. The diagnosis of disease of the coronary vessels is therefore a pathological inference which is helped by no symptoms directly attributable to alteration in them. Extensive disease may exist without giving rise to any suspicious signs; indeed a moment's consideration of the conditions of the coronary circulation suffices to enable us to see that they may be varied or interfered with independently of disease affecting the vessels themselves.

The coronary arteries are, unlike other systemic vessels, filled at the moment of cardiac relaxation by the systole of the aorta forcing the blood back upon the closed aortic valves. If the aortic valves be damaged so as to admit of free regurgitation, the pressure of blood in the coronary arteries is thereby more or less diminished, and the vigor of the circulation through them lessened. Again, atheromatous disease of the aorta at its origin not unfrequently leads to almost complete closure of the coronary vessels at their commencement. Other morbid conditions, such as an undue rigidity of the aorta or aneurism affecting it may by interfering with the rebound or systole of the vessel materially influence the coronary circulation. Hence those disease-phenomena, such as angina, or irregular or failing heart's action with syncopal attacks which, when no more definite physical signs are present, are regarded as being due to fatty degeneration of the heart in consequence of a diseased condition of its vessels, may equally be due to disorder or derangement of the circulation through the vessels arising from some one of several other causes.

Atheroma, calcification.—*Angina pectoris* is frequently connected with calcification of the coronary arteries, but by no means necessarily so. Of three of the most rapidly fatal cases of angina ever recorded, viz., those related by Dr. Latham,[1] in only one was disease of these vessels present to any appreciable extent. Dr. Dickinson, in the seventeenth volume of the Pathological Transactions, calls attention to occlusion of the coronary arteries at their commencement as a cause of angina. He relates three cases in which "soft atheroma spreading under the lining of the aorta" had caused great narrowing or complete closure of the mouths of the vessels which were otherwise quite healthy. The muscular substance of the heart was in each case slightly fatty but not atrophied. A similar case is recorded by Mr. Spenser Watson in vol. xix. of the same Society's Transactions. Professor Gardiner has more fully treated of this subject elsewhere in this *System of Medicine*. Dr. Quain[2] has shown in how large a proportion of cases of true fatty degeneration of the heart the faulty nutrition is traceable to diseased vessels.

Thrombosis.—Dr. Hayden[3] refers to the occasional occurrence of thrombosis affecting the coronary vessels as a cause of acute fatty degeneration of the heart. In a case of Dr. Quain's,[4] in which the aorta was dilated, the left coronary artery was found to be completely obliterated at the first part of its course and occupied for an inch further by an adherent clot, apparently the result of thrombosis. In this case great cardiac agony was experienced by the patient, only relieved by sedatives for two months before death; there was however only a slight amount of fatty degeneration present.

Aneurism.—Aneurism of the coronary artery has been met with as a "museum curiosity" in several instances. Dr. Gee records a remarkable case in the St. Bartholomew's Hospital Reports, vol. vii., in which three aneurisms were found upon these arteries in a boy aged seven years, who had died with scarlatinal dropsy, pneumonia and meningitis. In the St.

[1] Diseases of the Heart, New. Syd. Soc. Edit. p. 450 et seq.
[2] Med.-Chir. Trans. vol. xxxiii.
[3] Diseases of the Heart and Aorta, p. 1017.
[4] Path. Trans. vol. xxiii. p. 57.

Thomas's Museum Catalogue[1] a specimen is described showing aneurismal dilatations along the course of the coronary arteries varying in size from that of a pea downwards, sacculated, some empty, others completely filled by adherent buff-colored clot. The heart was removed from a man aged 22, who had died of pulmonary apoplexy and hemorrhage into the kidneys, and who therefore was presumably the subject of general arterial disease.

The materials are not at present available for any further clinical consideration of diseases of the coronary arteries. In minute anatomy these diseases present no peculiarities.

[1] Vol. iii. No. 81.

[HÆMOPHILIA.

By Henry Hartshorne, M.D.

This is often designated as the *hemorrhagic diathesis*. Its characteristic is, a tendency to spontaneous hemorrhages from various parts of the body, and a disposition to bleed copiously, or for a long time, from very slight wounds. Lancing the gums, for instance, in a hæmophilic child, may be followed by a serious flow of blood, hard to arrest. Later in life, the extraction of a tooth may endanger life in the same way.

In married women, who are "bleeders," coitus may be followed by hemorrhage from the vagina; and, during lactation, the nipples have been known to bleed at the time of suction. Ecchymoses, in such persons, take place upon occasion of the slightest bruises. Sometimes purpuric vesicles form upon the skin, which burst, discharging blood. Epistaxis is common in those who suffer from Hæmophilia, and may be fatal in spite of treatment. The most trifling surgical operations are dangerous to such persons. A well-known clergyman in Philadelphia, a few years ago, lost his life by hemorrhage following the excision of a small wen, no larger than an olive, from his side.

Hæmophilia is usually *hereditary*. Several children of a parent so affected may exhibit the diathesis; although it is not rare for some of them to escape. A mother belonging to a hæmophilic family may herself be free from the tendency, which reappears in her offspring.

It is a common statement with authors, that women are much less subject to Hæmophilia than men; and that, in members of "bleeding" families, pregnancy is not especially liable to dangerous hemorrhages. This last statement is of doubtful accuracy. Investigations by two German pathologists, Börner[1] and Kehrer[2], have brought to light facts showing that very dangerous *post partum* hemorrhages do occur in women of such families; and that such a hereditary proclivity probably accounts for many deaths by uterine hemorrhage, for which some other explanation has been accepted. Dr. Börner believes that reproductive activity intensifies the hæmophilic predisposition, which, before puberty, may have been latent. Menstruation, however, in such persons, is not apt to deviate greatly from the conditions belonging to health. Sometimes menorrhagia occurs, and, in a few cases, vicarious hemorrhagic discharges. Abortion is not frequent in hæmophilic subjects; but, when it takes place, it is always dangerous, and often fatal, from profuse hemorrhage.

The climacteric period, in hæmophilic women, is sometimes attended by violent menorrhagia; in other instances the cessation of the menses is delayed to a late period of life.

Immermann[3] asserts, on the basis of statistics, that Hæmophilia is more common in Germany than elsewhere in Europe; next, in Great Britain; then, successively, in Sweden, Norway, Denmark, North America, Holland, Belgium, Switzerland, Russia, and Poland. More rare in France, it does not appear to have been known in Italy, Spain, Portugal, Greece, or Turkey. Such a negation, however, of knowledge concerning its occurrence may perhaps be explained by its having been, so far, overlooked in some countries, from the attention of the pro-

[1 Wiener Medicinische Wochenschrift, Aug. 17 to Sept. 21, 1878.]

[2 Archiv für Gynäkologie, Band x.]

[3 Ziemssen's Cyclopædia, vol. xvii.]

fession not having been specially called to it. Immermann also states that persons of Jewish descent are particularly liable to it, whatever may be the locality of their residence. This seems to point to a comparatively small influence of climate in its production or promotion.

Of the *pathology* of Hæmophilia, diverse views are held. Formerly, it was supposed that the blood was deficient in red corpuscles and fibrin. When it is examined after considerable hemorrhage has taken place, such a deficiency is probably often found. But, at other times, there seems to be rather a disposition to plethora in hæmophilic persons. They bear great losses of blood wonderfully well, and rapidly make them up. Before attacks of spontaneous hemorrhage, moreover, local congestions are sometimes apparent.

Congenital weakness and thinness of the walls of the bloodvessels is, by some pathologists, believed to account for the excessive facility of extravasation of blood. Sir W. Jenner says that "the tissues are soft and bruise easily; the blood is slow in coagulating, although it coagulates as firmly as in health; that is, blood is formed rapidly, and there is a tendency to plethora of the small vessels, so that when the patient is looking his best, injuries have the worst effect, and spontaneous hemorrhages are most likely to occur."

P. Kidd, in a paper read before the London Royal Medical and Chirurgical Society,[1] reported a microscopical examination of the aorta, vena cava, and small vessels of the mouth of a child, six years of age, which died from spontaneous oral hemorrhage. The coats of the aorta and vena cava were normal in appearance. The small arteries, veins and capillaries of the mouth, especially the smallest veins, were extensively altered. The morbid change consisted chiefly in a great proliferation of the epithelioid cells lining the vessels. This was observed in the *vasa vasorum* even of the aorta and vena cava. Some of the smaller arteries had also undergone degeneration of the muscular tissue of their middle coat.

Other investigators have asserted that, in Hæmophilia, there is an unusually superficial distribution of the cutaneous and subcutaneous vessels; also, in the absence of actual degeneration, marked thinness of the vascular walls. Again, in a certain proportion of cases, hypertrophy of the heart has been found to exist.

If an opinion in regard to the pathology of this affection may be ventured, it must be, that congenital and hereditary *delicacy and defect of resistance of the walls of the bloodvessels* is the main cause of proclivity to hemorrhage, and difficulty in its suppression. Hypertrophy of the heart may be induced by the absence, in great part, of the assistance which should be given by the smaller arteries in the circulation of the blood; obliging the heart to increase its own efforts to accomplish the round of blood-distribution. Local congestions may then easily occur, because of the dilatability of the weakened vessels; and, in their state of distension, a slight disturbing cause may cause rupture of the vascular coats and hemorrhage. This will be more or less serious, according to its seat and amount.

[1] British Med. Journal, May 25, 1878.]

Treatment. — For the *diathesis* of Hæmophilia there can be, we must suppose, no specific remedy; nor do we know of any therapeutical measures likely to do much towards insuring its removal or correction. Immermann objects to the use of iron (on account of the frequently plethoric condition of hæmophilic persons), except as a restorative tonic after loss of blood has been sufficient to produce temporary anæmia. J. Wickham Legg, in his work upon the subject, advises caution in the suppression of spontaneous hemorrhages; considering that this should be delayed until the distended vessels have obtained relief.

For the arrest of *excessive* hemorrhages, in hæmophilic subjects, the same general and local remedies are appropriate as in other hemorrhagic cases. Rest is imperative in every instance. Pressure is sometimes available; styptic applications, such as are described in works on surgery, ought to be promptly and perseveringly used. Ice, in some cases, and the hot water douche (110° to 120° Fahr.) in others, will answer a good purpose. Ergot, tincture of chloride of iron, acetate of lead, tannin, &c., may be given internally. Hypodermic injection of ergotin has been found efficacious in several instances.[1] For dental hemorrhage, particularly, Verneuil[2] advises quinine, internally, one or two grammes daily. Of course this treatment cannot be long continued.

Certain precautions are of importance with hæmophilic persons. Wounds and abrasions of all kinds must be sedulously avoided. Surgical operations, even of the most apparently trifling kind, should not be performed upon such persons, unless from urgent necessity, for the saving of life. Violent exercise, and even great emotional excitement, ought to be guarded

[1] Porak, reported in La Tribune Médicale, quoted in Phila. Med. Times, Aug. 16, 1879. His formula was, Bonjean's ergotin, two grammes, glycerin, thirty grammes. Twenty drops were hypodermically injected in cases of epistaxis, into the lip or cheek.]

[2] Journal de Médecine et de Chirurgie, June, 1879.]

against, on account of the danger of perturbation of the circulation.

Legg, Immermann and others consider that the marriage of hæmophilic persons should be discouraged or forbidden, so as to interrupt the transmission of so fatal an inheritance. Such a restriction, even if sustained by legislation, would be very difficult to enforce, and would sometimes work to social disadvantage. We can scarcely insist upon it absolutely, since, when one of the parents has an entirely untainted constitution, each generation has a prospect of greater and greater attenuation of the inherited morbid proclivity. Certainly, cousins, even of the second or third degree, in hæmophilic families, ought never to be allowed to marry. An extreme manifestation of the diathesis, moreover, ought, in any case, to prohibit marriage.]

INFLAMMATION OF THE LYMPHATIC VESSELS.

By J. Russell Reynolds, M.D., F.R.S.

Affections of the system of lymphatic vessels are closely associated with diseases of the skin, of the glandular apparatus, and of other organs which may be the seats of dyscrasic and diathetic disease; and hence the major part of their pathology as well as of their clinical history will be found in the articles on Erysipelas, Hodgkin's Disease, Leucocythæmia, and Pyæmia. Sometimes, however, from so-called "accidental" conditions, an inflammatory process may occur, independently of any one of those more general changes in the organism; and this may exist in such form in the lymphatic vessels as to merit a separate notice.

Synonyms.—The terms Adenitis, Angeioleucitis, Lymphangitis, and Lymphadenitis, have been used to denote this state.

Causes.—These may be placed in two categories: (1) those which are simply accidental, such as exposure to cold, wounds, bruises, strains; and (2) those which carry with them some toxic agent which affects the body generally, and, it may be, mainly through the lymphatic vessels. When the lymphatic vessels are the seat of inflammatory change, as the result of "accidental" injury, it is to be found that the constitutional state is unsatisfactory. There is to be traced some taint, either hereditary or acquired since birth, which disposes the individual to lymphatic disease, and without which a mere bruise or wound would have been inoperative. Occasionally rapid inflammation has occurred after a simple injury; when some poison has been introduced into the body and has become the starting-point of Lymphangitis, the body may have been previously healthy, but the impression so made upon it may be such as to lead to most mischievous results.

The most common causes are injury to the nails, especially of the foot, chronic ulcers of the skin, stings, punctured wounds, bites, the introduction of unwholesome animal matter from wounds, abraded mucous surfaces, morbid mucous secretions, or any tissue undergoing unhealthy change. The surface of the wound may absorb some poison from the air; diphtheria has been followed by this disease; the vessels in proximity to cancerous, tubercular, or other morbid growths, may become the seat of inflammation.

Symptoms.—The classical signs of inflammation are those which constitute the local indications of Lymphangitis, viz., pain, tenderness, redness, and swelling. These are obvious when the superficial vessels are inflamed, but less distinct when the more deeply seated lymphatics are especially involved. The pain is not, as a rule, severe; there may be only stiffness, or a stinging and burning sensation. The tenderness is in proportion to the superficiality of the inflammation and its association with dermatitis, either simple or specific, and it is sometimes very great. The redness, sometimes of vinous hue, is observed to run in long narrow lines along the course of the vessels, and often forming a network extending from the periphery towards the trunk, and reaching laterally beyond the lymphatics. The swelling of the vessels may be distinct; they are hardened, knotted, and enlarged, but the changes they exhibit do not pass downwards. The glands into which the lymphatics pass become speedily inflamed,

and the skin and cellular tissue are involved in a general inflammatory process, usually of erysipelatous sort. The inflammation of the vessels usually stops at the gland nearest to the seat of injury, and œdema of the skin and subcutaneous areolar tissue exists beyond the site of inflammation.

The course of the disease may be rapid; and, when associated with some classes of poisons, rapidly fatal, passing into suppuration, sloughs, or gangrene; but, on the other hand, the inflammation may be resolved; or it may pass into a chronic state, with much induration of skin, and hypertrophy of some of its elements.

The general symptoms, like those of erysipelas, vary with the nature of their cause. They may be slight when secondary to a merely local injury; but severe and of adynamic character when the result of poisonous infection.

Usually there is a feeling of chilliness, rather than a rigor, at the onset, followed by irregular alternations of heat and cold, with trembling of the limbs. The pulse is always frequent, but variable in force and volume. There is nausea and præcordial discomfort, followed by vomiting, insomnia, and delirium. Such febrile symptoms may precede the appearance of local changes, and become aggravated as the latter are developed. Rigors, attended with profuse sweating, and accompanied by distension of the abdomen, dyspnœa, very frequent pulse, and muttering delirium, are the signs of approaching death by blood-poisoning.

Diagnosis. — Phlebitis may resemble Lymphangitis in its mode of origin and in many of its symptoms, but differs at its commencement in the more distinctly localized character of the ailment, in the larger size and smaller number of the red lines which mark its existence, in their greater hardness, and less frequent tendency to become associated with changes in the cellular tissue. The swelling is less, the pain not so severe, and the general disturbance less pronounced. It must be remembered, however, that the two conditions may coexist.

From erysipelas this disease may be distinguished by the presence of those special vascular changes which are observed in inflammation of the lymphatics, and which are not present in erysipelas. The latter affection is of relatively shorter duration, exhibits more general inflammation of the skin, and is frequently associated with general toxæmia, much more highly marked than in Lymphangitis.

It is sufficient to say that Lymphangitis has been sometimes mistaken for simple erythema or erythema nodosum, to put the practitioner on his guard against a repetition of such errors.

Structural Changes. — Thickening of the walls of the vessels, infiltration of the connective tissue in their neighborhood, pus in and about them, glandular suppurations, and sloughs, are the most common appearances. The skin is often covered with phlyctenæ, or with spots of gangrene, while the central organs may present no departure from health, or only such as are common to all toxæmic and adynamic states. Secondary abscesses are sometimes found in liver or in lung, and phlebitis is by no means uncommon.

The General Treatment of Lymphangitis requires no special notice, as it differs in no respect from that which is required for the various maladies of which it forms a part; and the local treatment of its complications is such as falls into the province of the surgeon, and requires no description here.

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